PHTHISIS PULMONALIS

CAUSES.—The causation of phthisis pulmonalis is a matter of far-reaching importance to the human race, inasmuch as statistics show that one-seventh of the total death-rate of the world is attributable to this disease.

Phthisis was known to Hippocrates (460-377 B.C.); and in all probability it has existed from the earliest times.

Geography.—Laborious and careful research has established the fact that the geographical distribution of phthisis is coextensive with the habitable regions of the globe. We have the high authority of Hirsch for the statement that pulmonary consumption is "a disease of all times and countries."

But although it cannot be admitted that any part of the world manifests a complete immunity from this disease, Lombard's maps show that it is all but absent in certain Arctic regions, deserts, and places situated at great altitudes; in other words, as pointed out by Dr. Ransome, just where the population is most scanty. If we survey the statistics of various countries, a special incidence on certain districts and towns comes out in the clearest manner. It is perhaps doubtful, as Hirsch remarks, whether a comparison of the statistics of different countries possesses the same value as a study of the returns of individual towns or districts, which ensure a greater degree of accuracy.

Climate.—The influence of climate has been much discussed. Some writers hold that phthisis is commoner in hot than in cold countries; others again consider that it is of more frequent occurrence in temperate climates. But a review of the information at present available leads to the conclusion adopted by Hirsch, that "the mean level of the temperature has no significance for the frequency or rarity of phthisis in any locality." A few examples will illustrate this point. The mortality from this cause in Iceland is very low, whereas in North Greenland phthisis is one of the commonest causes of death. On the north coast of Africa, Morocco and Algiers are distinguished by a remarkable freedom from consumption; but at Tunis, and at Alexandria and Damietta on the sea-coast of Egypt, the disease is very prevalent: in the interior of Upper and Lower Egypt, on the other hand, phthisis is decidedly uncommon. Other discrepancies equally striking might be quoted to prove that places sharing a similar climate may widely differ in their phthisis death-rate.

Consumption seems to follow in the wake of advancing civilisation, especially where men congregate together in large numbers. All accounts agree as to its extraordinary prevalence in New Caledonia, Hawaii, Tahiti, and other South Pacific Islands. There is a strong consensus of opinion that phthisis has become far more rife in these parts, and also among the Maoris of New Zealand, since the date of the European
immigration; when, as Hirsch says, "the natives began to adopt the manners and habits of the Europeans." Until recent times the mortality from this disease among the troops of the British Army in the most widely separated parts of the world was deplorably high, more particularly in times of peace; and often considerably exceeded the mortality of the resident population. Improved hygienic arrangements in the barracks, especially as regards overcrowding and ventilation, have reduced the death-rate from phthisis in the most remarkable manner.

Such facts cannot be reconciled with the belief that climate is an important etiological factor. But while the evidence negatives the opinion that hot climates favour phthisis, all authors are agreed that the disease in tropical countries assumes a most acute and virulent type.

Moisture.—A maleficent influence has been ascribed to moisture of the atmosphere and soil. In America, Bowditch was led to believe, by an inquiry into the incidence of phthisis on the inhabitants of certain places, that the disease is most prevalent in areas where the soil is impregnated with moisture. He found in certain localities that efficient drainage was followed by a diminution of phthisis. Dr. Milroy's investigations in Scotland gave similar results. Working out the same idea in England, a few years later, Sir George Buchanan made the discovery that in several towns the phthisis death-rate had undergone a notable decrease since the introduction of an improved system of sewerage—a result which he attributed to draining of the subsoil water. He accordingly expressed the opinion that the mortality from consumption is directly related to the degree of dampness of the soil. But there are certain considerations which make it difficult to regard this conclusion as one of general application. For example, in one of the towns investigated by Buchanan the mortality from phthisis rose remarkably after carrying out the drainage work; moreover, in Berlin, and some other towns in Germany and in England, improvement in drainage has not been followed by the good results anticipated by Buchanan. Lastly, in some of the districts quoted by Buchanan in support of his hypothesis, subsequent investigation by Dr. Kelly has elicited the fact that the diminution in the phthisis rate noted at first has not been sustained in recent years; and, as Dr. Payne remarks, although the subsoil of London is becoming drier every year, owing to the large area covered by houses and almost impervious pavements, there is no corresponding decrease in the amount of phthisis in this town. It is indeed a significant fact that in so wet a country as Holland the death-rate from phthisis is rather low. From these considerations we are driven to admit with Hirsch that other etiological factors beside the influence of soil are probably concerned—factors that "serve to neutralise the benefits even of the most favourable conditions of soil."

Altitude.—Observations made in divers parts of the world have left no doubt of the infrequency of phthisis at higher altitudes, though instances of the disease are not wanting even at the very highest points. The sparseness of the population at such levels may account to some
extent for their relative immunity, but not entirely; for in some large commercial and not very sanitary towns in Mexico, and on the Andes, situated at an elevation of from 7000 to 13,000 feet, the extreme rarity of phthisis is generally admitted; as Hirsch writes, "that the influences which go with very considerable altitudes have the power to overcome those detrimental things that arise from a bad kind of hygiene and social life, in so far as they tend to produce consumption."

Statistics from Switzerland strongly confirm the opinion that a great elevation affords some protection, though they supply no proof of anything like complete immunity. The explanation of the beneficial action of altitude is by no means clear. The extreme freedom of the air from impurities of all kinds and the dryness of the atmosphere have been alleged as the principal causes. But in the case of the unsanitary towns on the Andes above referred to, the absence of organic atmospheric impurities cannot be assumed; and that dryness of the air does not in itself confer any protection is clearly shown by the returns from the towns on the sea-coast of Egypt to which attention has already been directed. Hirsch makes the suggestion that people living at great elevations and breathing rarefied air are obliged to make deeper inspiration, and acquire in consequence a more vigorous development of the respiratory organs, which are thus enabled to offer a more powerful resistance to external influences. The bearing of this view on the bacillary origin of the disease will be discussed further on.

No race is exempt from the ravages of consumption. Hirsch states that among the Kanakas—the natives of New Caledonia—two-fifths of the total mortality is due to phthisis. And Dr. Osler states, on the authority of Surgeon Kennedy, that the mortality from this cause in a tribe of Red Indians of the Rocky Mountains living in a splendid climate amounts to 23 per cent of the total death-rate. The Negro appears to be extremely vulnerable, especially when removed from his own country; and in this race phthisis runs a very acute course.

The evidence with regard to the Jews is somewhat ambiguous. There is a general impression that Jews are less afflicted with tuberculosis than Christians. This has been accounted for by the more careful selection of carcasses in Jewish slaughter-houses, and by the more frequent house-cleanings practised by the Hebrews. There is some reason to believe that this favourable estimate applies only to the well-to-do members of the community. Further investigation must decide whether the current opinion is correct or not.

Sociological.—We have now to consider another class of etiological factors; the density of population, and certain injurious influences connected with trades and occupations, particularly those which involve an indoor life.

As the general result of statistical inquiry in different countries, it is clearly proved that the mortality from phthisis is lower in the country than in the towns; and that in the case of towns the mortality on the whole increases with the population. The proneness to phthisis manifested
by dwellers in towns seems to be largely connected with overcrowding in rooms badly ventilated and lighted. Numerous investigations have attested the high death-rate from phthisis in convents, sisterhoods, military barracks, and above all in prisons. These institutions have been notorious for overcrowding and defective ventilation. In jails and convents insufficient food and indoor confinement have no doubt militated also against the health of the inmates, but these factors cannot be said to have been operative in the case of military barracks. Happily the hygienic reforms of recent times have effected an enormous reduction in the mortality from phthisis in such institutions.

Sedentary occupations, whether in town or country, appear to dispose to the disease. Certain trades, particularly those which are associated with much dust, enjoy an evil distinction on account of the prevalence of consumption among their workers. Attention was chiefly directed to this point by Greenhow's excellent reports, in which he traced the influence of dusty occupations in originating diseases of the lungs.

It is generally believed that the sharper particles of dust are most injurious. Flint workers, needle-polishers, file-cutters, grinders and potters supply the largest contingent of pulmonary diseases. It is still undecided whether most cases of this sort attributable to dust ("pneumoconoisoses" of Zenker) are of a tuberculous nature or not. Some authors, among whom is Hilton Fagge, have asserted that practically all these affections of the lungs are tuberculous. Others, including physicians who have seen much of this form of disease, refuse to admit the truth of this statement. It seems to be a fact that in some, perhaps in most of such cases manifest tuberculous lesions are found after death; but even then it may sometimes be difficult to decide whether tuberculosis constitutes the substantive disease, or whether it has implanted itself, as a secondary process, in lungs already the seat of fibroid changes. The researches of Zenker, Virchow, and others leave little doubt as to the occurrence of circumscribed non-tubercular fibroid lesions of the lungs, in consequence of the irritation of finely divided particles of iron and other metallic or mineral substances. But it remains to be proved that massive induration of the lung can be produced by this cause alone without the coexistence of tuberculous disease. This much, however, is certain, that pulmonary affections in persons following dusty occupations, if not always in the first instance tuberculous, are very liable to become so; the chronic inflammation of the bronchi and lung being favourable to the development of this infection.

The comparatively small post-mortem experience in this variety of pulmonary disease which has fallen to my share has almost invariably revealed the presence of chronic tuberculous lesions associated with excessive blackish pigmentation of the lungs. In one or two cases where no evidence of tuberculosis could be detected the lesions consisted of scattered patches of fibrous induration in the peribronchial districts. For a full account of pneumonoconiosis the reader is referred to the article on the subject in the present volume (p. 242).
No age is exempt from pulmonary tuberculosis, though it is less common at the two extremes of life.

The view, which prevailed until recent times, that infants and children under two years of age are scarcely ever attacked, has been proved to be incorrect. Laffouzy found that in several fatal cases of broncho-pneumonia in children under two years, some of which during life were ascribed to measles or cold, tubercle bacilli were present in the broncho-pneumonic patches; although the naked-eye appearances of tuberculosis were not recognisable. These observations were made at an infant asylum in Paris, in which institution one-third of the deaths among the children proved to be the result of some form of tuberculosis.

**Sex.**—The following statistical account by Dr. W. Ogle, formerly Registrar-General, deals with the subject of age and sex on a very large scale.

"There is practically no difference between the two sexes in their respective liabilities to death from phthisis when all question of age is put aside. For the mean annual mortality of males on an average of thirty years from this cause is 2418, and of females 2428 per million living. . . . But when instead of taking the aggregate rates, that is, the death-rates of each sex en bloc, irrespectively of age, we take the rates at each successive age period, there are found to be very remarkable differences between the sexes. In the first quinquennium of life (0-5) the male and female rates are pretty nearly the same, the male being only very slightly the higher. In the next five age periods, covering between them the interval between the ends of the fifth and thirty-fifth years of life, the female rate is in marked excess of the male rate, the excess being especially notable in the periods from ten to twenty years of age. After the thirty-fifth year the reverse is the case, and the male rate becomes the higher, and remains so in each age period to the end of life.

**Table A**

*(Slightly abridged)*

<table>
<thead>
<tr>
<th>Ages.</th>
<th>All</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>20</th>
<th>25</th>
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<td></td>
</tr>
<tr>
<td>Both sexes</td>
<td>2423</td>
<td>1013</td>
<td>461</td>
<td>838</td>
<td>2549</td>
<td>3742</td>
<td>4060</td>
<td>3954</td>
<td>3313</td>
<td>2648</td>
<td>1687</td>
<td>613</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>2418</td>
<td>1084</td>
<td>432</td>
<td>616</td>
<td>2088</td>
<td>3676</td>
<td>3941</td>
<td>4097</td>
<td>3850</td>
<td>3274</td>
<td>2112</td>
<td>730</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>2428</td>
<td>933</td>
<td>491</td>
<td>1061</td>
<td>3008</td>
<td>3798</td>
<td>4165</td>
<td>3826</td>
<td>2812</td>
<td>2075</td>
<td>1322</td>
<td>523</td>
<td></td>
</tr>
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</table>

Mean Annual Mortality from Phthisis (1851-80) per 1,000,000 living at all ages, and at twelve successive age periods.
TABLE D

Mean Annual Mortality (1861-80) per 1,000,000 living of Children in each Year of the first Quinquennium of Life from Phthisis, Males and Females.

<table>
<thead>
<tr>
<th></th>
<th>Under 1</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>All under 5 Years.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>1589</td>
<td>1341</td>
<td>634</td>
<td>394</td>
<td>339</td>
<td>880</td>
</tr>
<tr>
<td>Females</td>
<td>706</td>
<td>1295</td>
<td>655</td>
<td>409</td>
<td>360</td>
<td>842</td>
</tr>
</tbody>
</table>

"It appears from this table that the male death-rate from phthisis in the first year of life is more than twice as high as the female rate; in the second year it is also the higher, but only to the extent of about 3½ per cent; while in the third, fourth, and fifth years of life the female rate is slightly the higher; namely, 3·3 per cent in the third year, 3·8 per cent in the fourth year, and 6·2 per cent in the fifth. These percentage differences are slight; but they run in an ascending series, and, in combination with the figures in Table A, seem to justify the statement that the female liability to phthisis begins to exceed the male liability in the third year of life, and continues to be in excess till somewhere about thirty-five years of age—the maximum of excess being in the ten to twenty years of age period, when the excess reaches 50 per cent.

Dr. Ogle expressly states that the contrast between the male and female mortality described above characterises pulmonary phthisis; and that no similar contrast is observable in the death-rate from other tuberculous diseases.

It is not easy to account for the great excess of the male rate in the first year of life. But as regards the decided excess of the female mortality over the male between the ages of ten and twenty, Dr. J. F. Payne points out that it is at this period of life that the difference between the outdoor life of boys and the indoor life of girls begins; and he suggests that the excessive female mortality at this age is connected with the existence of unfavourable conditions in the house. But, as he further remarks, "the greater liability of the male sex to phthisis after thirty-five years of age seems to be quite unexplained by such considerations."

Dr. Ogle's Tables show that the excess of the female death-rate is not related specially to the child-bearing period, as the excess over the male rate begins to decline after the age of twenty; and after thirty-five the rate has fallen below that of the male sex.

In connection with differences of sex we may now briefly consider the influence of menstruation, pregnancy, parturition and lactation.

Menstruation.—There is no clear evidence that this function, whether at its commencement or subsequently, exerts any definite causative
influence. Irregularity or arrest of the catamenia seems at times to be connected with the onset of haemoptysis, but the nature of this association is probably less intimate than at first sight it appears to be. For it may be safely concluded that a considerable pulmonary haemorrhage at the outset of the phthisical symptoms is a sure sign that the disease is already of long standing, although perhaps hitherto quite latent. Arrest or disorder of the menstrual flow may occasion reflex vascular disturbance, and so give rise to haemorrhage from lungs already diseased; but the occurrence is not by any means common.

Pregnancy.—The influence of pregnancy has been much debated. The disease not infrequently appears to extend during this period; but the impossibility of fixing the date at which the disease begins renders the question a difficult one. Wilson Fox considered that the rapid increase of phthisis in woman between the ages of twenty and thirty-five points somewhat strongly to the influence of pregnancy in the development of the disease. The force of this argument is weakened by the fact that the increase in the male rate (see Ogle's Table A) in the same period is almost as pronounced. Dr. R. E. Thompson, as the result of a statistical inquiry into the subject of phthisis in women, concludes "that the susceptibility of single women is rapidly diminished after thirty years of age, while that of married women maintains its intensity between twenty-five and forty years of age (that is, during the child-bearing period)."

Parturition.—It is not common to find that symptoms of phthisis set in directly after parturition, though this event has generally an accelerating effect upon pre-existing disease.

Lactation, by its debilitating influence on weakly women, may no doubt act as an indirect cause. Dr. Pollock states that the periods of puberty, of gestation, of parturition, and of lactation are fraught with danger to persons disposed to phthisis.

The influence of these conditions on the established disease will be considered in a subsequent section.

General depressing influences.—Among the remoter causes of phthisis may be reckoned all conditions that tend to lower the standard of health; such as insufficient food, anxiety, grief, excessive mental work, want of exercise, fresh air, and sunlight. Among diseases that have the same general effect diabetes mellitus must especially be named—a point on which all authorities are now agreed. It is interesting to note the liability of diabetics to another disease of microbic origin, namely, carbuncle.

Concerning the etiological importance of chronic alcoholism, malignant disease, and syphilis, agreement is less general. Both clinical and post-mortem experience alike support the view that topers are prone to tuberculous affections. The frequent association of peritoneal tuberculosis with cirrhosis of the liver is generally recognised by pathologists. It has been urged that alcohol has a preventive action; and that it tends to promote fibroid changes if tuberculosis should be contracted, both statements are at variance with my own experience.
The association of pulmonary tuberculosis and malignant disease of various organs is by no means rare, though it is very uncommon to find evidence of simultaneous activity of the two diseases. Mr. Roger Williams' statistical investigations appear to him to justify the conclusion that the proclivity to cancer is closely allied to the tuberculous diathesis.

A history of syphilis is not rarely obtained from phthisical patients; but if the former disease be possessed of any etiological influence it can only be of an indirect character.

Rheumatism, gout, insanity, chlorosis, dyspepsia have also been regarded as etiological factors; but their connection with phthisis is not intimate.

The frequency with which pulmonary tuberculosis appears after measles, influenza, and, to a less degree, after whooping-cough is well known. Although these diseases cannot be regarded as immediate causes, it must be admitted, more particularly in the case of influenza and measles, that they are apt to precipitate an eruption of tuberculosis; whether it be in consequence of the attendant pyrexia, or of some other action of their specific virus. There is more to be said in favour of the view that a latent tuberculous focus is lighted up, than that tuberculosis is initiated by the presence of another disease. On the other hand, it is possible that influenza and measles may cause certain changes in the bronchial and pulmonary epithelium which result in a lowering of their power of resistance, and thus lay them open to the invasion of tubercle.

Pneumonia.—It is an old belief that croupous pneumonia may terminate in phthisis; but it is now quite certain that this sequence of events is infinitely rare. Two principal fallacies have been concerned in the origination of this erroneous opinion: in the first place, certain rare cases of pulmonary tuberculosis begin with severe constitutional symptoms, and with signs of extensive infiltration of the lung, simulating acute pneumonia; secondly, delayed resolution, or the exceptional supervision of chronic pneumonia, may give the impression that croupous pneumonia has terminated in phthisis. Patients suffering from the latter disease often declare that their illness began with "inflammation of the lungs"; but such statements will seldom stand the test of a critical examination.

Bronchitis not uncommonly appears to have been the starting-point of phthisis, and there is nothing improbable in such a belief; but, more often, careful inquiry will elicit the fact that symptoms of phthisis preceded the attack of bronchitis. In many instances where the tuberculosi appeared to have supervened on chronic bronchitis, an autopsy has demonstrated that the bronchitis was itself symptomatic of chronic pulmonary tuberculosis. The fact that many phthisical persons attribute their illness to a simple catarrh possesses little scientific value; for what ailment do patients not put down to catching cold?

Pleurisy.—The oft-repeated observation that an attack of pleurisy is frequently followed by phthisis, led to the belief that the latter disease is the result of pleurisy. But there is no doubt that, under such circum-
stances, the original pleurisy has itself been of a tuberculous nature; and secondary, as a rule, to tuberculosis of the lung. There is no reason to think that simple pleurisy disposes to phthisis. In metapneumonic pleurisy, whether sero-fibrinous or purulent, where the effusion is directly due to the pneumonic process, recovery is generally complete and permanent. It is believed by Koch and others that pleuritic adhesions, by impeding the movement of the chest walls and lung, may dispose to phthisis. But patients suffering from deformity of the chest—as the result of kyphoscoliosis, rickets, or caries of the spine—wherein the thoracic movements are greatly restricted—so rarely acquire pulmonary tuberculosis that Rokitansky came to the conclusion that the two affections are antagonistic.

Traumatism has been supposed to play an important part in the causation of phthisis. Mendelssohn has published nine cases of his own, with a reference to seventeen other recorded cases, in which injuries to the chest, of various kinds, were followed by pulmonary tuberculosis. In some instances haemoptysis occurred at the time of the injury or soon afterwards; in others cough and symptoms of pleurisy ensued within a few days or weeks. In one or two cases an interval of a few months, and, in one case, of two years, separated the accident from the appearance of definite symptoms. Mendelssohn ascribes the occurrence of phthisis in such circumstances to laceration or contusion of the lung, and infiltration of its tissues with blood or inflammatory products, favouring the entrance and germination of the tubercle bacilli, which he assumes to be more or less ubiquitous. From the rapidity with which pulmonary symptoms appeared in most of these instances, it seems more reasonable to suppose that injury to the chest wall may rouse into activity some latent tuberculous focus, possibly by laceration or loosening of its fibrous capsule. As the result of direct questions put to many hundred patients suffering from phthisis, I have met with but a comparatively small number who referred their complaints to an injury of any kind. In one or two instances, when the patient gave a history of injury to one side of the chest, physical signs of disease were confined to the opposite side.

Antagonism of other diseases.—Ague has been said to confer a protection against phthisis, but investigations in malarious localities in various quarters of the world have proved that no such antagonism exists.

An attack of erysipelas has been followed by arrest of the pulmonary disease in a few recorded instances.

Disease of the heart.—Rokitansky taught that all conditions which induce a state of vasoconstriction in the heart produce an immunity from tuberculosis. Among the affections included in this category he placed cardiac dilatation and hypertrophy, whether primary or the result of valvular disease, congenital malformation of the heart and great vessel-aneurysma, deformity of the chest depending on rickets, lateral curvature or caries of the spine, pleural effusions, chronic bronchitis, emphysema, bronchial dilatation, pregnancy, or of any other condition tending to obstruct the passage of venous blood through the right side of the
The relation of some of these affections to phthisis has been already mentioned; but the influence of cardiac disease must now be considered. It is undoubtedly uncommon to find phthisis and disease of the heart in the same patient, but this association is by no means so rare as Rokitansky's statement would imply. Most writers agree in saying that mitral stenosis is scarcely ever met with in this association: this combination is rare, but I have seen at least a dozen clinical examples; and in five other cases the two conditions were found on post-mortem examination to be associated.

Rokitansky's view that the antagonism of the two affections depends on the venosity of the blood has been objected to by Lebert and others, on the ground that the subjects of congenital stenosis of the pulmonary artery are exceedingly prone to contract tuberculosis. But in such persons, as Lebert himself admits, the lungs are often small and undeveloped, and their nutrition must be below the average. And, although the bronchial arteries are abnormally developed, in order to supplement the pulmonary circulation, the lungs are still very inadequately supplied with blood, and are less capable than normal lungs of resisting the action of the tuberculous virus. It appears then that, in the case of pulmonary stenosis, increased venosity of the blood is not the sole or perhaps the chief influence at work; and Lebert's objection is possibly not so fundamental as it has been held to be. Dr. Pollock considers that hypertrophy and dilatation of the heart retard the progress of tuberculosis, and that under such conditions a prolonged duration may safely be anticipated. This observation is a very true one, and affords support to the theory of antagonism.

It seems, then, that Rokitansky's doctrine is true, though true in a more limited sense than that in which he intended it to be taken. All diseases of the heart which bring about a passive congestion of the lungs, and consequently an increased venosity of the blood, confer a certain degree of protection against pulmonary tuberculosis; but, in the words of Peacock, "this opposition certainly in no degree amounts to an incompatibility."

Gout.—In the rare instances in which gouty persons acquire tuberculosis the disease runs a very chronic course.

Infection.—The doctrine of the infectious nature of tuberculosis, promulgated by Villemain in 1865, was verified by Robert Koch's discovery in 1882 of the immediate cause of the disease, the tubercle bacillus. After much difficulty he succeeded in isolating and cultivating the microbe. In artificial nutrient media the bacillus was found to grow with extreme slowness. Pure cultures inoculated into healthy animals produced tuberculosis with unfailing certainty. From the fact that the micro-organism can only be cultivated within certain narrow limits of temperature (82° to 105° F., the best temperature being that of the interior of the human body), Koch regarded the bacilli as true parasites, "that is, as finding the conditions necessary to their existence only in the animal or human organisms."
These facts have been confirmed by many observers, but Sir H. Beevor, by means of the method of cultivation introduced by Nocard and Roux, claims to have obtained a very slow growth at a temperature of 60° F. If this observation should be confirmed, the tubercle bacillus could no longer be considered to be an obligatory parasite. The experience of all investigators supports Koch's statement that the microbe resists prolonged drying for months; though when exposed to the action of putrefaction it loses its virulence much sooner. The presence of oxygen is necessary for the growth of the bacillus; sunlight has been said to retard or prevent it.

Persons suffering from tuberculous disease of the lungs are constantly expectorating tubercle bacilli in enormous numbers. That the sputa are infectious has been abundantly proved by experiments on animals. Koch and many others consider that tuberculous sputum is the chief source of the parasite. The extreme tenacity of life which characterises this bacillus warns us that the sputum is dangerous long after it has been expelled from the lungs of a phthisical patient. In many well-authenticated cases accidental inoculation of human beings with sputum or other material derived from tuberculous persons has been followed by local or generalised tuberculosis. In one case, a patient dying of gangrene of the leg was inoculated with tuberculous sputum, and at his death three weeks later a few recent tubercles were found in one lung.

Accidental inoculation of the skin, mostly of the hands, has occurred in different ways; for instance, from washing soiled linen of tuberculous people; by a scratch from a broken spittoon used by a phthisical patient; from wearing the earrings of a person that had died of phthisis; by the prick of a morphia syringe; by post-mortem examinations of tuberculous men or animals. In most of these cases the tuberculosis remained localised, and, in some instances, the disease was cured by timely excision of the affected parts. Ritual circumcision in Jewish infants has been followed in several cases by tuberculous ulceration of the prepuce and swelling of the inguinal glands: in some instances it was proved that the operator, himself tuberculous, had sucked the wound to stop the bleeding; in one case where a phthisical operator had not employed suction, he had squirted wine from his mouth over the wound. Bacteriological examination of the ulcer and enlarged glands of the infant, and of the sputum of the operator, was carried out in some instances, and established the infective and tuberculous nature of the process beyond all doubt. But while the inoculability of tuberculous sputum can no longer be denied, it is nevertheless apparent that direct inoculation is a rare occurrence in man, and in no way accounts for the great mass of human tuberculosis. The rarity of this mode of infection is explained by Baumgarten's discovery that tuberculosis cannot be induced by inoculation of the superficial layers of the skin, subcutaneous puncture being required to ensure a successful result.

The fact that the disease, in the great majority of cases, appears to
begin in the lungs suggested to Koch that the tubercle bacilli enter the body by the air passages. He further expressed the belief that the bacilli were derived from dried sputum which had become pulverised, diffused in the atmosphere, and inhaled into the lungs. This view has been adopted by most subsequent writers. It may be objected that this mode of infection is insusceptible of direct proof; a striking example, however, has been recorded.

Tappeiner, by spraying tuberculous sputum into a cage where dogs were confined, succeeded in inducing pulmonary tuberculosis in some of the animals. In spite of repeated warnings, Tappeiner’s servant, a very robust man aged forty, and free from hereditary taint, insisted on going into the cage, and contracted acute pulmonary tuberculosis from which he died in fourteen weeks. It may be freely admitted that the conditions in this case were not strictly parallel to those that obtain under ordinary circumstances where the amount of tuberculous dust inhaled must be very small; yet the case demonstrates the possibility of man acquiring tuberculosis by inhalation. All observers have admitted the difficulty with which tuberculosis can be communicated to animals by means of inhalation, a fact which Baumgarten maintains is opposed to Koch’s conclusions. After the discovery of the tubercle bacillus the view was very generally expressed that the parasite is ubiquitous, and that every one, especially in towns, must be frequently inhaling the microbe. But a careful and extensive research, conducted in Berlin by Cornet, proved that the bacillus is not so widely distributed as had been assumed. The plan which Cornet adopted was to collect dust with sterilised instruments from the walls of hospitals, prisons, asylums, and private houses, and from the public streets. The dust was mixed with sterilised broth and injected, with full antiseptic precautions, into the peritoneal cavity of guinea-pigs. Many of the animals died rapidly of septic peritonitis; others remained in good health, and a certain number contracted tuberculosis. The specimens of dust which communicated tuberculosis to the animals were obtained from private rooms or wards that had been inhabited by phthisical persons; whereas in surgical wards, out-patient departments, and in quarters not occupied by such persons, the dust, as regards tuberculosis, gave negative results.

In the course of experiments made, after Cornet’s method, by Dr. Heron and Dr. Chaplin with dust from the Victoria Park Chest Hospital, only two out of a total of a hundred guinea-pigs inoculated were attacked by tuberculosis. In both these cases the particular specimen of dust came from the main ventilating shaft, which had not been swept for forty years. Dust taken from the wards, out-patient waiting-room, and pathological laboratory failed to cause tuberculous infection in a single instance.

Dr. C. T. Williams succeeded in detecting a few tubercle bacilli in the air of the Brompton Hospital. His method consisted in the exposure of glass plates, smeared with glycerine, in the ventilating shafts of a ward set apart for phthisical patients. After some days the plates were
examined microscopically and a few bacilli were found. It is very probable that in this case the microbes were conveyed to the glass plates with dust.

Dr. Ransome, by condensing the breath of consumptive people in a glass globe surrounded with a freezing mixture, was able to discover a few tubercle bacilli in two cases. But in such experiments it must be difficult to prevent patients from coughing and expectorating minute quantities of sputum or saliva which may contain bacilli. Numerous workers have failed to verify these observations, and we may assume that the bacillus is not exhaled from the lungs during ordinary respiration. Cornet believes that the great majority of cases of pulmonary tuberculosis are the result of inhalation of dried sputum in association with dust.

Others think that the alimentary canal is a more important channel of infection. Experiments on animals have proved that tuberculous material when swallowed may induce tuberculosis of the mesenteric glands and intestine. Considering that tubercle bacilli may lie dormant amid the dust of houses inhabited by phthisical people, it is not improbable that children may accidentally contaminate their food and thus acquire abdominal tuberculosis. Milk and butter from tuberculous cows, and the flesh of oxen, pigs, and fowls, when imperfectly cooked, may also communicate the disease.

For a detailed discussion of this topic the reader is referred to the article "Tuberculosis" (vol. ii. p. 29). The preponderance of primary disease of the lungs, however, seems to support Koch's view that the virus in most instances enters the body through the respiratory system.

The difficulty found in producing tuberculosis in animals by inhalation may seem to oppose this view. But the pulmonary affection in man, as compared with the artificially induced disease in animals like guinea-pigs and rabbits, is always a chronic process. If an animal after inoculation fail to show evidence of disease in a few weeks or months, the experiment is generally regarded as unsuccessful; whereas, to make the parallel complete, continued observation for a much longer period would be required. For in man the period of latency of pulmonary tuberculosis is generally one of months, or even perhaps of years. The success of infection is largely a matter of dose; a very small dose producing a chronic affection or no result at all, a large dose, on the other hand, causing an acute infection. Moreover, variations in the virulence of the bacillus may be evidenced by corresponding differences in the type of disease. Bacilli subjected to the action of desiccation for months are less capable of active growth than when freshly removed from the animal body or from artificial cultivations.

That Cornet's injections of dried tuberculous dust proved fatal to guinea-pigs in a comparatively few weeks does not constitute a serious objection to this conclusion; injections of such dried matter under the skin or into the peritoneal cavity is a much stronger measure than its introduction by inhalation; for in the latter case the ciliary movement
of the epithelium and the vital resistance of the cells of the respiratory tract represent a powerful defensive mechanism that cannot be claimed for the subcutaneous tissue or the serous membranes. The existence of such a mechanism is well shown by the history of anthracosis and other dust affections of the lungs. Children and animals living in the dusty atmosphere of towns, as Baumgarten remarks, seldom show any of the pigmentation of the lungs and bronchial glands, which is never altogether absent in adults living under similar conditions. It seems as if the ciliary action of the epithelial cells can remove all the foreign particles introduced with the air up to a certain point and for a certain length of time; but after a time the carbon and other particles enter the lymphatic vessels and become deposited in the lungs and in the neighbouring bronchial glands.

It is very probable that tubercle bacilli entering the mouth are taken up by the tonsils and carried to the cervical glands; thence they may pass into the large lymphatic vessels, and thus ultimately reach the lungs.

House infection.—A considerable number of observations have now been recorded in support of the view that the tuberculous virus clings to certain dwellings.

Dr. Ransom’s investigations in Manchester and Salford have shown that tuberculosis is especially apt to haunt houses situated in close courts, narrow streets, and, above all, houses built back to back, where ventilation is necessarily defective. Similar observations have been made in America and Germany. In some of the cases published the evidence is very strong, as, for instance, in the following by Engelmann. A newly-built flat, in a fairly sanitary condition, but badly lighted and ventilated, had been occupied for eight years by three families in succession; all of them had presented a clean bill of health until the family X took up their residence in the same quarters. In this family the mother was consumptive when she came, and died in the flat. Shortly afterwards the family left, having lived there for one year only. The flat was next occupied by the family Y, of seven persons, all healthy; after a year’s stay they left, and some years later the father, mother, and one son died of phthisis, and a boy of chronic peritonitis. A third family, Z, all healthy to begin with, next took the rooms: one child died of meningitis, another of marasmus, and a third contracted hip disease: subsequently the father died of phthisis, another child of meningitis, the mother acquired consumption, and a child became scrofulous. A fourth healthy family, W, next came into residence; after a time the mother became phthisical, and two children died of meningitis. In reference to these facts, Dr. Payne remarks: “Summing up the history it appears that for eight years the dwelling was free from tuberculous diseases. Then came one year’s tenancy by a person already tuberculous. After this, in a period of twelve years, at least twelve cases of tuberculous disease were traced to this source. It is noted that the dwelling was never vacant, the new tenants entering while it was, so to speak, still warm from the last; and
during the whole period it was never painted or cleaned." In other parts of the same house, where cleaning was not neglected, but the conditions were otherwise the same, no cases of tuberculosis could be traced. The facts point strongly to infection in the case of the third and fourth families (Z and W); but in respect to the second family (Y) the evidence is not so convincing, as according to Engelmann's statement some years elapsed between the tenancy of the infected house and the deaths of some of the members from tuberculosis. The hypothesis that the virus is air-borne, and intimately connected with dust, helps us to understand how house-infection may come about. In most of the instances recorded the victims lived in small, ill-ventilated rooms, so that the chances of infection were thereby much increased. The smaller the room the less the likelihood of adequate ventilation, and the greater the opportunity for the accumulation of dust.

An important side light is thrown on this part of the subject by the returns of the mortality from phthisis in the male and female sex among certain agricultural populations in England and Germany, which show a marked excess of the female over the male death-rate. In other words, the males who lead an outdoor life suffer much less from consumption than the females, who spend most of their time indoors. Although, as Payne points out, this disparity in the phthisis death-rate may be explained on the ground that the open-air life of the men is healthier, it is quite as logical to say that the indoor life of the women exposes them to some injurious influence derived from the dwellings. We know that the tubercle bacillus is apt to cling to ill-ventilated and insufficiently cleaned rooms inhabited by phthisical persons, conditions only too well fulfilled in the houses of the poor. It is hard to resist the conviction that these facts are most readily to be explained by the more prolonged exposure of the women to the risk of house infection. In towns the male death-rate from phthisis exceeds the female. The difference here, no doubt, depends on the unfavourable conditions under which men commonly work in rooms badly ventilated and dusty.

The great preponderance of the phthisis rate on the female side between the ages of ten and twenty, as shown by Ogle's tables, corresponding, as it does, with the period in which the outdoor life of boys and the indoor life of girls differ most widely, points in no uncertain manner to the dwellings as the source of the mischief.

Contagion.—The contagious nature of phthisis, long an article of popular belief in parts of Southern Europe, appears then to derive confirmation from Koch's discovery.

Since 1882 many cases have been published in support of this doctrine. In most of these the parties concerned were married couples; and the disease seems to have been communicated from husband to wife more frequently than in the reverse direction. We may briefly consider the ways in which contagion may possibly occur.

(i.) By the skin. Direct inoculation has been already discussed and shown to be a very exceptional occurrence.
(ii.) By the alimentary canal. Tubercle bacilli might be accidentally introduced into the mouth, as by kissing; or less directly, by the use of knives, forks, spoons, or drinking-vessels. The great rarity of primary disease of the tongue, oral cavity, and alimentary tract generally, and the comparative infrequency of primary tuberculosis of the mesenteric glands, except in children, negative this mode of infection.

(iii.) By the respiratory system. Although the bacilli are not given off in the breath, the possibility of their being expelled by coughing with small quantities of mucus or saliva must be admitted; but this accident cannot be regarded as playing an important part.

(iv.) By the generative system. Where the generative organs are tuberculous, it is possible that contagion may take place during sexual intercourse, in either direction; but the occurrence of primary tuberculosis of these organs in either sex is extremely rare. Direct contagion must be very uncommon, and it can have little bearing on the causation of the disease. It is possible that the bacillus or its spores may pass with the sperm cell to the ovum without infecting the mother, as is believed to occur in syphilis. This question will be again referred to under the head of heredity.

If the views already expressed as to the part played by dried sputum and tuberculous dust be correct, there is no necessity to invoke the supposition of direct contagion, which, in truth, stands on no firm foundations. Husband and wife living in the closest relationship and in the same rooms, are necessarily exposed to the same risks; although the member who spends most time in the infected rooms is more likely to contract the disease. If husband or wife be already tuberculous a fresh dwelling may be converted into a focus of infection, and the healthy one may indirectly acquire phthisis by inhaling tuberculous dust.

Heredity.—Phthisis has always been accounted one of the most hereditary of all diseases. Numerous statistics, dealing with this point, are at hand; but, seeing that some refer to parental inheritance only, while others include collateral influence also, and in view of the fact that information concerning collaterals is less likely to be precise, we may confine our attention more particularly to parental inheritance.

The extent to which parental heredity is manifested in the subjects of phthisis has been very variously stated, Portal rating it as high as 66 per cent, Louis as low as 10 per cent. We may, perhaps, regard 30 per cent as about the proportion in which, according to most investigators, a history of parental heredity can be obtained. It has been maintained that fathers transmit to sons more frequently than to daughters, mothers to daughters more frequently than to sons. But the statistics of Walshe, R. E. Thompson, and Wilson Fox do not support this assertion. Heredity is generally but not universally regarded as playing a more important part in females than in males. It is stated that more female than male patients give a history of phthisis in the parent; and that among all hereditary cases maternal is in excess of paternal inheritance. In some cases inheritance seems to have been derived from grand-
parents or great-grandparents, the parents having played the part of silent carriers of the disease. According to several observers, phthisis is manifested at an earlier age in those that evince an hereditary taint. After the age of twenty-five the acquired cases equal the inherited, and ultimately outnumber them. According to Dr. R. E. Thompson, a greater severity of form and a shorter duration of life characterise the hereditary cases; but the experience of Dr. C. T. Williams does not confirm this conclusion.

Enough has now been said to show that, after all the labour expended on this subject, no general agreement has yet been reached.

It is evident that the investigation of this question is exposed to many fallacies, a few of which may be mentioned. In the first place, many deaths of parents and grandparents may have been wrongly attributed to bronchitis, pleurisy, or pneumonia when the affection was really tuberculous. Against this, of course, in other cases death may have been erroneously ascribed to tuberculous disease. In dealing with a large number of cases these opposing fallacies will to some extent neutralise each other. A more important source of error depends on the undoubted fact that many ancestors reputed healthy have been the subject of arrested tuberculosis. Again, parents may not manifest signs of phthisis till after the death of some of their offspring from this cause.

In the case of heredity among collaterals—brothers and sisters, uncles and aunts, cousins—the same fallacies must arise, but with an important addition. In all families, but especially among the poor, the mortality of infants and young children is very high; and there can be no doubt that the existence of tuberculosis at this age is very largely overlooked, death being ascribed to marasmus, diarrhoea, bronchitis, or bronchopneumonia. On the whole, it seems that the tendency of the fallacies referred to would be to underestimate rather than to exaggerate the influence of heredity. The heredity of phthisis has received two widely different explanations. According to the prevailing opinion, it is not the disease itself that is inherited, but a disposition or tendency to acquire the disease when exposed to the necessary influences; the other view is, that the germ of the disease is directly communicated from the parent to the embryo.

The doctrine of hereditary predisposition has been assailed on more than one ground. In the first place, the existence of a peculiar bodily conformation in the children of phthisical families, the tuberculous and scrofulous diatheses so much insisted upon by some writers, has been called in question. It is admitted that some of the features described are often seen in persons suffering from phthisis, though it is believed that to a considerable extent they are attributable to wasting of the muscles and adipose tissue, or to enlargement of external lymphatic glands, and are, therefore, manifestations of existing disease. These objections seem to be justified; but the hypothesis of hereditary proclivity does not necessitate the assumption of a special bodily habit, and
the abandonment of this postulate does not materially weaken the position of those who hold to the doctrine of predisposition. It has been objected that the percentage of family inheritance reckoned up from phthisical patients does not truly represent the influence of heredity, and that the percentage should be compared with the incidence of the disease in healthy families. Moreover, it is suggested that what is inherited is not a special disposition to tuberculosis only, but a general delicacy or vulnerability to adverse conditions of all kinds. According to Beneke this vulnerability is connected with the relatively small size of the heart in such persons. Others again would explain the prevalence of the disease in certain families by the greater opportunities of infection that exist in the dwellings of such persons. The first objection may be admitted as valid; but in order to arrive at accurate conclusions on this basis the subject would require investigation on a much larger scale than has been hitherto attempted. As Dr. Kingston Fowler points out, it is obviously misleading to work back from the consumptive member of a family to the parents, and to deduce the influence of heredity from a comparison of the percentage incidence of phthisis in the children of the phthisical and non-phthisical—a method adopted by some investigators in this field. For this practically assumes that there is a consumptive in every family, and takes no account of the families in which, in many unselected series, no member is phthisical.

With regard to the explanation of heredity on the hypothesis of family infection, it seems that although this may account for many cases it will not explain all. Instances are not wanting where several members of a family, widely separated from one another, have manifested the disease in succession. If, however, the extreme latency of the tubercle bacillus postulated by some writers could be substantiated, the question of heredity would at once assume a new aspect altogether. Before proceeding to discuss the doctrine based upon this hypothesis, it may be pointed out that the existence of a family susceptibility to other infectious diseases—as to typhoid fever, scarlatina, and diphtheria—has long been recognised by epidemiologists.

To Cohnheim we owe the suggestion that heredity depends upon the direct transmission of the tuberculous virus to the embryo—a view which has been further developed by Baumgarten. This author holds that infection of the respiratory and digestive tracts will only account for a small proportion of the cases of tuberculosis; and by a process of exclusion he is led to the belief that heredity is the most potent factor in the continued existence of the disease. After rejecting the notion of hereditary predisposition mainly on the strength of arguments derived from the results of the experimental inoculation of animals, Baumgarten embraces the doctrine of the direct inheritance of the tubercle bacillus or its spores. According to his view the microbe may either be introduced through the placenta and thence infect the foetus through the umbilical vein ("placental infection"), or it may find access to the ovum itself either in the ovary or after its passage into the Fallopian tube ("germina-
tive or conceptional infection”). In the latter case the microbe would mostly be conveyed by means of spermatozoa, though an observation of Jani's suggests that the bacilli may enter the Fallopian tube from the peritoneal cavity. The possibility of germinative infection from paternal sources cannot be denied in view of the discovery, by Jani and Weigert, of tubercle bacilli in the healthy testes and prostate glands of phthisical men. Virchow objects to the view that germinative infection plays an important rôle in heredity, on the ground that the presence of the bacillus must interfere with or arrest the development of the ovum; but Baumgarten urges that this argument is negatived by the history of congenital syphilis, and by the analogy of the pébrine disease of silkworms. In the case of syphilis, although miscarriages may occur, it commonly happens that the child is apparently healthy at birth, and signs of the disease do not appear for some weeks; a period of latency, therefore, undoubtedly ensues between infection of the ovum or foetus and the first few weeks of extra-uterine life. In the pébrine disease, which is caused by a psorospermial organism, Pasteur has shown that the ova of the silkworm become infested with the spores of the parasite; but in spite of this the eggs are hatched normally, though the caterpillars ultimately succumb to the growth of the parasite in their bodies.

Baumgarten would explain the latency of the pébrine disease, congenital syphilis, and inherited tuberculosis by the supposition that the actively growing embryonic cells inhibit the development of the respective microbes.

Some interesting researches by Maffucci have an important bearing on this question. Tubercle bacilli from a tuberculous fowl were introduced into fertilised hen's eggs and incubation was allowed to proceed. Maffucci found that the bacilli did not multiply, but underwent a regressive change into granules exhibiting the staining reactions characteristic of the normal bacilli. The chick was hatched out in the usual way, but after about the twentieth day the bacilli began to develop, and a typical tuberculous infection ensued, the liver being conspicuously involved. If the dose of the bacilli introduced be small no visible tubercles form, but the chicken nevertheless dies of extreme marasmus and bacilli are found in the organs in small numbers. The analogy suggested with congenital syphilis, the pébrine disease, and congenital tuberculosis of fowls is both interesting and instructive.

Placental or germinative infection may explain the rare cases in which tuberculosis is found in the foetus or new-born infants; and also perhaps the less uncommon instances where the disease arises during the first few months of life. But there seems to be no sufficient reason for the belief that the tubercle bacillus or its spores may remain dormant from the time of conception of the ovum to adult or middle life. Baumgarten would go even farther, for he applies his hypothesis to explain atavism occurring in tuberculous families; and would trace the inheritance of the microbe to a grand-parent or even more remote ancestor when the parents have remained healthy. The evidence in favour of
Baumgarten's hypothesis is not strong, and is mainly drawn from observations on animals. Fœtal tuberculosis has now been demonstrated in several cases in calves, but in man such an occurrence is extremely rare. Landouzy and Martin have published a case where the apparently healthy fœtus of a phthisical mother proved capable of infecting animals with tuberculosis, to show that tubercle bacilli may be present in the tissues without exciting any manifest lesion. The hypothesis of direct inheritance does not appear to be reconcilable with the facts disclosed by Ogle's statistics. A reference to his table shows that the mortality from phthisis declines greatly after the completion of the second year until the tenth year, when it begins to rise again, attaining its maximum from twenty-five to thirty, but maintaining a high level up to the age of sixty-five. Moreover the marked difference in the incidence of the disease on the two sexes between the ages of ten and twenty is quite inexplicable on Baumgarten's theory.

The only conclusion at present warranted is that direct inheritance is of decidedly subordinate importance to extra-uterine infection, however acquired.

Pathological Anatomy.—Tuberculosis is in its origin a local disease depending on the lodgment and growth of the tubercle bacillus; but in virtue of its infective character it not only extends by continuity from the primary lesion, but it tends also to invade other parts of the body.

Fever and other constitutional effects of tuberculosis are often out of all proportion to the extent of the local disease, and must be ascribed to the circulation in the blood of some as yet unrecognised chemical poison produced by the bacillus.

We have now to consider the changes in the lungs that result from the invasion of the tubercle bacillus. The initial lesions exhibit certain differences according to the manner in which the microbe is introduced into the organ. Excluding the comparatively few cases in which the pulmonary disease is due to direct extension from neighbouring lymphatic glands, or from the osseous parietes of the thorax, it may be said that the bacillus gains entrance in one of two ways, through the blood-vessels or through the bronchial tubes. In the former case the entry of a large number of bacilli into the circulating blood gives rise to an eruption of miliary nodules disseminated through the whole lung, and through many organs of the body. In such cases, as was first pointed out by Buhl, a caseous focus will almost invariably be found in some lymphatic gland; or, possibly, in the lung itself. It is probable that the introduction of a small dose of the bacilli may have as its result a circumscribed lesion of the lung. In either case infection is brought about by an embolic process, the microbe being arrested in the alveolar capillaries. The presence of the bacilli in the first instance provokes a specific cellular growth in the capillary wall, but the process soon extends into the cavity of the air sacs, where a similar cell growth develops. If the microbes enter the lung through the air-passages they appear to become arrested in
the terminal bronchioles or alveoli, in which parts the epithelium is not ciliated. From the bronchiole the cell growth invades the peribronchial sheath and alveolar cavities, the result being an aëret of peribronchitis and broncho-pneumonia. Tuberculous growths, wherever situated, are devoid of blood-vessels.* In generalised miliary tuberculosis the pulmonary changes are but a part of a general infection of the body, though the lung may suffer most. As death results in a few weeks at the latest the tubercles in the lungs have not time to go through the usual cycle of changes, manifested in the cases which run a more chronic course.

Inasmuch as the lesions of chronic tuberculosis differ in degree rather than in kind it will be convenient to study the process in the chronic form. We have seen that in primary tuberculosis of the lung the bacilli are probably introduced as dust with the air. Since the time of Louis Pasteur the preference of tuberculosis for the apex of the lung has been universally recognised; the earliest lesions are found about one to two inches below the extreme apex. In rare instances the disease begins in other parts of the lung, as at the base of the lower lobe; but in adults a primary basic origin is exceedingly rare, and is probably not found in more than one in 400 or 500 cases: in children it is relatively less infrequent, but this is due to the fact that in them primary tuberculosis of the bronchial glands is more common and attains to greater proportions than in adults. Many cases of tuberculosis in children apparently basic in origin are really due to direct extension from caseous bronchial glands. In rare cases of irregular localisation, whether in children or adults, the disease has originated in the vertebral. The special proclivity of the apex of the lung to tuberculosis has been variously explained: this part of the lung undoubtedly possesses a smaller range of movement than the lower portions in consequence of the greater rigidity of the upper ribs, and this condition must favour the retention of foreign matter in the bronchial tubes and alveoli, and will thus favour the lodgment of the bacilli. Moreover, Dr. R. E. Thompson points out that the comparatively fixed position of this part of the thorax tends to keep the bronchial tubes of the apex widely open, whereby the entrance of dust and other extraneous matter is promoted. But in generalised miliary tuberculosis also, where the bacilli enter the lung through the blood-vessels, the lesions are often most advanced at the apex, a fact which points to the existence of a special vulnerability of this part of the lung itself. It has been stated that the circulation in the apex is less vigorous than in other parts of the lung, and that this part of the lung being drier is more susceptible, but of these opinions there is little direct proof. At an early stage of the disease the lesion will be found to consist of one or more small grayish nodules, the centre of which corresponds to a bronchiole; as these nodules increase in size they tend to acquire a racemose shape owing to the growth of miliary granulations at the periphery. In man it is not easy, as a rule, to trace the earliest steps of the process in the primary nodule, as before the patient's death regressive changes have already set in; but from a study of the secondary nodules developed in similar cases we may
conclude, as Rinnfleisch has long taught, that the process begins in a terminal bronchus and thence spreads to the corresponding lobule—that is to say, the lesion is essentially broncho-pneumonic. In the early stages

![Diagram](image-url)

**Fig. 6.**—Composite photograph taken from three different sections, illustrating the development of the tubercular process. (Low power.) 1. Inlet of tubercular perbronchitis; a bronchiule with surrounding tubercular infiltration. 2. Patch of tubercular "pneumonia." a. Early stage—aizeu contained large pale epithelioid cells (not recognizable with so low a power); b. Later stage—alveoli contain granular necrotic masses, the outlines of the cells having disappeared. 3. Fibro-caseous tubercular nodule, showing a pale amorphous caseous centre embedded in concentrically arranged fibro-cellular connective tissue, which contains a few giant cells in its inner zone. Coarse black carbon particles in places.

we find the mucous membrane of the bronchiule swollen and infiltrated with cells, and the surface more or less denuded of epithelium. The cavity of the tube contains mucous secretion mixed with pus cells. Tubercle bacilli may sometimes be recognised in the secretion as well as

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in the cellular infiltration. A similar cell growth, with a varying number of bacilli, is found in the peribronchial sheath and in the corresponding alveoli. The tuberculous growth, whether in the bronchile or in the air-sacs, is seen to consist at first mainly of cells of an epithelial type—epithelioid cells. In some cases the nodule seems to be composed exclusively of small round cells. Subsequently large multinucleated cells ("giant cells") appear singly here and there in varying numbers. At the periphery of the tuberculous area a zone of small lymphoid cells can generally be recognised. In the last-named region the cells are entangled in a scanty meshwork of delicate fibres; but in no cases, save the most chronic, is fibrillation visible at the centre of the tubercle. By degrees the cellular growth extends to neighbouring lobules, where the same process is enacted; and thus the original nodule may become more or less lost in a diffuse infiltration or tuberculous pneumonia. The direct propagation of the disease is brought about by the spread of the tubercle bacilli along the lymph spaces and vessels in the interstitial tissue of the lung. In the course of the tuberculous process the walls of the alveoli and, in a lesser degree, the coats of the small arteries and veins become involved in the cell growth. Perforation of the wall of a pulmonary vein and entrance of the bacilli into the blood is, as was first shown by Weigert, one of the commonest modes of general infection of the body in cases of pulmonary tuberculosis. A small artery may be affected in like manner, and a localised eruption of miliary tubercles take place in the lung in the area of distribution of the affected vessel; but in most cases extra-alveolar lesions are of quite subordinate importance to the changes occurring within the air-sacs. In the most chronic varieties of tuberculosis the development of fibrous tissue is the predominant feature; and this leads to a thickening of the interalveolar, peribronchial, interlobular; and subpleural connective tissue. Tubercle bacilli are found in and among the epithelioid cells, in the giant cells, and occasionally in the small-celled area; but not in the fibrous tissue itself. Except in miliary tuberculosis, and in certain instances of acutely spreading disease, the number of the microbes that can be demonstrated is generally small, and contrasts strongly with the large numbers met with in the artificial tuberculosis of animals. The paucity of the bacilli in the former case agrees with the far more chronic course of the disease in man; but it may be, as Ehrlich suggests, that in certain stages of their growth the bacilli cannot all be successfully stained by our present methods.

The destiny of the tuberculous growth is twofold. In the first place, the cells undergo necrosis; their outline becomes indistinct, the nucleus disappears, and the cell is converted into a finely granular or hyaline mass, which then becomes fused with neighbouring cells in a similar state of degeneration. As a result of this change large areas of the affected organ become transformed into opaque, yellowish white material, resembling cheese—a result known as caseous degeneration, or necrosis. The cause of this change is uncertain, but it may depend, as Professor Watson Cheyne suggests, on some chemical poison elaborated by the
bacilli. Caseation is regarded by Weigert as an instance of coagulative necrosis. Cheesy foci may remain unaltered for a considerable time; but they are very liable to undergo liquefaction, and when a communication is established with a bronchus the softened material is evacuated and a cavity or vomicus is formed. In some cases the process of softening and excavation originates in a small bronchus. Tubercle bacilli are found in immense numbers in the cavities, but in caseous material they are generally very scanty. Cavities also contain various pyogenetic cocci; but, according to some observers, suppuration may be excited not only by such cocci, but also by the tubercle bacillus. A caseous or necrotic change is probably never altogether absent at some period in the development of any tuberculous formation, and, as a rule, this is the prevailing feature; but at the same time, in most cases of pulmonary tuberculosis in man, and in all chronic forms without exception, another process of a conservative or reparative character is recognisable in the shape of a growth of connective tissue. This change begins at the margin of the tuberculous area, where it constitutes a species of fibrous capsule. It is doubtful whether this be a direct result of the tuberculous process, or whether it be attributable to reactive inflammation around the tubercle. In some cases the central caseous mass becomes thus shut off from the surrounding parts and complete arrest of the disease is effected. The cheesy matter may subsequently become calcified, or it may be gradually permeated by connective tissue and converted into a solid fibrous knot. Fibrosis and encapsulation are the natural mode of healing. The well-attested frequency with which old fibrous tubercles or calcareous nodules are found in the lungs at post-mortem examinations of patients dying of other diseases, shows that recovery from tuberculosis is by no means so rare as was formerly supposed. In the great majority of cases, however, the fibrous change is more limited, indications of a capsule can scarcely be recognised, and fresh islets of tuberculous disease spring up on the confines of the original patch. These secondary foci, which depend on the spread of the bacilli along the lymphatic spaces, go through similar stages of cell growth, necrosis, and connective tissue development. According as the necrotic or fibrous change preponderates, the case assumes an acute or chronic complexion; but the combinations of the two processes are subject to infinite variety. Thus a case originally of a marked fibroid type may be complicated by the occurrence of acute destructive disease in other parts of the lung; or, again, though this is far less common, rapidly progressing tuberculosis may undergo partial arrest and pass into a chronic fibroid stage. In ordinary cases we find some indications of healing at the apex, while in the more recent lesions necrosis is the predominant factor.

Histogenesis.—The origin of the cells constituting the tubercle has long been a matter of dispute; some authors maintain that they are the result of the proliferation of fixed tissue cells, others regard them as leukocytes that have escaped from the vessels.

An important experimental research by Baumgarten has gone far to
prove that the specific tubercle cells are epithelioid in type, and are the
offspring of the epithelial, endothelial, and connective tissue cells of the
body. In miliary tuberculosis of the lung—the process begins with
swelling and nuclear division of the endothelium of the alveolar capillaries,
of the epithelial cells lining the alveoli and bronchioles, and, in a minor
degree, of the interstitial connective tissue cells. For a fuller considera-
tion of this point and for further histological details the reader is referred
to the article "Tuberculosis" (vol ii. p. 6). Although in the case of
miliary tuberculosis the bacilli reach the lung through the blood-stream
and become arrested in the capillaries, yet the earliest lesions consist
predominantly of an accumulation of epithelioid cells within the air-sacs;
and miliary tuberculosis of the lungs has not inaply been designated a
miliary pneumonia.

For simplicity's sake the development of the disease has been sketched
as it affects individual sections of the lung; but as a matter of fact the
process is far more complicated. While separate foci of tubercle go
through the stages just described, various secondary changes of a congestive,
inflammatory, and edematous nature commonly ensue in the intervening
portions of lung. These conditions are partly the result of compensatory
hyperæmia; but in the main they depend on obstruction of the smaller
bronchi and on the lobular collapse that follows.

Collapse soon passes into broncho-pneumonia, which, if not from the first
actually tuberculous, soon becomes so. In such cases scattered caseous
spots are seen embedded in reddish, solidified lung. The pneumonic
condition, at first, is usually patchy or lobular in distribution; though
from the coalition of numerous individual foci the consolidation may
ultimately involve the greater part of one lobe. On section the surface
is moist, flat, and glazed; seldom dry and granular, as in croupous
pneumonia: this difference depends on the fact that the exudation is
mainly composed of cells and edematous fluid, and contains little fibrin.

In some instances the consolidation has a pale pinkish-gray gelatinous
appearance, the "gelatiniform infiltration" of Laennec. This condition
seems to be due to the existence of marked anæmia and oedema in
addition to the alveolar catarrh and collapse. In such infiltrations it is
not uncommon to discover small specks of caseous necrosis, which stamp
the process as essentially tuberculous. Sometimes a large area or even
a whole lobe presents a more or less uniform grayish or yellowish con-
solidation known as caseous pneumonia. In such cases, as a rule, old
caseous foci, or a cavity, will be found at the apex, suggesting the sec-
ondary nature of the diffuse infiltration.

Acute croupous or lobar pneumonia is stated by many authors to be a
common termination of pulmonary tuberculosis. I am convinced that this
is an error. An experience of some thousand necropsies of cases of this
disease has not furnished me with more than one undoubted instance in
which progressive tuberculosis of the lung was complicated by acute
fibrinous pneumonia. During the first influenza epidemic, two or three
cases, in patients who succumbed to an acute pneumonia, of more or
less lobar dimensions, revealed an oedematous, ill-defined consolidation, consisting microscopically of cells and oedematous fluid without any fibrin; these may have been modified instances of acute lobar pneumonia, but with these few exceptions the above statement holds good.

Phthisical patients are indeed often cut off by acute intercurrent disease of the lower lobes; but this is essentially broncho-pneumonic, and probably depends on the inhalation of septic microbes from ulcerative cavities in the lung.

In the more chronic cases a localised emphysema is not uncommon, especially where contracting lesions are separated by tracts of unaltered lung. This condition, which is most pronounced towards the apex and anterior margin of the upper lobe, may be so extensive as almost to mask the original disease. The affected lung may present the appearance of large superficial bulle, or the form of emphysema may be more diffuse. The surface of such portions is often puckerét from the contraction of subjacent fibrous patches or cavities. Emphysema in these circumstances is compensatory, and results from obliteration of adjacent alveoli. It is necessary to distinguish clearly between true emphysema, which is a degenerative atrophy of the alveolar walls and capillaries, and what may best be described as pulmonary distension. When one lung is contracted, the opposite lung undergoes vicarious enlargement, the alveoli becoming uniformly enlarged without being otherwise altered; the expanding lung passes across the middle line of the sternum and encroaches upon the space formerly occupied by its fellow. The effect of this enlargement is an increase of alveolar surface, and consequently an improved aeration of the blood. This condition has been named "hypertrophy of the lung," a description which implies increased function, and is therefore strictly correct. It is possible, however, that this condition may, in time, pass into true emphysema.

Cylindrical dilatation of the smaller bronchi is not uncommon, and may be found in any part of the lung, whether its texture be spongy or indurated. Bronchiectasis is to be attributed mainly to the positive expiratory pressure of cough acting on the bronchial walls softened by inflammatory or other changes. The existence of contracting fibrous tissue in the surrounding lung, by drawing apart the walls of the bronchi, will further contribute to the dilatation.

In all chronic cases pigmentation is a more or less marked feature; it depends mainly on the deposit of particles of carbon derived from the atmosphere. Old fibroid lesions have a blackish or slaty colour, which contrasts sharply with the red, yellow, or grayish tint of other parts of the lung.

The process of softening of the caseous material and the formation of cavities have been already briefly alluded to. The liquefaction which occurs has been likened by Duclaux to the ripening of cheese; but the nature of the chemical transformation is still unknown. The shape of pulmonary cavities varies greatly. They may be rounded or oval; but more often they are sinuous or anfractuous, in consequence of the
coalescence of separate vomicae, and of the irregular extension of the excavating process. Cavities are often traversed by tough septa and bridles, and are then described as trabeculated. The trabeculae were formerly said to consist of persistent bronchi and blood-vessels, but they have been shown by Dr. William Ewart to be chiefly composed of condensed airless lung, representing the remains of collapsed alveolar tissue originally separating discrete cavities. The ridges and stumps often observed on the walls of vomicae are relics of trabeculae destroyed by ulceration.

In acutely developed and extending cavities the wall is ragged, and formed by soft caseating or necrotic material. Such cavities are commonly filled with thick pus, their walls softened and in a state of purulent infiltration. Chronic and quiescent vomicae are lined with a definite pyogenic membrane like that of a chronic abscess. The lung tissue around may be indurated or simply collapsed; less frequently spongy or emphysematous. Extension usually takes place by slow ulceration of individual cavities, which tend ultimately to coalesce; but in some cases acute suppuration and sloughing cause rapid destruction of the lung. Excavation sometimes begins as a tuberculous bronchiolitis, ulceration subsequently extending through the thin bronchiolar wall to the surrounding alveoli. In other cases a dilated bronchus may undergo secondary ulceration and become sacculated. It may be very difficult to decide whether such cavities were originally bronchiecatic or pulmonary. True bronchiecatic cavities are seldom very large, whereas those of pulmonary origin may involve the greater part of a lobe, or even the whole of one lung. Excavations of this magnitude are always the result of fusion of several cavities. In the great majority of cases excavation originates in the lung, and is not bronchiecatic. A fuller treatment of this subject is to be found in the article on "Bronchiecasis" in the present volume [pp. 59 and 74].

In the course of excavation the bronchi become ulcerated and eaten away, so that ultimately their wall passes insensibly into the lining membrane of the vomica. Cælitisisation may cause narrowing or virtual obliteration of the bronchial orifices. Chronic cavities not uncommonly undergo a considerable reduction in size, as the result of contraction of their capsule or of the neighbouring lung. It has even been asserted that they may close completely, but there is no proof of this, and such an event, in view of the imperfect removal of the secretions effected through the bronchi, must be regarded as highly improbable. A vomica resembles a chronic abscess discharging externally through a narrow sinus; now unless the abscess can be freely opened it will not granulate up thoroughly, and will continue to secrete for years. Contraction of a cavity is, however, promoted by a spongy yielding condition of the adjacent lung.

Fibrosis is an essential feature in all chronic excavation, and, as we have seen, the same change is always present in chronic tubercular consolidation. In the most pronounced examples of this condition fibroid induration is associated with excavation. In either case fibrosis caus-
shrinking of the lung, the upper lobe as a rule being most affected; but at times the whole lung becomes uniformly contracted. In extreme cases the lung may be reduced to the size of a man's fist. Contraction of the lung is followed by elevation of the diaphragm and of the abdominal viscera, displacement of the heart and mediastinum to the affected side, and a varying amount of depression of the chest wall. When the lung is not too firmly adherent to the ribs, a contracting cavity at the apex may shift slightly outwards and backwards towards the fixed point, the root of the lung, as shown by Dr. C. T. Williams.

The primary cavity is situated at the apex of the upper lobe. Secondary cavities may be formed in any part of the lung, but Dr. William Ewart pointed out that excavation is especially prone to attack a definite region, the apex of the lower lobe, and at a date anterior to the implication of the lower part of the upper lobe. The base and anterior border of the lower lobe are least prone to excavation, just as these parts are the last to be involved by the disease.

It remains now to consider the mode in which the tuberculous process extends through the lungs. In generalised miliary tuberculosis, where infection, for the most part, is derived from a caseous lymphatic gland—that is, from a source external to the lungs—the pulmonary blood-vessels are flooded with bacilli, and the lungs become stuffed with miliary granulations from apex to base. In chronic pulmonary tuberculosis the lungs become gradually but progressively invaded by a process of auto-infection, the primary focus being situated at the apex of one upper lobe. In a moderately advanced case we find one lung more diseased than its fellow, and towards the apex of the former a cavity or cavities with tough walls, the tissue around being pigmented and fibroid, and often containing some caseous nodules. In the lower part of the same lung we see scattered tuberculous nodules and masses, some softening to form small cavities. The other lung presents lesions of a similar appearance and localisation, but in a less advanced stage. It sometimes happens that the disease becomes partially arrested in the lung first attacked, while in the lung secondarily involved it extends progressively from apex to base.

It cannot fail to strike the observer that the secondary lesions in the lung are not the result of direct extension by continuity from the apex, for the individual foci are separated by tracts of healthy lung tissue. Nor is it possible to believe that tuberculosis spreads exclusively or mainly by lymphatic or vascular channels; for, in cases where the disease is not too advanced, the lesions often consist solely of a cavity at the apex of the upper lobe surrounded by a zone of tuberculous infiltration, and some caseous masses of tubercle at the apex of the lower lobe; the rest of the lung being unaffected. Extension to the lower lobe is evidently effected through the bronchial tubes, infective secretion being inhaled from the apical cavity into the bronchi of the lower lobe. This view is in harmony with the results of the “inhalation tuberculosis,” artificially produced by exposing animals to a spray of tuberculous sputum. It also accords with the fact that the prevailing lesions in man are broncho-pneumonic in char-
acter. Dr. Ewart explains the marked proclivity of the apex of the lower lobe to secondary excavation by the fact that the bronchus supplying this part is a wide, straight tube coming off horizontally from the main bronchus, a condition which appears to favour the inhalation of infective secretion from cavities in the upper lobe. Dr. J. K. Fowler also has pointed out that the distribution of tuberculous disease follows a very definite path. From the initial lesion at the apex the process spreads downwards in the upper lobe. Excavation of this region is followed by secondary disease of the apex of the lower lobe on the same side, and of the apex of the upper lobe of the opposite lung. Dr. Fowler states that the former district is involved before the latter; but in my experience the apex of the opposite lung is quite as often the first point to be affected with secondary disease, though the apex of the lower lobe of the lung primarily attacked is almost always implicated at an early date. The lower part of the upper lobe is then gradually infiltrated, and simultaneously the disease extends from the apex of the lower lobe forward and downward along the interlobar septum. The base and anterior border of the lower lobe are the last parts to be affected.

In the process of destruction blood-vessels for the most part become obliterated as the result of thrombosis; but when rapid excavation is taking place, ulceration may extend into large vessels and cause severe haemorrhage. In cases of a more chronic nature it is not uncommon to find aneurysmal dilatation of branches of the pulmonary artery lying in the walls of a vomica. In my post-mortem examinations aneurysms were found in 15 per cent of all cases of pulmonary tuberculosis; these aneurysms consist of a lateral expansion of the vessel on its exposed side. In rare instances an artery crossing a cavity becomes uniformly dilated to form a fusiform aneurysm. In either case the dilatation is to be attributed to two causes: (i.) to arteritis and softening of the arterial coats, the result of extension of inflammation from the cavity; (ii.) to withdrawal of support from the wall of the exposed vessel. Pulmonary aneurysms vary in size from that of a pin’s head to that of a plum, but they are seldom larger than a cherry. It is usual to find only one aneurysm; though, at times, several may be discovered in the same cavity or in different parts of the lung. In one extraordinary case I found twenty-two aneurysms in one lung. Rupture of the sac is a common event, and is by far the most frequent cause of profuse haemorrhage. In a series of eighty cases of fatal hemoptysis, examined by myself, a ruptured aneurysm was found in seventy. When rupture does not occur, thrombosis is apt to ensue. Thrombosed aneurysms are often met with in cases where no haemorrhage has taken place. Observation shows that aneurysms after leaking for some time may become ultimately cured by coagulation of their contents. When the cavity containing the aneurysm is small, the pressure of the effused blood may be sufficient to prevent further haemorrhage. If the patient live long enough, the healed aneurysm in time undergoes necrosis, and may entirely disappear.

Localised gangrene occasionally takes place in connection with rapidly
spreading excavation. It is, however, a remarkable fact that, in spite of the existence of numerous profusely secreting cavities, putrid changes are very rarely met with as a result of tubercular disease.

Pleurisy is a well-nigh constant accompaniment of the pulmonary disease, and is mostly due to extension. Pleurisy may also be consecutive to peritonitis, the virus being transmitted from one serous cavity to the other through the lymph spaces of the diaphragm. Primary tuberculosis of the pleura is said to occur, but of this there is some doubt. In cases of apparently primary pleural origin the disease may have started in a small caseous bronchial gland which has escaped detection.

Fibrinous exudation is the commonest form, but sero-fibrinous effusion often ensues. Empyema is uncommon in adults, though less rare in children. Hæmorrhagic exudation is occasionally met with, and may be attributed to rupture of the newly-formed capillaries of the inflamed pleura. In many cases tuberculous granulations and, less frequently, caseous nodules can be recognised in the serous membrane. But it is not infrequently impossible to discover any naked-eye signs of tubercle, whether in cases of fibrinous, sero-fibrinous, or suppurative pleurisy. In some instances of this description the microscope may reveal the presence of isolated miliary tubercles in the thickened pleura. There can be little doubt that the granulations in the pleura, as in the peritoneum, may undergo complete fibrous transformation. It is not improbable that in some instances pleurisy may have a non-tuberculous origin. In any case the ultimate result of pleurisy is to cause more or less thickening and adhesion of the pleura. The former may attain to considerable dimensions in chronic cases, especially at the apex of the lung, where the pleural investment may measure as much as an inch in thickness.

Rapid softening and excavation of the peripheral parts of the lungs are apt to cause perforation of the pleura and entrance of air into the serous cavity, if the pleural space at the affected spot have not previously been obliterated by adhesions.

Pneumothorax causes collapse of the lung, and is followed in most cases by effusion of serous or, more often, of purulent fluid in consequence of the entrance of tubercle bacilli and pyogenic cocci from the lung.

It is not unusual to discover more than one perforation of the pleura. The opening may be situated at any point where the pleural surfaces are not adherent. The middle third of the lung corresponding to the lower part of the upper lobe and upper part of the lower lobe is the most frequent site of perforation. Occasionally the air escapes into the subcutaneous tissue of the chest wall or into the mediastinum, and surgical emphysema is produced. At times a cavity in the lung may extend outwards through the pleural adhesions and give rise to emphysema, or to an abscess in the chest wall communicating with the lung. Pneumothorax was found in 11 per cent of the cases of phthisis which I examined after death.

The bronchial, mediastinal, and tracheal glands are very often the seat of secondary tuberculous deposit. They may also be primarily affected, and, as already mentioned, the disease may extend thence to the lung or
pleura. The extreme frequency with which arrested tuberculous lesions, in the shape of calcareous nodules, are found in these glands is well known to all who are in the habit of making necropsies.

Stenosis of a main bronchus is occasionally caused by enlarged glands in children; but this very seldom occurs in adults, as their bronchial tubes are much firmer. The smaller bronchi may be compressed in adults as in children. Marked obstruction entails some degree of collapse of the lung, and sometimes gives rise to bronchial dilatation beyond the seat of pressure. In one case I found that a large calcareous bronchial gland had perforated the bronchus and set up ulceration, which had extended at another point into a larger branch of the pulmonary artery.

Suppurating caseous bronchial glands may perforate the trachea, bronchi, lung, oesophagus, or pericardium. Sudden death has more than once resulted from the entrance of a caseous gland into the trachea. In cases where a fistulous communication is established between the oesophagus and the air-passages a septic broncho-pneumonia ensues, and pulmonary gangrene has been a relatively frequent complication.

From the foregoing sketch it will be seen how manifold are the lesions of phthisis pulmonalis. The unity of phthisis, that is to say, the essentially tuberculous nature of the disease first advocated by Laennec, was long and vehemently disputed; but the truth of this doctrine was at length removed from the sphere of controversy by Koch's discovery of the tubercle bacillus. The presence of the specific microbe in miliary granulations, caseous nodules, caseous pneumonia, and pulmonary cavities supplies a positive demonstration of the pathological identity of these apparently different manifestations. Hence such distinctions as tuberculous, pneumatic, tuberculo-pneumonic, catarrhal and scrofulous phthisis, always artificial and unworkable, are now entirely superfluous. Phthisis is tuberculous disease of the lungs.

SYMPTOMS.—The manner of invasion of pulmonary tuberculosis varies somewhat in different cases. From the slowness of growth manifested by the tubercle bacillus we might expect the invasion of the disease to be gradual. In the great majority of cases this is the case, and certain general or constitutional symptoms often precede those of local disorder of the respiratory organs. But as in a considerable number of cases the disease begins more or less abruptly, we must distinguish (A) acute, and (B) chronic tuberculosis.

A. Acute pulmonary tuberculosis.—Three forms of the acute disease may be recognised.

1. Lobar-pneumonic form.—In this form—the rarest of the three—the whole of one lobe, nearly always the upper lobe, or the greater part of one lung becomes converted into a solid gelatinous or caseous substance. The consolidation, though massive, usually presents some scattered foci of older date, suggesting that the diffuse pneumonia is secondary to an originally localised form of tuberculosis.

This sequence of events is well illustrated by cases where a cavity
exists in the apex or other part of the lung, under which circumstances the diffused infiltration may be attributed to the inhalation of infective secretions from the cavity. But in a few recorded instances the caseous infiltration has been perfectly uniform, which observations support the belief that the affection was, from the first, lobar and acute, all parts having been simultaneously and equally attacked. In some of these cases there was a cavity in the lung which may have been the starting-point of the pneumonia. Miliary tubercles may sometimes be discovered in the lower lobe or in the opposite lung; caseous nodules are more common. Tuberculous pleurisy, mostly of the dry variety, is a constant accompaniment.

The disease may begin sharply with a rigor, high fever, dyspnoea, pleuritic pain, and a short cough with mucoid, tenacious sputum, which may be rusty or may contain florid blood. Occasionally the attack begins with hæmoptysis. Herpes labialis is not uncommon. The patient often attributes his illness to a chill.

The foregoing mode of invasion closely simulates acute pneumonia. In other cases the onset may be less abrupt, the patient experiencing a malaise, aching in the back and limbs, and slight cough and expectoration, before the onset of marked pyrexia and other pneumonic symptoms. Physical examination discovers signs of consolidation, dulness, tubular breathing, crepitant or subcrepitant râles, bronchophony and increased tactile vocal fremitus. The breath-sounds may be merely weakened, and no tubular breathing may be heard for some time. Pleuritic friction is frequently met with; signs of effusion are somewhat rare. The whole picture is that of acute pneumonia, for which the disease is almost invariably mistaken at first. But no crisis appears, and the fever persists for weeks. In a few instances the temperature becomes lower, and after a few days the symptoms abate somewhat; but the improvement is only short-lived, and the patient relapses into his former condition. The fever for the first two or three weeks manifests a remittent character, the evening temperature being one or two degrees higher than the morning, and ranging from 103° to 104° F. Later the temperature falls somewhat and assumes a hectic character. From the first the patient wastes rapidly and exhibits extreme prostration, sometimes passing into a typhoid state with dry tongue, subsultus, and mild delirium. In the less rapidly fatal cases signs of excavation of the lung gradually come on. The sputum becomes muco-purulent, and is found to contain tubercle bacilli, and perhaps elastic tissue. A fatal termination may be reached in less than a fortnight; more often life is prolonged for six weeks or two months. Now and then the disease gradually loses its acute character and assumes the form of chronic pulmonary tuberculosis.

The diagnosis during the first week or ten days presents great difficulties. In some instances the invasion is less sudden, and the severity of the symptoms less pronounced than in cases of acute lobar pneumonia. But these distinctions are often wanting. In the tuberculous form the fever is less continuous, and is generally marked by irregular remissions.
The pulse-respiration ratio, again, is less deranged than in acute pneumonia; for the pulse-rate is greatly increased, often reaching 130 to 140, with a respiration of 30 or 40.

It has been said that in tuberculous cases the breath-sounds over the affected lobe are more often weak and suppressed than tubular, but this sign is by no means constant; moreover, this sign is not very rare in croupous pneumonia. From acute pneumonia with delayed resolution the disease may be discriminated by the progressive wasting and prostration, as well as by the fluctuating high temperature which accompanies it; for in the former complaint, in spite of the persistent pulmonary consolidation, the general condition mends and the temperature falls. In doubtful cases the appearance of signs of excavation, and, above all, the detection of tubercle bacilli in the sputum, are the only facts on which a positive diagnosis can be based. The complications of this form of tuberculosis do not differ materially from those attending the chronic variety, under which head they will be discussed; but it may be said that complications are much less frequent in acute cases, owing to the rapid termination entailed by the severity of the pulmonary lesions.

II. Broncho-pneumonic form.—This form, which is much less uncommon than the last, represents what has been called galloping consumption or phthisis florida. The special anatomical features consist of disseminated tuberculous foci, of various sizes, which may be soft, yellowish white, and cheesy; or grayish, slightly pigmented, of racemose shape, and somewhat indurated. Miliary tubercles are seldom to be seen. In most cases rapid softening and excavation of the nodules is a very prominent feature. Small suppurating cavities with soft ragged walls are scattered through both lungs. In the apices of the upper lobes the cavities are generally larger, and in some cases the apex is the seat of old fibrosis and excavation. The lung tissue separating the nodules is often hyper-inflated, especially towards the anterior borders; in other parts the nodules are embedded in tracts of grayish red consolidation, more particularly towards the back. This fusion of the individual foci may ultimately result in a diffuse infiltration of lobar dimensions. The bronchi are always deeply injected and contain abundant purulent secretion. The localisation of the lesions is essentially broncho-pneumonic and lobular, and depends on the inhalation of tubercle bacilli from a cavity in the lung or from external sources. Pleurisy in some form, whether dry, sero-fibrinous, or sanguineous, is always present. The larynx and large air-passages are more prone to tuberculous ulceration than in the lobar-pneumonic form, in consequence of the more profuse secretion discharged from the cavities and bronchi in the present variety.

The mode of onset is subject to considerable variations. Occasionally without any early period of ill-health the patient is suddenly seized with rigors and other symptoms of acute pneumonia: more often the disease begins insidiously with a cough, which, after the lapse of a few weeks, is succeeded by fever, malaise, and other constitutional symptoms. Hæmoptysis is occasionally the first symptom. In some instances the disease
begins with symptoms of gastric disturbance, loss of appetite, furred tongue, and vomiting; and the real nature of the malady is not recognised until the chest is examined.

In recent years this form of tuberculosis has not uncommonly followed an attack of influenza. Whatever the mode of invasion, marked wasting and loss of strength soon appear. Hæmoptysis is not very common, and is seldom profuse. The sputum at first is muco-purulent, but it soon becomes more puriform, and sometimes acquires a greenish yellow colour; in some cases it has a reddish brick-dust colour for weeks. Tubercle bacilli and elastic tissue are generally recognised before long. Night sweats are frequent, and often are very profuse. The temperature ranges high, reaching 104° F. at times; the fever is fluctuating, being marked by morning remissions of one or two degrees; as the disease proceeds, the temperature becomes more hectic. Anorexia, vomiting, aphthous stomatitis, a dry red tongue, and diarrhoea are very common, and the patient is apt to pass into a typhoid state. In the worst cases death ensues in three or four weeks, but the end is more often deferred for three or four months. Very occasionally the acute progress of the disease is stayed, and the patient lingers on for eight or nine months.
Physical examination at first reveals nothing more than signs of general bronchitis; but subsequently pleuritic friction and patches of dulness on percussion, more particularly at the apices, make their appearance, and signs of excavation may ultimately be discovered. In some instances the signs may predominate at the base of the lower lobe. In the most acute cases no cavernous signs can, as a rule, be recognised, as death takes place before the cavities have reached sufficient size to permit of their detection. Moreover, the patches of distended lung tissue which separate individual foci tend to obscure the existence of extensive disease. The diagnosis at first rests on the discovery of physical signs of bronchopneumonia, accompanied by great prostration and loss of flesh. But the detection of tubercle bacilli in the sputum may alone enable us to decide whether the disease be tuberculous or not. In the case of young children the differences are greatly enhanced, for no sputum is obtainable, and death commonly takes place before softening and excavation can be recognised.

III. Acute miliary tuberculosis.—In this form the pulmonary condition is frequently dwarfed by the symptoms of general infection. This condition, from its resemblance to typhoid fever, is sometimes described as the typhoid form of acute tuberculosis. In other instances the disease manifests a special incidence on certain organs, and types have been distinguished varying with the parts of the body principally affected; for instance, the cerebral, the abdominal, and the pulmonary.

It has been the custom to draw a sharp distinction between acute miliary tuberculosis and phthisis on account of the marked difference in the clinical symptoms of the two affections; but an eruption of miliary granulations in the other organs is a fairly common complication of chronic pulmonary tuberculosis, and is to be attributed to the entrance of a large number of tubercle bacilli into the pulmonary circulation. Moreover, many cases, clinically indistinguishable from the typhoid or disseminated type of acute tuberculosis, are found after death to present old circumscribed tuberculous lesions of the lung, which had escaped recognition during life. In fact, the acute miliary form differs from chronic tuberculosis of the lung only in the acuteness of its course and in the more widespread infection of the body. In the pulmonary type, which alone will be considered here, the disease may advance in an acute or subacute manner without any premonitory symptoms. In a large proportion of cases a period of ill-health, of variable duration, precedes the onset of the disease. The symptoms first noticed are cough, expectoration, dyspnoea, and occasionally pleuritic pain. Dyspnoea, as a rule, soon becomes the predominant feature, and is often accompanied by marked cyanosis. Hæmoptysis is uncommon; but now and then it is the earliest symptom. The temperature is generally high, reaching 103° to 104° F., and the morning remissions are less pronounced than in the broncho-pneumonic form.

Some cases have been known to run their course without any definite elevation of temperature. The pulse, from the first, becomes rapid and weak. Examination of the chest reveals signs of general bronchitis, or
bubbling râles, and rhonchi on both sides. At first no dulness on percussi-
on can be elicited, but the anterior parts of the lungs are found to
be rather hyper-resonant, the change depending on compensatory dis-
tension of the alveoli—the so-called “acute emphysema.” As the disease
progresses, pleuritic friction sounds are often heard; and patches of
dulness pointing to secondary broncho-pneumonia may sometimes be
recognised. In these parts the breath-sounds may be tubular, but more
often become muffled. This difference does not depend on the prepon-

![Diagram](image)

**Fig. 8.**—Photograph of a section of the lung from a case of acute miliary tuberculosis, showing the
mode in which general infection occurs through branches of the pulmonary veins. (Low power.)
V, Small pulmonary vein; T, tubercular growth from intima, projecting into the lumen of the vein;
B, small bronchus showing tubercular infiltration of its walls.

...derance of consolidation or pleural effusion; for, in the absence of
pleuritic exudation, the vesicular breathing may be greatly diminished
by the concomitant bronchitis and lobular collapse. Lobar pneumonia is
an occasional complication. The patient rapidly loses flesh and strength,
dyspnoea and cyanosis increase, the cough grows more troublesome, and
the sputum—which at first was mucoid—now becomes muco-purulent.
It is rare to find tubercle bacilli in the expectoration, and when this
happens, a cavity, often a very small one, will generally be found in some
part of the lung. The spleen is more or less enlarged, and may some-
times be recognised by palpation.
The diagnosis is occasionally easy, but more often difficult. In the presence of general bronchitis, associated with marked dyspnoea, cyanosis, pyrexia, and rapid emaciation, the diagnosis presents little difficulty. But in cases where the evidence of bronchitis is slight or absent, the disproportionate amount of dyspnoea is a diagnostic point of considerable value. Tubercles may occasionally be recognised in the choroid by means of the ophthalmoscope, and tubercle bacilli have, in a few instances, been found in the blood; but unfortunately such evidence is rarely to be obtained.

B. Chronic pulmonary tuberculosis.—The following modes of invasion may be recognised in their order of frequency:

(i.) Insidious.—The commonest prodromal symptoms are loss of flesh and strength, accompanied, in some cases, by a slight evening rise of temperature. Less frequently the disease is ushered in under the guise of anaemia, or of a functional derangement of the digestive system.

(ii.) Bronchitic.—After frequent attacks of bronchial catarrh, or without any previous tendency to bronchitis, the disease begins with cough and expectoration, which are attributed at first to a common catarrh; but after a few weeks or months pyrexia and other constitutional symptoms make their appearance. In some instances careful inquiry will establish the fact that a period of ill-health existed before the appearance of the cough. Many cases with such a story have originated in influenza.

(iii.) Pleuritic.—The first definite symptom is pain of pleuritic type, increased by cough or deep inspiration. The pleurisy is generally of the dry form, but effusion may take place. Pyrexia and other symptoms of phthisis may follow hard on the pleuritic seizure, or the pleurisy may gradually disappear, and the patient make a temporary recovery, only to fall ill again later, with pronounced symptoms and signs of pulmonary tuberculosis.

(iv.) Hæmoptoe.—In this class, the "phthisis ab hæmoptoe" of the old authors, the first symptom to attract attention is hemoptysis. When the hemorrhage is profuse, it almost certainly indicates rupture of an aneurysm in a vomica, that is, old-standing disease; even when no other evidence of a pulmonary lesion is forthcoming.

(v.) Laryngeal.—Phthisis occasionally begins with laryngeal symptoms; hoarseness, loss of voice, hyperæsthesia, or paræsthesia of the throat being the most common.

Symptoms.—A constant and perhaps the most important symptom from the diagnostic point of view is cough. At first dry, short, and infrequent, it is accompanied, sooner or later, by expectoration, and may become so incessant as to prevent sleep and to set up vomiting, whereby the patient’s strength becomes reduced in the most serious manner. There is no direct relation, however, between the gravity of the disease and the severity of the cough. Some patients, with extensive pulmonary lesions, have little or no cough; whilst in others, with comparatively slight disease, cough may be the predominant symptom. Cough is generally most troublesome in cases of progressive disease, and where the larynx, trachea, and large bronchia are actively engaged; but it
depends to a considerable extent on the excitability of the nervous centres. When the larynx is extensively affected the cough is peculiarly muffled and hoarse. In some cases, especially when large cavities form in the base of the lung, it assumes a paroxysmal character. Coughing fits occur most frequently in the early morning, owing to the accumulation of secretion in the larger air-passage during the night. In some cases an irritable cough is excited by the ingestion of food, and the fit may end in vomiting. This occurrence is partly to be explained by the mechanical compression of the stomach and abdominal viscera against the diaphragm; but vomiting so often follows slight fits of coughing that it seems necessary to assume the existence of a neurosis of the vagus in these patients.

Expectoration.—In the early stages expectoration is scanty and mucoid; but it soon becomes muco-purulent, and is commonly very viscid. At times it is thin and watery, from admixture with saliva. As the disease progresses, the sputum collects into small thick lumps of a dirty white or yellowish colour; this “nummular sputum” is more common where cavities have formed in the lung, but it may be met with in cases of simple bronchitis and of bronchiectasis. It is not uncommon in the same specimen to find small yellowish spots or streaks mixed with frothy mucous secretion—the mixture representing bronchial secretion and pus from cavities in the lung. At times, especially in advanced cases, the sputum becomes uniformly opaque and thick, and may assume a greenish colour. Expectoration is sometimes markedly paroxysmal, especially where cavities exist in the lower part of the lungs. Blood is often discharged with the sputum. The blood-stained sputa may be bright red, or, when blood-clots have been retained in cavities or bronchi for some time, the colour may be dark purple or blackish. In certain instances the sputum presents a brownish or chocolate colour from decomposition of blood in the cavities or bronchi. In other instances it may be of a brick-red tint, especially when active ulceration of the lung is going on; but it seldom has the rusty, tenacious character of acute pneumonia. Fæces is practically unknown in the absence of such complications as bronchiectasis or gangrene. In the more chronic forms of phthisis small particles of calcareous matter, consisting mainly of, phosphate of calcium, are coughed up from time to time. These pulmonary calculi represent caseous material that has undergone calcification, and has become loosened in the process of excavation. Sometimes they show a tendency to branch, so that some think that they may come from the small bronchia. I have found them on many occasions in the recesses of old cavities in the lung. Calcareous bronchial glands occasionally perforate the air-passages and are expectorated. The discharge of pulmonary calculi implies ulceration of the lung or air-passages, and is a sign of chronic disease; but no further diagnostic value can be assigned to it.

As regards the importance of the sputum of phthisis, it must be allowed that the naked-eye appearances alone possess no certain and pathognomonic significance, if we except the presence of blood and cal-
careous matter. The former will be considered under the head of hæmoptysis. It is doubtful whether chalky masses are expectorated in any disease other than tuberculosis of the lungs or bronchial glands; consequently this event has a certain diagnostic significance. Microscopic examination of the sputum is a most valuable method of diagnosis. By this means we recognise various forms of cells—squamous, flattened, spheroidal, columnar and ciliated epithelium, blood corpuscles, pus cells, mucin, crystalloid products of chemical change, such as cholesterol, leucine and tyrosine, fatty acids and drops of myeline, carbon particles, elastic tissue from the lungs, and microbes of different kinds. Of all these constituents of the sputum two only are pathognomonic, elastic tissue and tubercle bacilli. The presence of the former is a positive proof of destructive disease of some portion of the respiratory apparatus, though it does not enable us to distinguish the precise nature of the disease; but as tuberculosis is by far the commonest ulcerative affection of the lung, the presumption is in favour of this being the process at work. When the elastic tissue shows an alveolar arrangement we may be certain that it is derived from the lung; but isolated fibres may possibly come from the larynx, or from the trachea and bronchi; though, unquestionably, their main source is the lung.

Elastic tissue.—If the opaque whitish particles seen in the sputum be teased out with needles and examined in a drop of water, branching elastic fibres with their curled-up ends, or portions of the more characteristic alveolar framework of the lung, may be recognised under a low power. Sometimes, in cases of chronic excavation, the fibres are encrusted with minute particles of lime salts. The persistent presence of elastic tissue with alveolar grouping is a sign of progressive destruction of the lung. A better and more certain method is that devised by Dr. Fenwick. The sputum is mixed with an equal quantity of a solution of caustic soda of the strength of 20 grains to the ounce, and boiled for a few minutes until the mixture becomes clear. The fluid is now allowed to stand in a conical glass for a few hours, when the elastic fibres fall to the bottom. A drop of the sediment is then withdrawn with a pipette and examined for elastic tissue under a low power (90 to 100). It is important not to continue the boiling too long, as the elastic fibres themselves ultimately become much altered.

Microbes—Tubercle bacillus.—The sputum voided in the early morning
should, if possible, be chosen for investigation, as it contains no particles of food, and as, being composed of the secretions accumulated during the hours of sleep, it represents a mixture of the products of the various sections of the respiratory tract. The expectoration is poured out into a flat glass dish, and examined against a dark background. The small opaque specks and streaks, or, where the sputum is uniformly opaque, the most curdy portions, are the most suitable for examination. A small portion should be removed with a scalpel, needle, or platinum wire and transferred to a perfectly clean cover-glass. A second cover-glass is pressed gently on the first, so as to distribute the sputum in as thin a layer as possible; and the two glasses are then separated by a sliding movement and allowed to dry. When quite dry, the cover-slips are seized with a forceps and passed three times quickly through the flame of a Bunsen burner or of a spirit-lamp to coagulate the albumin. Various methods are in use for staining the bacillus. Ziehl's modification of Ehrlich's method gives excellent results and will alone be described. The following reagents are required:—(a) Ziehl's solution of carbol-fuchsin, consisting of 10 c.c. of a saturated alcoholic solution of fuchsin, added to 90 c.c. of a 5° per cent watery solution of carbolic acid. (b) A 25 per cent solution of sulphuric acid. (c) A concentrated aqueous solution of methylene blue. The staining fluids should be filtered before they are used. The cover-glasses are placed in some of the fuchsin solution (a) in a watch-glass or porcelain dish, which is then heated carefully over a spirit-lamp or a sand-bath until bubbles are given off. The staining fluid is then set aside for two or three minutes to cool. Next, the cover-glasses are removed with forceps and passed through some of the acid solution (b) for a few seconds, until the red colour changes to a yellowish gray. The preparations are then washed in a gentle stream of water running from a tap for eight or ten seconds, when the sputum again turns red. Lastly, the slips are stained with a drop of the blue dye (c) for half a minute to a minute, washed again in a stream of water for a few seconds, and the excess of water allowed to drain away. The cover-glasses may now be left to dry; or rapid drying may be effected by pressing them gently between two pieces of clean blotting-paper. When they are quite dry the preparations are mounted in a drop of Canada balsam, dissolved
in xylol or benzol. If the illumination be good, the tubercle bacilli can be recognised with a magnifying power of 300; but in order to obtain satisfactory results it is well to use Abbé's substage condenser and a onetwelfth oil immersion lens.

With the above mode of staining the tubercle bacilli appear as delicate rods of a red colour, in length from a quarter to a half the diameter of a red blood corpuscle, often straight or slightly curved, and in many cases presenting a finely-headed appearance. This beading, supposed by some to depend upon the presence of spores, possesses no special clinical significance. The number of bacilli in the sputum is very variable. They may be scattered singly through the preparation, or they may be found in groups. In some instances the sputum seems to be an almost pure cultivation of the bacilli. Many other microbes, pyogenetic and putrefactive, the nuclei of pus and epithelial cells and threads of mucin are stained blue by this process, but no definite importance can at present be assigned to any microbe but the tubercle bacillus. The detection of tubercle bacilli in the sputum is a certain sign of tuberculosis of some part of the respiratory tract.

Instances of primary tuberculous ulceration of the larynx or pharynx are infinitely rare, but the number of bacilli shed from the surface of such ulcers is insignificant when compared with the enormous masses discharged from cavities in the lung. When the sputum contains a large number of bacilli we may reckon on the existence of a vomica, whether large or small.

Hæmoptysis is one of the most striking symptoms of the disease. Streaks of blood may be seen in the expectoration of many other affections, and are the result of capillary hæmorrhage from the lungs or air passages: such streaks are seldom of much importance; nevertheless, they occasionally herald the approach of a more profuse hæmorrhage. When the amount of blood brought up is considerable, the significance is far greater.

Hæmorrhage from the lungs may occur as the result of hyperæmia and rupture of capillaries, or of gross pulmonary lesions involving perforation of vessels of considerable size. Slight attacks of hæmoptysis are mainly due to capillary hæmorrhage from the lungs, less frequently from the large air-passage, and are indicative of inflammatory or congestive states. When, however, the blood expectorated can be measured by ounces, the bleeding must be attributed to rupture of an artery or vein of some size. Perforation of vessels, generally of an artery, may be effected in three ways:—(a) The walls of the small pulmonary arteries and veins may become infiltrated with a tuberculous growth. The usual consequence of this change is thrombosis of the affected vessel; but in the case of the arteries, softening of the vascular wall may lead to rupture, and some of the small hæmorrhages of phthisis are probably thus produced. (β) The ulcerative process associated with excavation of the lung may perforate an artery of considerable size, and occasion alarming hæmorrhage. It seems strange, at first sight, that this does not happen more often; but
the tendency in all but the more rapid forms of tuberculous destruction is towards thrombosis of vessels. This is a more important cause of hæmoptysis than the preceding, but it is very much less common than the next (γ), namely, rupture of an aneurysm in a cavity in the lung: this is by far the most common cause of profuse hæmorrhage.

Hæmoptysis may prove directly fatal from cerebral anæmia, though a termination by syncope is uncommon. The usual cause of death is asphyxia, which results from flooding of the bronchia with the effused blood. Ruptured aneurysms may become closed by thrombosis and the patient recover. There is every reason to believe that most cases of profuse hæmoptysis which end in recovery are due to the rupture of an aneurysm, and that ulceration of large vessels is a much less frequent cause.

The old view that the extravasation of blood can set up inflammatory and destructive changes in the lung—"phthisis ab hæmoptoe"—is no longer entertained.

One of the points adduced by Niemeyer in support of this notion, namely, that pyrexia often appears a few days after the hæmorrhage, is more easily explained by the aspiration of infective cavity secretions, mixed with blood, into other parts of the lung, leading to tuberculous broncho-pneumonia. An attack of hæmoptysis is occasionally determined by some obvious cause of vascular excitement, such as mental agitation, muscular exertion, straining at stool, or menstruation; but more often the patient suddenly begins to cough up blood without any warning, often while in bed. Hæmoptysis is generally repeated frequently, and may last for hours or days with intermissions. The blood expectorated is generally bright and frothy; but when it has gathered slowly in cavities or in the bronchial tubes it may be dark and dotted. The quantity lost varies considerably; as much as two or three pints may be brought up in a short time. When the flow is not excessive the blood is often mixed with sputum, a point of considerable diagnostic importance; and in most cases expectoration of blood-stained secretion continues for a day or two after all active hæmorrhage has ceased. The bleeding may manifest a marked tendency to recur at intervals for some time; in such cases the rent in the walls of the aneurysm has undergone only partial repair, and leaking goes on from time to time. The patient is almost always greatly alarmed by the supervision of hæmorrhage. The face is pale and bedewed with sweat, the extremities cold, and the pulse is feeble; the bodily temperature sinks below the normal. Blood is brought up with a frequent short cough, and is often swallowed. When the hæmorrhage is arrested the temperature returns to the normal range, and on the third or fourth day may rise three or four degrees. After the attack, patients are much exhausted and depressed; partly in consequence of the loss of blood, but still more as the result of nervous shock.

Some patients show no serious deterioration of health after the immediate debilitating effects of the hæmorrhage have passed away; but in not a few instances, under the influence of repeated attacks of hæmo-
ptysis, chronic disease assumes a subacute, progressive character, a result attributable to the violent inspiratory efforts provoked by the presence of blood in the bronchia and the consequent insufflation of infective secretion into healthy lung.

Some writers have described a special variety of ptysis under the name "hemorrhagic," but there does not appear to be any sufficient reason for the subdivision. Cases beginning with a sudden haemoptysis, repeated, perhaps, at intervals for a considerable time, may subsequently run the ordinary course of chronic ptysis without any further hemorrhage. Other patients presenting the usual form of disease may succumb after a succession of attacks; or their first hemorrhage may prove fatal. Hemorrhage is an accident which may complicate any case of the disease, and is not a satisfactory basis for classification.

Dyspnoea.—A subjective sense of dyspnoea is seldom complained of, save in the later stages of the disease; though most patients with progressive ptysis exhibit increased frequency of respiration, especially on slight exertion. The rate of respiration rises slightly in the evening. The absence of dyspnoea is explained by the tolerance acquired during the slow, insidious progress of the pulmonary affection, and also, as has been suggested, by the low standard of respiratory requirement due to the reduced volume of blood.

When chronic lesions are complicated by acute tuberculosis, especially in its miliary form, and when pneumothorax occurs, urgent dyspnoea may arise.

Pain in the chest.—Many patients have pain in the chest, mostly in the axillary or mammary regions, varying in degree from a slight aching sensation to the agonising stitch of pleurisy. Severe pain is nearly always referable to the implication of the pleura, in which case tenderness to percussion is often met with. Vague rheumatoid pains in the chest have been regarded as very significant; but in the absence of other symptoms more directly pointing to the lungs, little importance can be attached to them. They are, not infrequently, of muscular origin, and may be attributed to the violence of the cough. Dragging pain over chronic cavities, associated with retraction of the chest wall, is sometimes a persistent symptom depending on stretching of the adjacent pleura and intercostal nerves. The muscles of the chest wall in some cachectic patients are extremely tender to percussion, and the slightest tap may promote muscular contraction; but this increased excitability of the muscles is not peculiar to the disease.

General symptoms.—Pyrexia is a symptom hardly less significant, from the point of view of diagnosis, than cough; and of infinitely more value as a measure of the activity of the disease. The cause of the elevation of temperature must be ascribed to the presence in the blood of some soluble poison produced by the bacillus. It is generally agreed that the pyrexia of tuberculosis attains its maximum, and may often be exclusively present, in the post-meridian hours of the day. A slight evening rise of temperature may be one of the earliest symptoms. Some observers have
noted a persistently subnormal temperature as the first definite indication of the disease. Accordingly careful thermometric observations, night and morning, should be made in all cases of obscure ill-health, especially in young persons.

The onset of fever is sometimes accompanied by slight shivering, but a marked rigor is seldom observed except in acute cases. The maximum temperature is found from 2 to 10 P.M., and the minimum from 2 to 8 A.M. In exceptional cases the order is reversed, the morning temperature being higher than the evening—the "inverse type." This may be only temporary, or the same relation may be preserved throughout the whole course of the case.

Two main forms of pyrexia may be distinguished, the intermittent and the remittent. One or other form may predominate or prevail exclusively for weeks or months, but various combinations are apt to arise; in fact, one of the characteristics of tuberculous fever is its fluctuating and irregular course.

In the first or intermittent type the temperature is normal or slightly subnormal in the morning, and reaches 100° to 103° F. in the evening. In the higher grades of this form the fall is still more pronounced, and may amount to 7° or 8° F., the thermometer sometimes registering a temperature as low as 94° or 95° F. The second or remittent type shows a maximum temperature of 103° to 104° F., the minimum temperature being 2° to 3° lower, but not reaching the normal level. A slight degree of intermittent fever, where the maximum, for the most part, does not exceed 101° to 102° F., is often found in the early phases of the complaint; but a similar temperature curve may be recorded at any stage. When the range of temperature is greater, and more particularly when the morning reading is below normal, profuse sweating is very common, and the resemblance to the hectic fever of pyæmia is very close. The remittent form of fever commonly betokens active tuberculous infiltration, and is more often met with in the acute varieties of tuberculosis, but may also appear temporarily in chronic phthisis as the result of acute exacerbations or of intercurrent disease. In acute miliary tuberculosis, uncomplicated by suppuration in the lung or elsewhere, the type of fever is generally remittent, a fact which would point to this being the form of pyrexia peculiar to tuberculosis.

Pulmonary phthisis never runs its whole course without fever, but in many chronic cases there may be no appreciable rise of temperature for long periods of time. Observations by Dr. C. T. Williams have shown that pyrexia may be absent even when the disease is making rapid progress. But this is a very unusual course, and it may be stated as a general principle that activity of the disease is indicated more surely by pyrexia than by any other symptom or sign.

A high evening temperature with a markedly subnormal morning temperature (95° to 97°) is a common feature of advanced and progressive disease, though in such cases the fever may assume the remittent type at any time. Towards the close of life the temperature generally
tends to fall. The very low temperature registered in pneumothorax, in
some cases of excessive pulmonary hemorrhage, and in the comparatively
rare instances where perforation of the intestine occurs, must be attri-
buted to the effects of shock. When we consider the various processes
of infiltration, necrosis and suppuration occurring in the lungs, as well
as the numerous complications that may arise, we cannot be surprised at
the great variations exhibited by the temperature chart.

It is necessary to mention the assertion of Peter, that the temperature
of the skin differs on the two sides of the thorax, the higher reading
being found on the side corresponding to the lung more affected. Most
observers have failed to verify this statement, and a similar want of
symmetry in the temperature of the two armpits has occasionally been
observed in other conditions.

Sweats.—Profuse perspiration is a common symptom in pyrexial
cases, though it has no constant relation to the fever. Sweating is most
pronounced in the early hours of the morning, when the temperature of
the body is at its lowest; but it also occurs sometimes while the fever is
continuously high. Night sweats may occasionally occur in apyrexial
periods, in which case they seem to be due to fits of coughing.

Dr. Lauder Brunton has suggested that sweating is the result of
exhaustion of the respiratory centre in the medulla, and consequent
accumulation of carbonic acid in the blood; the effect of this being to
stimulate the sweat centres. This symptom is certainly more prevalent
in advanced cases, associated with excavation and suppuration of the
lung; but it is not uncommon in early and circumscribed disease, in
which case Brunton's hypothesis seems less applicable.

Emaciation is one of the most frequent and important symptoms, and
may proceed to an extreme scarcely reached in any other disease; hence
the names phthisis and consumption.

The greatest loss of weight is witnessed in chronic cases, but although
sometimes at first comparatively slight in the acute type, which termin-
ates in a few months, it is never absent altogether. Wasting affects all
the soft parts, but especially the fatty and muscular tissues. It has
been said that the liver does not share in the general wasting, but this
statement is probably to be explained by the great liability of the liver
to congestion and to fatty and amyloid degenerations, conditions which
involve enlargement of the organ. The loss of flesh is mainly, though
not exclusively, due to the increased metabolism inseparable from the
febrile process. Patients with a high temperature lose weight as long
as the fever continues; and, as a rule, when the heat of the body becomes
normal, wasting ceases. Moreover, a certain correspondence between the
degree of the fever and the loss of weight can often be recognised. At
the same time a considerable degree of fever is not incompatible with an
actual increase in weight, if an adequate supply of food can be taken,
and if digestion and absorption be unimpaired. In apyrexial cases the
weight of the body may remain stationary for months, or even years;
but when pyrexia appears, loss of flesh soon follows.
The early emaciation, which not uncommonly precedes any appreciable rise of temperature or other signs of disease, cannot be thus explained. In the absence of any definite knowledge on this point we may adopt the provisional hypothesis that the toxins of tuberculosis may cause a general failure of nutrition apart from any febrile movement. Functional derangements of the stomach and diarrhoea, by their interference with digestion and assimilation, are potent causes of wasting.

Debility.—A sense of weakness and loss of energy, both of mind and body, are commonly felt at a very early date, and not infrequently appear to be out of all proportion to the extent of the disease.

Anemia.—In certain patients the complexion acquires a peculiar faded yellowish tint, which has been well likened to a dead leaf. On examination the blood shows the changes of chlorosis—a considerable reduction of the haemoglobin with a relatively slight diminution in the number of the red corpuscles, and also a diminution in mass. In active pyrexial disease a moderate degree of leucocytosis is common.

The pulse in all progressive cases is rapid and of low pressure; sometimes it is full, but more often small. The frequency of the heart’s action is not invariably determined by the degree of fever, but seems rather to stand in direct relation to the extent and activity of the disease, and to the strength of the patient; consequently the pulse is a most valuable index of the gravity of the case. The pulse is generally somewhat more frequent in the evening, but exceptions to this rule are met with. Some authors have regarded a persistent rapidity or ready excitement of the heart as important premonitory symptoms; and there is no doubt that cardiac crepitation is often present at a very early stage of the disease.

Cyanosis is seldom a marked symptom, except as the result of serious pulmonary or cardiac complications, though the fingers, toes, lips, ears and nose often present a slightly dusky or livid hue, in marked contrast to the general pallor. Coldness of the extremities and extreme sensitiveness to trifling depressions of temperature are a common complaint, and further testify to the feebleness of the circulation.

Skin and hair.—In connection with the subject of nutrition reference must be made to the state of the skin and hair. The skin of tuberculous patients is generally very oily, and the sweat has a peculiarly pungent garlicky odour. In some emaciated subjects, on the contrary, a dry branny condition, “pityriasis tabescentium,” may be observed. The texture of the skin in one type of patients is delicate and thin, and the complexion transparent; while in another class the skin is coarse and the complexion dull and muddy,—distinctions which are included in Sir William Jenner’s classical description of the tuberculous and scrofulous diatheses respectively. But in the majority of phthisical persons no such peculiarity can be recognised, though in all cases of long standing some degree of pallor is wont to appear. Pigmentation of the skin may become so marked in certain chronic cases that Addison’s disease may be simulated; but the patchy pigmentation of the tongue and buccal mucous membrane,
so characteristic of the latter affection, does not occur. The cause of this pigmentation is unknown. "Pityriasis versicolor" is observed rather frequently on the chest and back, but no special significance can be assigned to this parasitic complaint. Lupus is only occasionally found in phthisical patients. The terminal phalanges of the fingers and toes frequently show a curious bulbous enlargement associated with incurvation of the nails; the swelling is believed to be due to thickening of the subcutaneous tissue, but it is possible that the bony structures may also be involved. This clubbing of the fingers and toes is not peculiar to tuberculosis, and it is found in empyema, in chronic pneumonia, in certain forms of heart disease, and in emphysema—conditions in which impediment of the pulmonary circulation and consequent engorgement of the systemic veins are a common factor.

The hair, participating in the general malnutrition, may become thin and straight; but this change is by no means constant, as in some persons the hair of the head and beard remains very thick, and the trunk may be unusually hirsute. In children and young persons the body is sometimes covered with a growth of fine downy hair.

**Physical diagnosis.**—Certain abnormal forms of chest are met with in many phthisical subjects. Two special varieties may be mentioned on account of the frequency with which they occur. In the first, named alar or pterygoid by Galen and Aretaeus and in our own day by Dr. Gee, the angles of the scapulae project like wings, the ribs are unduly oblique, the shoulders fall, and the length of the thorax from above downwards is increased, but the antero-posterior diameter is small. In the second or flat type the chest in front is flat instead of being rounded, and the sternum may even be depressed below the level of the costal cartilages, which lose their curve and become straight. These peculiarities are certainly common in tuberculous persons, but they are frequently met with also in persons who remain free from the disease. Moreover many phthisical persons have large and well-formed chests. It cannot be said, in other words, that there is any type of thorax peculiar to phthisis, although the chest, in common with the muscles and bones, is often ill developed. Much more importance is to be attributed to partial deformities of the chest walls, the result of pulmonary disease.

Before discussing the physical diagnosis of the disease in its early stages, it may be well to recall briefly a few anatomical facts. The initial lesion consists of a small nodule or group of nodules situated somewhat below the extreme apex of the lung. The nodule is broncho-pneumonic; that is, it consists of a localised bronchitis with surrounding lobular consolidation. The neighbouring parts of the lung at first remain spongy and practically unaltered, so that the nodule is enclosed in a shell of healthy pulmonary tissue.

Physical examination of the lungs at this period may yield a completely negative result, especially when the focus of disease is small, and the layer of spongy lung around is fairly thick. As long as the surrounding lung is crepitant, percussion gives no dulness. The earliest signs are almost exclusively discovered by auscultation, though at times, on
inspection and palpation, a slight diminution of respiratory movement may be recognised in the subclavian region. Owing to the persistency of the apical catarrh, and to the consequent lobular collapse, the entry of air into the corresponding section of lung is diminished, and the breath-sounds become weakened. Jerky, interrupted, or wavy breathing—the "respiration saccadée" of the French—is not very uncommon, but is not pathognomonic, and may often be heard in neurotic or hysterical, or even in healthy persons. Weakness of breathing at the affected apex is often associated with increased loudness of the vesicular murmur on the opposite side—a condition known as compensatory or puerile breathing, which is sometimes erroneously regarded as an indication of disease.

Another important and early sign is furnished by harshness of the breath-sounds affecting the expiratory sound to a greater degree, and at an earlier date than the inspiratory. The expiratory murmur at the same time acquires a higher pitch, and becomes so prolonged as to equal or exceed the length of the inspiratory sound. This change is an early indication of consolidation, the character of the breath-sounds being modified, without having actually attained to the bronchial or tubular type. It is necessary to distinguish this condition from mere prolongation of the normal expiratory murmur, which may be the result of bronchial obstruction, as in bronchitis, emphysema and asthma, and is then generally associated with a weak vesicular inspiration. At this period the vocal resonance and tactile fremitus may be slightly increased, or there may be no recognisable alteration.

It will be convenient at this point to make a passing reference to what may be called the physiological dissimilarity of the right and left apices. In the large majority of healthy persons, especially in thin subjects, the breath-sounds are louder, the expiratory murmur more audible and prolonged, and the vocal resonance and fremitus more pronounced at the right than at the left apex. Occasionally the breathing may even be tubular at the extreme right apex. This difference probably depends upon the following facts: the right main bronchus is slightly wider and more vertical in direction than the left; the bronchus to the upper lobe is given off higher up, that is, nearer to the trachea; and the apex of the lung lies slightly closer to the trachea on the right side. The general effect of these conditions is to favour the conduction of the glottic sounds to the right apex. Accordingly we must bear this in mind in estimating the importance of any slight want of symmetry of the auscultatory signs at the apices.

In some instances a slight impairment of the inspiratory expansion of the affected apex and some flattening below the clavicle may be the only physical indications of disease. The want of mobility may be recognised by inspection, but is more easily detected by palpation; the hands of the observer being placed on the subclavian region on each side, and the patient directed to breathe deeply meanwhile. At this time also fine crackling or subcrepitant râles may be heard over the affected area. These râles, which are less fine than the true crepitant râle of Laennec,
are mostly heard during inspiration, and are probably caused by the separation of the moist surfaces of the small bronchi. In some cases where the bronchial obstruction is more pronounced no adventitious sounds are audible during ordinary respiration, but when the patient coughs, a shower of crackling râles is produced by the explosive separation of the swollen bronchial walls. At times subcrepitant râles can only be elicited during the deep inspiration that follows cough.

Persistent rhonchi at one apex may sometimes be the only adventitious sounds. A systolic murmur, heard beneath the clavicle, was thought to indicate consolidation of the apex of the lung, and was attributed to the effects of pressure of the infiltrated lung on the subclavian artery; or, with greater probability, to contraction of the thickened pleura at the apex. Similar murmurs may be heard in anaemic and other persons, and are not any certain guide to disease of the lungs. Thus far physical signs give evidence of bronchitis confined to the apex of the lung, the character of the breath-sounds possibly suggesting the existence of a small patch of consolidation surrounded by spongy lung. As the infiltration is often massed at several centres, islets of spongy tissue separate the individual nodules, and for a time mask to a great extent the signs of consolidation. Thus when the lobules around the tuberculous patches are hyper-inflated the percussion may be slightly higher pitched than normal, tympanitic, or even hyper-resonant.

As the disease extends, the lung becomes more airless, and adhesive pleurisy is set up. The inspiratory expansion becomes decidedly restricted, vocal fremitus is increased, and the percussion resonance undergoes progressive impairment. The breath-sounds assume a more definitely tubular or cavernous quality; bronchophony or pectoriloquy appears, and the râles become larger and more ringing or metallic. This complete assemblage of signs is by no means generally, or indeed often presented, except in fairly advanced cases. Tubular breathing may appear at a comparatively early period, but this is unusual; and with marked dulness, bronchophony, and coarse crackling râles, the respiratory murmur may remain simply weakened with slight prolongation of expiration.

The comparatively late appearance of tubular breathing is mainly due to the obstruction of the bronchi, which is so generally present, and to the irregular composite nature of tuberculous consolidation.

Dulness appears first at the supraclavicular and supraspinous fossae, and thence extends downwards over the front of the chest. For the recognition of slight degrees of dulness light percussion and careful attention to the sense of resistance are required. Increased conduction of the heart’s sounds to the corresponding apex generally accompanies and sometimes precedes loss of resonance to percussion. Increasing size of the râles, with a sharply conducted or ringing character, is generally described as marking the presence of softening; but the same signs may be furnished by profusely secreting bronchi of considerable size situated in solid lung.
Phthisis Pulmonalis

Rhonchi of a croaking or metallic quality are not uncommon at this period, this peculiarity being imparted to them by the adjacent solid lung or cavities. Dulness gradually extends over a considerable portion of the upper lobe, and rales become audible at the apex of the opposite lung, and at the infraspinous fossa on the same side; that is, at the apex of the lower lobe.

The date at which signs of excavation can be recognised varies greatly. In some cases a cavity may be detected almost as soon as consolidation can be diagnosed, whereas in others it may be months or even years before this is possible.

Signs of excavation.—Over a cavity of considerable size the percussion is generally more or less impaired, and it may be markedly dull in consequence of the surrounding infiltration and of the pleuritic thickening which so often coexist. As excavation proceeds, the dulness may diminish. The resonance is often of tympanitic, tubular, or amphoric quality, as well as slightly dull, resembling the note produced by percussion of the trachea in the neck. Percussion may elicit the cracked pot sound where a cavity communicates freely with the bronchus, and its walls are sufficiently elastic. This sign is not pathognomonic of a cavity, as it may often be obtained by percussing the chest of a healthy infant while crying, and is sometimes found in cases of pneumonia and pleural effusion. The bell sound—"bruit d'airain"—is occasionally heard over large cavities.

According to Wintrich, the pitch of the tympanitic percussion sound over a cavity becomes raised when the patient opens his mouth. An alteration of pitch may sometimes be recognised when the patient changes from the sitting to the lying position, or conversely (Gerhardt); but these changes are seldom pronounced, and give little practical assistance. It is, however, to auscultation that we must mainly trust for the diagnosis of pulmonary excavation. In well-marked cases the breath-sounds are tubular or cavernous—the term "tubular" is used here as synonymous with "bronchial."

Some writers maintain that there is no difference between tubular and cavernous breathing, unless it be in the greater intensity and hollowness of the latter. Flint makes the relative pitch of the inspiratory and expiratory sounds the basis of distinction. According to this author, cavernous breathing is generally of lower pitch than tubular, and the expiratory sound is of lower pitch than the inspiratory; whereas the pitch of tubular breathing is generally higher than that of cavernous respiration, and expiration is higher pitched than inspiration.

The breath-sounds over a cavity may be very weak, or even absent when the bronchial opening is small or obstructed in any way, as by profuse secretion or by cicatricial contraction. If the vomica be separated from the chest wall by a zone of spongy lung the respiration may be simply blowing, with prolonged expiration.

The "metamorphosing" breathing of Seitz consists of an inspiratory sound, harsh or rough at its commencement, becoming hollow or tubular
towards the end of the act of inspiration. This sound is supposed to be due to the removal of a partial obstruction of a bronchus as inspiration proceeds. It is not a common sign, and it is not certain that it is exclusively a cavernous sign. Amphoric breathing is pathognomonic of a large air-containing cavity with smooth walls. Large gurgling râles are often heard where cavities contain abundant secretion, and this may be the only auscultatory evidence available at times. When such sounds are audible in regions like the apex, which contains no bronchi of large size, they are very significant of cavities. Auscultation of the cough gives valuable, perhaps the most valuable, evidence of excavation. In a cavity containing fluid and air the agitation produced by cough often gives rise to râles of a splashing character, resembling on a small scale the succussion sound of pneumothorax. Râles of this description are very suggestive of a cavernous origin. A metallic or amphoric echo of the cough is less common but is quite characteristic.

Post-tussic suction is another highly significant sign; it consists of a high-pitched, sucking, inspiratory sound, immediately following the forced expiration of cough, and is due to the elastic recoil of the cavity walls. This has been well named the "india-rubber ball sound" by Dr. Mitchell Bruce.

Metallic tinkling is occasionally heard over large smooth-walled cavities. The vocal resonance is generally increased, bronchophony or pectoriloquy being very common; but the latter is not so decisive a test of excavation as is generally believed. In rare cases an amphoric quality is imparted to the voice when other metallic phenomena are present. In some cavities, where the breath-sounds are feeble, the resonance of the voice may be diminished, especially if the bronchus be obstructed. Cardio-pulmonary systolic murmurs are sometimes heard over large thin-walled superficial cavities lying close to the heart, mostly in the left upper lobe. These murmurs are caused by expulsion of air from the cavity through a bronchus by the impact of the heart on the lung.

Similar cardio-pulmonary murmurs may be occasioned in the absence of any cavity in the lung, if the heart’s action be much excited. In cases of contractile disease of the left upper lobe, a systolic murmur is not uncommonly audible in the second left interspace close to the sternum, the bruit being due to traction of the lung on the pulmonary artery. In one case of this sort there was also a marked systolic thrill in this region, which suggested the possibility of stenosis of the pulmonary artery; but an autopsy showed that it was due simply to contracting lung.

A few instances have been recorded in which a systolic murmur was produced by an unsupported and dilated branch of the pulmonary artery crossing a cavity. It should be mentioned that the chest wall may be markedly retracted over the site of a chronic contracting cavity.

Some writers, following Sir Andrew Clark, recognise "fibroid phthisis" as a peculiar variety. Most of these cases are but pulmonary tuberculosis in a very chronic form. There is little in the physical
signs to distinguish them from non-tuberculous chronic pneumonia, except that in the former the disease is nearly always most pronounced in the upper lobe, and the apex of the other lung is often involved. In the fibroid or contractile form of pulmonary tuberculosis, signs of excavation are generally to be recognised at one apex, associated with much dulness over the upper lobe or over the whole lung, together with displacement of neighbouring organs. When the left lung is affected the heart is drawn outwards and upwards, and pulsation may be felt as high as the second rib or clavicle; or the apex beat may be discovered in the axilla. In such cases the shock of the second sound may often be recognised in the upper intercostal spaces by palpation. In two of the most extreme instances of displacement of the heart in this disease I found the heart beating under the angle of the left scapula.

When the right lung is contracted the heart is drawn over and may lie wholly to the right of the middle line, the pulsation sometimes reaching as far out as the right axilla. The diaphragm and abdominal viscera are raised by the contracting lung, especially when the upper lobe is principally involved. On the left side the tympanitic resonance of the stomach may extend as high as the fourth or fifth rib. In extreme contractile cases the opposite lung is always considerably enlarged, and may pass beyond the middle line of the sternum into the opposite half of the thoracic cavity. It is often extremely difficult to detect any signs of disease in a lung thus distended, though a post-mortem examination in these circumstances will nearly always reveal the existence of deeply-seated tuberculous lesions. The fact cannot be too strongly insisted upon, that in the presence of distension or emphysema of the lung extensive foci of disease may escape recognition altogether.

Irregular forms.—It seems advisable, at this point, to make a few remarks concerning the physical diagnosis of certain irregular forms of the disease.

Emphysematous form.—In this variety the history as well as the physical signs are those of bronchitis and emphysema. In addition to hyper-resonance on percussion, together with weak inspiratory and prolonged expiratory murmurs, careful percussion will sometimes elicit slight comparative dulness at one supraspinous fossa, and perhaps above the clavicle. There may be no further deviation from the normal type of emphysema. In other cases, on coughing, a few muffled râles may be audible at one apex. If, as often happens, diffused rhonchi are also present, the difficulties of diagnosis are much increased. The shape of the chest is often flat instead of being rounded, a matter of some importance. In emphysematous people with such a formation of thorax, especially if there be much wasting or if hæmoptysis have occurred at any time, the possibility of tuberculosis should be carefully considered, and the sputum should be repeatedly examined for tubercle bacilli.

Pleuritic form.—Reference has already been made to the onset of pulmonary tuberculosis with symptoms of pleurisy. Signs of fluid effusion, thickened pleura, or dry pleurisy in one axilla or at the base,
may be the only recognisable signs. It is of the utmost importance in all cases of pleurisy to keep in mind the close relation of this affection to tuberculosis. Double pleurisy, whether there be effusion of fluid or not, is nearly always tuberculous—the principal affections that have to be excluded being renal disease, acute rheumatism, and intrathoracic growths. Where a large effusion occupies the whole of one pleural cavity, no evidence of tuberculosis can be obtained from physical examination of the affected side.

At times rales or other morbid signs may be detected at the apex of the other lung, but too much importance must not be attributed to such a discovery, as in cases of this description the unaffected lung is often the seat of compensatory hyperaemia and oedema. Similar evidence of apical disease in cases of basic dry pleurisy, on the contrary, has a very definite and positive value. But the sputum may be the only trustworthy evidence of the tuberculous nature of the complaint.

It is commonly said that an insidious onset characterises tuberculous pleurisy, whereas an acute invasion is more suggestive of the simple idiopathic variety. No reliance can be placed on such statements. Tuberculous pleurisy may commence in the most acute manner; and a chronic insidious onset is not rarely witnessed in cases of a comparatively harmless nature. In any case of pleurisy, marked wasting, or a history of haemoptysis, should arouse suspicion.

Anomalous distribution of physical signs.—When signs of infiltration or excavation are confined to one base, or predominate there, an accurate diagnosis may be very difficult, in view of the extreme rarity of primary tuberculosis of this part. The fact that physical signs of disease are confined to or predominate at the base, by no means proves that there is not, at the same time, older disease of the apex of the upper lobe, a point which I have several times established on post-mortem examination. This depends on the fact that when the lesions are covered by a shell of healthy lung considerable masses of tuberculous disease, or even cavities, may exist towards the central part of the upper lobe without giving any evidence of their presence.

Disease confined to the base of one lung in most cases is not tuberculous, and we have, in such instances, to exclude various affections, the most important of which I may here enumerate:—chronic pneumonia with or without bronchial dilatation, localised pleurisy, abscesses of the liver, new growths, hydatid cysts of the lung or liver, and hypophrenic abscesses. Examination of the sputum is of the utmost value under such circumstances.

It is well, at the same time, to remember that these diseases may be complicated by a secondary tuberculosis, and the discovery of tubercle bacilli may divert attention from the primary affection. Chronic contracting lesions of the apex of the upper lobe, particularly on the right side, may so uncover the great vessels at the base of the heart as to cause pulsation to be felt in the upper intercostal spaces, and thus aneurysm may be simulated. This is more likely to occur on the right side,
where, on more than one occasion, I have known the association of dulness, pulsation, systolic murmur, and accentuated second sound to give rise to considerable suspicion of aortic aneurysm in middle-aged men.

Laryngeal form.—Where laryngeal obstruction exists the entry of air into the lung may be so greatly diminished that auscultation may give no trustworthy indications of the actual condition of the lungs. The amount of pulmonary disease, without any corresponding auscultatory signs, which may exist under such circumstances is surprising, and can only be appreciated by those who have been able to compare the post-mortem appearances with the results of physical examination during the patient’s life. Percussion sometimes gives more valuable assistance than auscultation; but the most certain information is often afforded by the spumtum test.

Diagnosis.—The diagnosis rests, in the first place, on the presence of chronic disease of the lung, affecting mainly or exclusively the apex of the upper lobe. Signs of persistent catarrh, consolidation, or excavation of this part are, for practical purposes, conclusive evidence of tuberculous disease.

The existence of tubercle bacilli in the spumtum is an absolute proof of tuberculosis of some part of the respiratory tract. In the absence of tuberculous ulceration of the larynx, pharynx, or oral cavity, the lung may be regarded as the source of the bacilli, even if auscultation and percussion give no indication of any pulmonary lesion, or if physical signs of disease be found in aberrant situations.

Most writers consider the subject of physical diagnosis under three stages—the first, second and third stages of phthisis. Such a division of the subject implies that physical examination may be trusted to decide at which of these stages the disease has arrived—an assumption by no means warranted by the facts. As a description of the history of individual tuberculous foci, there is not much fault to be found with the time-honoured division into three stages of consolidation, softening, and excavation; but these distinctions are, to some extent, misleading. In the first place, as soon as the stage of softening is reached excavation has begun; in other words, the two processes are, more or less, concomitant; moreover, the rule is to find in the same lung—often in close proximity—solid nodules, softening caseous masses and fully formed cavities; in other words, all three stages are run simultaneously.

On the clinical side of the question auscultation and percussion enable us to recognise consolidation with no little accuracy, and in many cases the existence of a cavity is revealed by certain physical signs; but there is no distinctive sign of softening whatever. In the majority of cases, where, as the result of physical examination, the patient is said to be suffering from phthisis in the first stage, cavities already exist. This is frequently proved by demonstration by the detection of elastic tissue and numerous bacilli in the spumtum of cases in which auscultation and percussion point only to catarrh, or to slight consolidation of one apex. It is a
matter of everyday experience that cavities in the lung may escape detection during life; and I have known the most experienced physicians diagnose excavation where post-mortem examination showed that none existed. The effects of this artificial classification in the patient's mind have, in many instances, been most pernicious. For, knowing that there are three stages, and hearing that he has a cavity in his lung, he concludes that, as he is in the last stage, his days are numbered. As a matter of fact, many persons in whom a cavity can be diagnosed are in a better condition, and have far more favourable prospects, than others in whom there are only signs of the first stage. It is time that the three stages were consigned to a well-merited oblivion. I make these remarks in no wish to detract from the importance of physical examination; my object is rather to recognize the limitations of this valuable method, and to give a caution against the overweening confidence still reposed by some physicians in auscultation and percussion, to the exclusion of other means of diagnosis.

The complications of phthisis are mostly referable to the transmission of the tubercle bacilli to other parts of the body. In the case of the pharynx, larynx, and trachea, tuberculous changes are mainly produced by the direct inoculation of these parts with the sputum which is constantly passing over them. But in secondary tuberculosis of the genito-urinary, nervous and osseous systems, infection is conveyed by the blood—the microbes, for the most part, effecting an entrance into the circulation through branches of the pulmonary veins.

Laryngeal tuberculosis is almost always secondary to the same disease of the lungs, though in a few well-authenticated cases the lungs have been found on post-mortem examination to be unaffected. The larynx is very frequently implicated; according to my post-mortem statistics this happened in 50 per cent of all cases of pulmonary tuberculosis. In many cases the lesions were recent, and were evidently due to late infection of the larynx. If we exclude all patients in the last stages, it may be said that laryngeal tuberculosis is clinically recognisable in from 20 to 25 per cent. The lesions consist of infiltration or swelling and ulceration. The localisation is a matter of great diagnostic importance.

Tuberculous affections show a marked preference for the posterior part of the larynx, the hinder extremities of the vocal cords, the interarytenoid fold, and the laryngeal surface of the arytenoid cartilages. The epiglottis is less frequently implicated, and the ventricular bands are seldom involved, except in widespread disease of the larynx. The progress of tuberculosis is slow, contrasting strongly with the relatively rapid course of tertiary syphilitic ulceration. The early symptoms are those of chronic laryngitis; hoarseness, tickling, a sense of fatigue on using the voice, and various other paresthesia referred to the throat. Pain on swallowing is a far more important symptom, and is generally associated with swelling or ulceration of the epiglottis or arytenoid regions. Inspiratory stridor and dyspnoea depend for the most part on massive swelling of the epiglottis and aryepiglottic folds; but in certain cases extreme stenosis occurs from mechanical fixation of the cords in the
median position, in consequence of infiltration around the crico-arytenoid joints.

In the obstructive form of laryngeal tuberculosis difficulties in physical examination of the chest frequently arise; for when the entry of air into the lungs is much curtailed auscultation may discover nothing more than weakness of the breath-sounds. Hence the importance of an accurate laryngoscopic diagnosis, and repeated examination of the sputum cannot be too strongly insisted upon. For an account of the laryngoscopic appearances, and for further details of this important affection, the reader is referred to the article "Larynx." It should not be forgotten that aphony in phthisical persons is not uncommonly the result of functional paresis of the adductor muscles of the vocal cords. The trachea is rarely affected except in advanced cases of pulmonary tuberculosis, and the larynx nearly always shows similar and more extensive disease.

Bronchial glands.—The bronchial, mediastinal, and tracheal glands are very prone to tuberculous disease. In adult cases this adenopathy, as the French style it, scarcely ever gives rise to definite symptoms or physical signs. The glands most affected are the anterior or pretracheal, and the subtracheal which lie beneath the fork of the trachea. In children the enlargement of the glands may be so pronounced as to cause obstruction of the large bronchial tubes, or even of the trachea. Bronchial obstruction, if pronounced, leads to pulmonary collapse; in which case dulness on percussion and weakness of the breath-sounds, or tubular breathing, will be found over the affected area. When the upper lobe is concerned the similarity to phthisis may be very close. In some cases the continued absence of adventitious sounds may suggest the glandular origin of the lesion, as in some cases under my care which ended in recovery. Dulness and tracheal breathing over the manubrium may occasionally be found when the pretracheal glands are greatly enlarged. It is said that dulness may be recognised in the upper interscapular region; but I have never met with this myself; and it seems unlikely that enlarged glands in the fork of the trachea and, therefore, lying in front of the spine, should occasion dulness in the situation indicated. The subjects of this complaint sometimes suffer from a spasmodic cough like whooping-cough, and from attacks of dyspnoea, attributable to pressure on the vagus trunks.

Compression of the recurrent laryngeal nerve, more particularly on the left side, may cause paralysis of the corresponding vocal cord. Perforation of the esophagus by a suppurating caseous gland, when the abscess opens into a bronchus, is apt to give rise to septic bronchopneumonia, and gangrene of the lung may follow. Rupture of a glandular abscess into the trachea may cause fatal asphyxia. In many instances caseous glands undergo calcification, and the disease is thus arrested.

Pneumothorax is one of the most serious and fatal complications, statistics proving that the patient rarely survives this accident by more than one month at most; though exceptions to this rule are to be met with. It is at first sight remarkable that pneumothorax does not occur
more frequently, considering the tendency of pulmonary cavities to extend outwards towards the pleura. Dr. Samuel West's experiments enable us to understand why perforation of the visceral pleura is not necessarily followed by pneumothorax, even when there are no adhesions. For, as he shows, before the elastic recoil of the lung can assert itself, the normal cohesion of the two layers of the pleura must be overcome, and this requires considerable force; in other words, the force of cohesion considerably exceeds the elasticity of the lungs. He concludes that "pneumothorax, in its initial stage, must be an active process. Some force will be required to overcome the normal cohesion between the two layers of the pleura, and to separate them. This must be obtained by expiration, and pneumothorax, therefore, in its initial stage, is an expiratory process, and not essentially different in its production from surgical emphysema. As soon, however, as separation has been effected, the elasticity of the lungs will come into play, and air will enter the pleura until its retractility is completely satisfied" (vide p. 335).

Inasmuch as perforation of the pleura is always succeeded by inflammation the force of cohesion may soon be supplemented by adhesive pleurisy, and the entry of air into the pleural sac may be thus prevented. In cases where the opposite lung is extensively diseased the dyspnœa at first is very great, and death may occur in a few minutes; but the immediate consequences of the perforation are almost invariably recovered from. Physical examination on the affected side shows absence of movement, increased fulness of the intercostal spaces, diminished tactile fremitus, and hyper-resonance or tympanitic, percussion note. On auscultation the breath-sounds are absent or feeble—at times amorphic, and the vocal resonance is diminished; occasionally amorphic echo of the voice may be obtained. Percussion by means of coins, or with apeximeter and percussion hammer, while the stethoscope or naked ear is applied to the chest, yields a clear metallic sound, the bell sound, or bruit d'airain. Metallic tinkling and amorphic echo of the cough may also be heard. The hippocratic succussion splash can often be detected when fluid effusion has occurred, if the ear be placed on the chest and the patient be shaken sharply. The heart is displaced to the opposite side, except in the rare instances where it is fixed to the sternum by adhesions, or where the opposite lung is solidified or completely adherent.

This displacement is not due to the pressure of the pneumothorax as is commonly assumed; for in such cases, as shown by Sir R. Douglas Powell, manometric measurements may indicate no positive pressure in the pleural cavity; and his experiments have demonstrated that the dislocation of the heart is due to the unopposed elastic traction of the sound lung. The diaphragm and the abdominal visceræ on the corresponding side, being no longer held up by the elasticity of the lung, sink downwards. In some cases depression of the liver or spleen may be detected by palpation. Although effusion nearly always ensues, it may be difficult to obtain clear evidence of its presence. Sometimes there is a small area of dulness at the base, shifting, to an unusual degree, with
the position of the patient. In other cases there may be no signs of fluid except the succession splash, which, however, is quite decisive. The absence of dulness is to be explained by collection of the fluid in the cup-shaped space formed by the depressed diaphragm. In more chronic pneumothorax a copious exudation may occur, and the air gradually become absorbed. Under these circumstances there will be marked dulness and other signs of simple pleural effusion, from which the case can only be distinguished by the history. The effused fluid is generally purulent, but may be sero-fibrinous.

Instances of complete recovery after pneumothorax have been recorded by many observers. In most of these the perforation of the pleura occurred without any previous evidence of pulmonary disease; and, although it is probable that many of them were tuberculous, this cannot be stated with certainty. In a much smaller number of cases, where pneumothorax appeared in the course of manifest pulmonary disease, life has been prolonged for months or years. The occurrence of pneumothorax seems, in some instances, to exercise an inhibitory effect on the disease in the affected lung—a result probably to be attributed to the diminished blood-supply consequent on the pulmonary collapse.

Pleurisy.—A certain degree of pleurisy occurs in every case, although it may be unaccompanied by any symptoms. Signs of dry pleurisy, without any evidence of effusion, are often met with. When a dry rub is heard over a considerable area—usually the lower part of the chest—it not uncommonly indicates progressive disease; but there are many exceptions to this rule. Pleural effusion occurring in the course of pronounced phthisis is seldom very profuse, perhaps because the pleural cavity has been already partly obliterated by adhesions. The fluid is generally sero-fibrinous, sometimes purulent, and occasionally sanguineous. Cases have been recorded where rapid absorption of an effusion was followed by acute generalised tuberculosis. This, however, is a very rare sequence of events, and the relation may be accidental. Some cases of tuberculous empyema have originated in pneumothorax, where the opening has been closed by inflammation, and the air has been gradually absorbed. Empyema is much more unfavourable than sero-fibrinous effusion, as absorption cannot be expected, and treatment by incision is rarely successful. Small empyemas very occasionally undergo inspissation and arrest. Sanguineous effusion is less common than the statements of writers would lead one to suppose. Pleural effusion, like pneumothorax, exercises a retarding influence on the pulmonary disease in virtue of the collapse of the lung which ensues.

Pneumonia.—As already stated in the section on Pathology, croupous pneumonia occasionally attacks phthisical patients; but this is very uncommon. Most of the authors who mention this subject consider that the course of phthisis is not materially influenced by intercurrent pneumonia. In the only instance of this accident that I have met with, the pneumonia ended favourably with a well-defined crisis, and the old apex lesion was left in the same condition as before the acute attack.
Tuberculous persons are apt to acquire more or less acute broncho-pneumonia from time to time; but most of these attacks represent acute exacerbations of the tuberculous process. Influenza, attacking the subjects of phthisis, may set up pneumonia of the broncho-pneumonic kind, less frequently the lobar.

Circulatory system. — The heart of phthisical persons is small, and shows atrophic changes, occasionally slight fatty degeneration, and very rarely solitary tuberculous masses in its muscular walls. It is rarely that such lesions give rise to any functional disturbance. In some of the most chronic cases dilatation of the right ventricle may occur.

Endocarditis is not very uncommon, and is sometimes attributable to previous attacks of acute rheumatism, but by no means always. Some French observers state that they have discovered tubercle bacilli in the valvular vegetations in such cases; but the relation of endocarditis to tuberculosis is still in need of investigation. Dilatation of the heart, whether due to valvular defects or myocardial disease, exercises a retarding effect on the progress of pulmonary tuberculosis. Attacks resembling pseudo-angina pectoris may be encountered; and it is said that they occur more often where the left upper lobe is contracted and the heart much exposed. It is doubtful whether this association amounts to anything more than a coincidence.

Pericarditis is generally due to extension of tuberculosis from the pleura or anterior mediastinal glands, or occasionally from the peritoneum. In a few recorded cases a pulmonary cavity has perforated the pericardium, and produced pyopneumopericardium. Tuberculous granulations or caseous nodules may be seen in the serous membrane; or the tuberculous nature of the affection may only be demonstrable by the microscope. The effusion, as a rule, is scanty and sero-fibrinous, occasionally purulent or hæmorrhagic. There is always much fibrinous exudation, and usually more or less adhesion of the two layers. Tuberculous pericarditis generally escapes recognition during the patient’s life; though, from its weakening effect on the muscular wall of the heart, it must be regarded as an important complication.

Pulmonary embolism, from detachment of thrombi formed in the right ventricle or auricle, is an occasional occurrence. When hæmorrhagic infarction of the lungs ensues the condition may generally be diagnosed. But if no infarction be produced embolism may pass unrecognised, especially in moribund patients. Thrombosis of branches of the pulmonary artery may take place in the last stages, but this is not a common event. In some advanced cases we find great oedema of one leg from thrombosis of the large veins. Tenderness and induration can generally be discovered in the course of the affected vessel. Purpuric spots may appear on the lower extremities in conditions of cardiac debility.

Alimentary canal. — Tuberculous ulceration of the lip is extremely rare, but the tongue and other parts of the oral cavity are more often affected. Ulceration of the tongue appears most commonly on the dorsum,
but it may attack the sides, and occasionally the frenum. In cases of extensive tuberculosis of the soft palate and pharynx ulceration sometimes invades the buccal mucous membrane and the gums. The soft palate, uvula, and the pillars of the fauces are more often attacked; the prevailing lesion consisting of diffuse submucous infiltration and swelling, with shallow serpiginous ulceration. Miliary nodules may be seen in the base of the ulcer at times. Tuberculosis attacks the posterior wall of the pharynx less frequently than the palate. The usual lesions are circular ulcers with raised edges and granulations in the base, and superficial ulceration extending from the posterior pillars of the fauces. In some instances the larynx also is extensively affected, and the tuberculous disease appears to have originated there. But ulceration of the pharynx or tongue may occur without any laryngeal complication, and is generally due to infection from the sputum; but it may be part of a generalised tuberculosis.

Tuberculous ulceration of the oral cavity may be occasionally mistaken for syphilis, or for malignant disease. Herpes of the pharynx simulated miliary tuberculosis of the soft palate for a time in two tuberculous patients who came under my notice. For the diagnosis of such cases reference should be made to the article "Pharynx" (vol. iv. p. 745). In tuberculous affections of these parts pain is always a prominent symptom, and interferes greatly with the act of deglutition; in consequence of which the nutrition of the patient suffers seriously. Aphthous stomatitis is a fairly common complication in the terminal stages, and may occasion great discomfort.

The tongue presents no special features in phthisical patients, and its condition varies with the state of the oral cavity and alimentary canal. In cases of intestinal ulceration it is sometimes red, glazed and raw-looking; but similar appearances may be observed where no ulceration of the stomach or intestine exists. The red line on the gums, to which much attention was paid formerly, is by no means characteristic, and, moreover, is not very frequent.

Isolated instances of oesophageal tuberculosis have been recorded, but the gullet rarely shows any morbid change. Tuberculous ulceration of the stomach is extremely rare. A mammillated condition, pointing to chronic gastritis, is not uncommon. Chronic interstitial gastritis, atrophy of the glandular cells, and dilatation of the stomach have been found in some cases, but, as a rule, no morbid appearances are presented; the gastric symptoms are mostly dependent on functional derangements. Symptoms of dyspepsia, such as loss of appetite, cardialgia, flatulence, and constipation, are very common. Vomiting is often a very troublesome symptom: sometimes it is associated with a red irritable state of the tongue and epigastric pain, and is attributable to gastric catarrh; but more frequently it is unrelated to any affection of the stomach, and is excited by fits of coughing, which are apt to arise after meals and are possibly a result of hyperesthesia of the vagus. Attention to the state of the stomach and digestion is of great importance in the treatment of all cases.
The intestine is more often the seat of secondary tuberculosis than any other organ. In my post-mortem examinations the intestine was involved in 70 per cent of all cases of phthisis. The lesions are mostly situated close to the ileo-caecal valve; the last few feet of the ileum, and the caecum being most frequently attacked: but tuberculosis may show itself in any part of the alimentary canal from the duodenum to the anus. The fact that the process begins in Peyer's patches and the solitary follicles, where the lymphatic system is most highly developed, suggests that the virus is absorbed from the intestine; and it is probable that the bacilli are conveyed by sputum, which has been swallowed.

In the small intestine the ulcers are at first more or less rounded, and extend laterally, the edges and base being thickened, and the latter often studded with granulations or small caseous foci. On the peritoneal surface groups of miliary tubercles are often seen, with localised peritonitis; and on this surface whitish beaded cords, representing lymphatics filled with tuberculous material, may be traced from the ulcer towards the mesentery. In the colon the ulcers are more elongated in a transverse direction, and often partially or wholly encircle the gut. Thickening is less conspicuous than in ulceration of the small intestine, and subserous tubercles and localised peritonitis are seldom seen. Partial cicatrization of tuberculous ulcers is not uncommon, and at times stenosis may result. Owing to the thickening of the base of the ulcers, and the marked tendency to the formation of adhesions between neighbouring coils of intestine, perforation is generally prevented; but this accident is less rare than is generally supposed: the peritonitis which ensues will be restricted or general according to the presence or absence of adhesions. Circumscribed purulent peritonitis is a rare event; and, when occurring in the caecal region, is very liable to be mistaken for simple perityphlitis. The symptoms of intestinal tuberculosis are few and uncertain; they may be indicated as diarrhoea, localised pain and tenderness in the abdomen; but, unfortunately, none of these can be depended upon. Cases of the most severe ulceration of the small intestine or colon may run their course without any definite pain or tenderness, and may be accompanied by obstinate constipation from paralysis of the muscular fibres of the gut. Diarrhoea may be due to other causes, especially enteric catarrh and lardaceous disease. In the case of ulceration the stools may have a pale yellow or drab colour, but they commonly present no characteristic features. Local tenderness is more common with the diarrhoea of ulceration. In some instances the discovery of tubercle bacilli in the motions will put the diagnosis beyond all doubt. The presence of pus in the stools cannot often be detected, and is generally symptomatic of ulceration, in which case bacilli are likely to be found; but an abscess communicating with the intestine will have to be excluded: a large amount of pus would be in favour of an abscess. Small quantities of blood may be discharged with the motions, but copious hemorrhage is very rare: however, in two patients under my care death resulted from profuse bleeding. In one case only could a post-mortem examination be
obtained, and here a tuberculous ulcer of the colon was found to be the cause of the hemorrhage. In severe cases of ulceration the activity of the process in the lungs seems, at times, to become arrested.

Fistula in ano can sometimes be traced to a burrowing tuberculous ulcer of the rectum; but it is not uncommon, in cases of this description, to find the lower part of the bowel free from ulceration or obvious disease.

It is by no means certain that ischio-rectal abscess is always or indeed generally of tuberculous origin.

In two female patients who came under my observation, with advanced tuberculous ulceration of the intestine and rectum, the muco-cutaneous margin of the anus and the neighbouring skin were affected with a superficial serpiginous ulceration of similar nature.

The diagnosis of lardaceous disease of the intestine can only be arrived at when there are signs of similar disease of the liver, spleen, or kidney. Enlargement of the spleen or liver, with albuminuria, casts in the urine and polyuria, coexisting with diarrhoea, would strongly suggest lardaceous disease; but it must be remembered that lardaceous degeneration and tuberculous ulceration may exist in the same patient and in the same intestine. A marked degree of anemia is very general in cases of lardaceous degeneration. Transient diarrhoea is mostly attributable to simple catarrh, the diarrhoea of ulceration and amyloid disease being very persistent.

The liver may contain miliary tubercles, large caseous nodules, and occasionally tubercular abscesses; but, as a rule, these affections are clinically unrecognisable. In one case that I examined a hypophrenic abscess was caused by a perforating tuberculous abscess of the left lobe of the liver. Enlargement of the organ is most frequently caused by fatty and amyloid degeneration. The presence of a large spleen, albuminuria, and diarrhoea would be in favour of lardaceous disease, especially if the edge of the liver be thick and very firm. Cirrhotic enlargement is relatively of frequent occurrence in cases of chronic tuberculous peritonitis. It is possible that cirrhosis may be causally related to peritoneal tuberculosis. Miliary tubercles and extensive fatty degeneration are commonly associated with the cirrhosis of tuberculous subjects.

Enlargement of the spleen is a frequent symptom of lardaceous disease, and is only likely to be confused with the secondary splenic tumour of hepatic cirrhosis. In both cases the spleen is very firm. In acute generalised tuberculosis, as in other specific fevers, the spleen may be enlarged, whether it contain miliary tubercules or not; but its consistency is soft. Caseous nodules are often found in the spleen, especially in children, but they possess no clinical importance.

Tuberculous peritonitis may be part of a general tuberculosis, or it may be due to extension from the abdominal organs—intestine, lymphatic glands, and female generative organs; or it may be the result of infection from the pleura or pericardium, the bacilli being transmitted through the lymph spaces of the diaphragm.
Miliary tuberculosis of the peritoneum is often unaccompanied by any symptom whatever; but it may give rise to ascites, in which case some degree of chronic peritonitis will be found. In another form the tuberculous lesions consist of large nodules or masses, which are generally more or less caseous, but may at times be mainly or entirely fibroid. Caseous and fibro-caseous nodules may coexist in the same case. When the individual nodules coalesce large masses are formed which may be recognised by palpation during life. The great omentum is frequently much thickened, shortened and rolled up, forming a thick transverse band just above the umbilicus; but omental growths may be situated in the lower part of the abdomen also. The omentum may also undergo a general tuberculous infiltration, giving it the appearance of a thick apron hanging down in front of the intestine.

Retraction of the thickened mesentery, fixing the intestine against the spine, sometimes gives the appearance of a tumour. Large tuberculous masses may be found in the pelvis or in any part of the abdomen. When the pelvis is involved the disease has commonly originated in the female generative organs, the bacilli passing from the Fallopian tubes into the peritoneal cavity; but, at other times, the pelvic peritonitis may be secondary to disease in the upper part of the abdomen, the virus having apparently gravitated to the pelvis. In these cases the intestines are always much matted together, and patches of soft lymph, with crops of miliary tubercles, may be found, showing that the disease is still in progress. The fluid exudation is often purulent, but it may be serofibrinous or sanguineous. Softening of the caseous masses sometimes leads to perforation of the hollow viscera—intestine or bladder.

Partial or complete arrest of tuberculous peritonitis is by no means rare. Where cicatrisation takes place contraction may ensue and cause stricture of the intestine.

In cases of peritoneal tuberculosis the mesenteric and other lymphatic glands of the abdomen are always more or less enlarged and caseous. The glands may be the only abdominal organs affected, but intestinal lesions are very frequently present. Enlargement of these glands in adults is seldom so extensive as to admit of their being felt through the abdominal walls; but I have known caseous glands in the iliac and umbilical regions to form tumours as large as an orange. The tuberculous glands of children attain to a relatively larger size, and are more often susceptible of palpation; but even in children, and still more in adults, it may be difficult during life to decide whether a tumour be glandular or omental; though a deep situation and greater fixation of the tumour would be rather in favour of the former. The designation “tubes mesenterica” has been shown by Dr. Gairdner and others to comprehend not only tuberculosis of the mesenteric glands, but also tubercular peritonitis and other morbid conditions associated with wasting; and the name has consequently fallen into disuse.

Urogenital system.—Miliary tubercles and small tuberculous foci in the kidney may be accompanied by slight albuminuria, or may cause no
symptoms. In the important variety known as scrofulous or tuberculosis pyelitis, large areas of the kidney undergo caseous necrosis, and in most instances softening and ulceration ultimately ensue. Inasmuch as the process predominantly involves the pyramids and calices, disintegrated tuberculous material and pus are discharged with the urine from time to time. Tuberculous infiltration may ultimately involve the whole kidney, which then generally becomes enlarged, and may be converted into a loculated thick-walled cyst, containing soft putty-like caseous material; the dilatation of the pelvis being attributable to obstruction to the flow of urine. Both kidneys are affected as a rule, though one is usually in a more advanced stage of the disease. The infiltration and ulceration may extend from the pelvis of the kidney to the ureter, and thence to the bladder, prostate, vesiculae seminales, vas deferens and epididymis.

The symptoms of this form of renal tuberculosis are mainly the result of the pyelitis which constitutes the most salient feature of the affection—lumbar pain, mostly of dull character, but at times paroxysmal and colicky, when the ureter becomes obstructed, pus with a corresponding quantity of albumin, caseous debris, renal epithelium, and, at times, blood in the urine. Tubercle bacilli may be recognised in the urinary sediment, and are a conclusive proof of tuberculosis. If tuberculous disease of the bladder, prostate, and vesiculae seminales can be excluded, the existence of renal tuberculosis would amount to a certainty. Occasionally a definite renal tumour can be made out by palpation, but this is the exception.

In addition to the foregoing affection phthisical patients may acquire acute or chronic nephritis, lardaceous disease, and granular kidney. The commonest of these lesions is lardaceous disease. Slight degrees of this degeneration may need the application of iodine for their recognition, and in such cases no clinical symptoms would be presented. The higher grades of this disease are always combined with a varying amount of chronic nephritis, the kidneys in such cases being large, pale, and translucent, with yellowish opaque patches in the cortex. The surface is generally uneven, and the capsule adherent.

The amyloid disease affects principally the glomerular capillaries, but also the small arteries, the vasa afferentia and vasa recta. Degenerative changes in the convoluted tubules are due partly to the obstructive effects of the lardaceous disease of the vessels supplying these structures; and partly to the blood state, in which the lardaceous degeneration itself originated. In association with these changes a varying amount of scattered cell infiltration and fibrosis is nearly always found; these represent reactive inflammation secondary to parenchymatous degeneration. The urine in such cases is abundant, of low density, and contains albumin in considerable quantities, and hyaline casts. Dropsy is uncommon. The other forms of renal disease mentioned above present no features to distinguish them from similar affections in non-tuberculous subjects. Acute nephritis is uncommon, and is probably of haematogenous origin and attributable to absorption from ulcerative cavities in the lungs. Granular kidney is not uncommonly met with in elderly and
middle-aged persons, and is sometimes accompanied by slight degrees of lardaceous degeneration. It is very doubtful whether there be any causal relation between granular kidney and pulmonary tuberculosis. In cases where albuminuria supervenes a fall of temperature and a diminution of the activity of the pulmonary disease are not uncommonly observed.

Phosphaturia is said by Sir R. Douglas Powell to be an early indication of phthisis. Ehrlich’s diazo reaction is found in febrile progressive forms of tuberculosis, but no diagnostic significance can be attached to it. Tuberculous ulceration of the bladder is not very common, and is mostly associated with similar disease of other parts of the genito-urinary system. The symptoms are those of cystitis. Tubercle bacilli may be found in the urine. Tuberculous disease of the epididymis is much less uncommon; but this affection and tuberculosis of the prostate and vesicula seminales come rather within the sphere of the surgeon.

Tuberculosis of the uterus is decidedly rare. The disease, which attacks the lining membrane of the fundus, consists of tubercular infiltration, which is soon succeeded by caseous necrosis and ulceration. The uterine cavity commonly contains thick cheesy pus, and is apt to be somewhat dilated. There is rarely much enlargement of the organ. The Fallopian tubes are much more frequently attacked, and are seldom spared where the uterus is affected. In tuberculous salpingitis similar lesions are found in the mucous membrane; but the thickening and dilatation of the tubes attain to much greater proportions.

Tuberculosis of the ovary is one of the rarest occurrences: the only case I have seen is recorded by Dr. Habershon. In this case both ovaries contained tuberculous abscesses which communicated with the Fallopian tubes and intestine.

Tuberculous peritonitis is not uncommonly attributable to extension from the Fallopian tubes or uterus. It is probable that genital tuberculosis may also be caused by infection from the peritoneum; but more often the disease is communicated through the blood. The possibility of direct sexual infection cannot be denied.

Some writers have contended very strongly that pregnancy exercises a retarding influence on the disease; others hold that phthisis is aggravated by this condition: on the whole, pregnancy seems more often to intensify the symptoms of pulmonary tuberculosis. There is little doubt as to the injurious effects of parturition. It is a common experience that after confinement the pulmonary disease makes rapid progress, and is apt to terminate fatally in a few months. Hanau believes that this is to be explained by the inhalation of infective material from cavities into healthy parts of the lungs during the forcible inspirations that accompany expulsion of the foetus. The exhausting influence of lactation is notorious. Menstruation is nearly always much deranged, apart from any definite lesion of the generative organs. Amenorrhea, or scanty, infrequent menstruation, is the rule in this disease, and may be one of the earliest symptoms of it. Very occasionally menorrhagia occurs, but is seldom persistent.
The suprarenal bodies occasionally contain isolated caseous nodules, which cause no symptoms. Still more rarely both adrenals are converted into firm caseous or caseo-calcereous masses, in which case bronzing of the skin and other symptoms of Addison's disease supervene.

Osseous system.—Secondary tuberculosis of the osseous system and joints is not very common, and may show itself, among other places, in the vertebræ, sternum, and ribs, giving rise to chronic abscess in connection with the chest walls. This subject possesses more surgical than medical interest.

Nervous system.—The mental attitude of many phthisical patients is one of irrepressible hope, especially in the less chronic forms. Such persons often asseverate that if they could but get rid of some particular symptom, such as cough or shortness of breath, they would be perfectly well; and they go on making plans for the future within a few hours of their death. But in most cases presenting definite symptoms of mental derangement depression is the prevailing feature. Melancholia, stupor, delusions of suspicion or persecution, religious foreboding, insomnia, hallucinations, a suicidal tendency, and refusal of food are among the commonest symptoms. Maniacal excitement is much less frequent. For further information the reader is referred to the section on Insanity in the last volume of this work.

Tuberculosis is much less liable to affect the nervous system in the course of chronic phthisis than in acute tuberculosis. It is also of much more frequent occurrence in children than in adults. In most cases the tubercle bacilli are conveyed through the blood. The cerebro-spinal meninges are the parts most commonly attacked, the tuberculous process being grouped especially along the small vessels. The growth of tubercles is soon followed by fibrinous exudation, in consequence of which the pia mater becomes much thickened.

Meningitis nearly always predominates, or is exclusively localised at the base of the brain, and extends thence to the Sylvian fissures, the ventricles, the surface of the cerebellum, the pons Varolii, and the medulla. The ventricles are often much dilated and filled with turbid fluid—"acute hydrocephalus" of the old writers, the convolutions becoming flattened by pressure. The cortex of the brain and the walls of the ventricles are often much softened, from extension of the inflammation of the pia mater, so that the process is more correctly described as a meningo-encephalitis. Tuberculous nodules or masses may grow in the brain tissue, and sometimes attain to a considerable size. These solitary tubercles or tuberculous tumours are found most frequently in the cerebellum and cerebral hemispheres, but they may arise in any part of the brain and are often multiple. Small tuberculous nodules are not infrequently found in the cortex, extending inwards from areas of chronic tuberculous meningitis.

Lastly, meningitis, encephalitis and myelitis may be due to extension from neighbouring bones of the cranium or spine.

The symptoms of meningitis are many, and can only be briefly
enumerated:—headache, irritability of temper, fretfulness, coma, convulsions, marked retardation, acceleration, or irregularity of the pulse, Cheyne-Stokes respiration, vomiting, retraction of the head and abdomen, rigidity and weakness of limbs, paralysis of cranial nerves, optic neuritis. Retention of urine is very common towards the close, and pyrexia is nearly always present. Headache is perhaps the most common symptom in the more chronic form. Tuberculous tumours of the brain give rise to symptoms not differing from those of other cerebral tumours. For a full account of this subject reference must be made to the appropriate articles.

Peripheral neuritis has been observed in a small number of cases in the form of extensor paralysis of the arms or legs. The cause of the neuritis is uncertain. It may be the result of toxins, elaborated by the tubercle bacilli.

Some of the pains and tenderness affecting the limbs in phthisical patients may possibly be of neuritic origin. Beau grouped these together under the name "melalgia." It is difficult at present to discriminate the pains which many patients in advanced phthisis complain of. Some are probably neuritic, others myalgic; while, in some instances where pains fly about from one part to another and affect the joints, the resemblance to rheumatism is very close. In these last the rheumatoid pains are possibly a septicemic symptom, depending on absorption from pus-secreting cavities in the lung. Suppurative otitis media is not very uncommon, but it is seldom that tubercle bacilli can be discovered in the pus.

Course.—The course of pulmonary tuberculosis is essentially variable and fluctuating, intervals of quiescence or apparent arrest alternating with prolonged periods of fever and other constitutional symptoms. In a large percentage of cases the disease is for the most part slowly progressive, and death ensues in a few years at the latest. The average duration of phthisis has been variously estimated. Louis found that in more than half the cases observed death occurred in less than nine months. The mean duration has thus been stated:—twenty-three months (Louis and Bayle); two years (Laennec and Andral); four years (C. J. B. Williams and Sir J. Clark); Dr. C. T. Williams, from analysis of a thousand cases among private patients, put it at seven years and three-quarters. On account of the great difficulty so frequently met with in attempting to fix the date of onset of the disease such calculations are fraught with uncertainty. Those physicians who have had much experience of the disease at special as well as general hospitals, will probably agree that statistics derived from the latter source exclusively would give a very erroneous impression of the duration of phthisis. Patients admitted into general hospitals are either exceptionally ill, or are suffering from some serious complication. The mortality among such patients is naturally very high, and the duration of the disease may often be measured rather by months. Most valuable are the statistics collected by Dr. J. E. Pollock from 3500 cases of phthisis attending the out-patient department of the Brompton Hospital.

"Here (among the out-patients)," as he truly says, "are seen indi-
individuals of all classes, excepting the highest, and of all ages and occupations. The necessities of home cares and of continuing the daily work are but little interfered with by a visit once a fortnight to their physician; but these urgent claims of domestic life shut out large numbers from the possibility of availing themselves of indoor treatment in a hospital. The large class affected with chronic slow phthisis are, therefore, found chiefly among the out-patients.

"The average duration, while under observation, of all the cases taken together was two years six months and three-fifths nearly, but this represents only a part of the period of the affection, and in it are included cases of the most acute and rapid form as well as those which have become chronic."

The actual duration of the cases must have been considerably longer, and the whole average duration of the disease, as Dr. Pollock says, must be raised beyond four years. An experience of twelve years' out-patient work at the Brompton Hospital has convinced me that Dr. Pollock is far nearer the mark than those who would limit the average duration to two years.

The complexion of the malady, while running a chronic or slowly advancing course, is liable at any time to undergo a complete change, depending on acute exacerbation of the pulmonary disease. Fever and other constitutional symptoms often herald renewed activity of the tuberculous process before physical examination gives any decided indication of extension. In other instances we find the signs of disease slowly extending for some time without any corresponding aggravation of the patient's symptoms.

The lines along which the disease spreads in the lungs have been described in the section on the pathology. It is very important not to be satisfied with exploration of the front of the chest only, but to examine with care the back also, more especially the supraspinous fossa—that is, the posterior aspect of the upper lobe—and the interscapular region just below the spine of the scapula, which corresponds to the apex of the lower lobe, a part specially prone to secondary tuberculosis. And, as Dr. J. K. Fowler quite rightly insists, search should be made for signs of disease extending from behind forwards from the apex of the lower lobe along the upper border of the same lobe, the position of the septum dividing the upper and lower lobes being roughly indicated by the "vertebral border of the scapula, when, with the hand upon the spine of the opposite scapula, the elbow is raised above the level of the shoulder."

The upper part of the axilla is another region that must be carefully investigated, as it is in this space alone that the outer aspect of the upper lobe is accessible to examination; and signs of excavation may sometimes be found at the apex of the axilla only.

Towards the close of life bubbling râles are generally heard over the whole of the chest, and are an indication of pulmonary oedema, the result of cardiac failure. It is usual to find resonance to percussion over the lower part of one or both lower lobes up to the very end; a fact which
is to be explained by the persistence of patches of spongy lung between the tuberculous masses.

Where the fatal termination is not directly or indirectly dependent on complications, but is the result of slowly extending disease, death most frequently occurs from exhaustion. Asphyxia is seldom the cause of death except in acute forms of tuberculosis. In most chronic cases death is preceded by profound emaciation and debility, which steadily increase in spite of the considerable quantity of nourishment the patients often, continue to take. Bed-sore may form if the nursing be not vigilant, and edema of the legs is not uncommon. The pulse becomes more rapid and feeble, the temperature gradually falling often becomes subnormal, tracheal râles appear, and the end comes quite peacefully. In the comparatively few cases in which complete arrest of the disease takes place, the constitutional and local symptoms gradually subside, and the patient regains his health. The physical signs at the same time undergo certain modifications, or occasionally disappear entirely. In most cases, although râles and other adventitious sounds cease to be heard, signs of consolidation and contraction of the apex persist, and some degree of localised emphysema is often developed.

Prognosis.—Of the many complicated problems presented to the physician the prognosis of pulmonary tuberculosis is one of the most difficult. An accurate prognosis would involve full knowledge of the parasite and its host, as well as of their environment. At present little is known concerning variations in the virulence of the tubercle bacillus as it occurs in the body of man.

Still less information is forthcoming as to the histo-chemical and biological conditions of the human organism which retard or favour the development and activity of the parasite. Certain facts concerning the external conditions that appear to exert a salutary or injurious influence on the disease have been discussed under its causation. A complete understanding of these points is intimately connected with the question of immunity, a most difficult subject, which is only just beginning to be studied. For practical purposes we have to estimate the prognosis, in the first instance, by a careful consideration of the effects of the disease, immediate and remote, in each patient. By these means we are able to gauge, approximately, the severity of the malady and the resisting power of the individual. Furthermore, an acquaintance with the natural history of tuberculosis, including the influence of heredity, of previous or concurrent diseases, and of various conditions of life, and lastly, the knowledge of the effects of treatment, will be required if we would forecast the probable course of pulmonary consumption. The symptoms of the patient, representing the result of disordered function, are of the first importance. Of all the general symptoms fever is the most important. A markedly intermittent or remittent pyrexia, in the absence of acute intercurrent affections, is very significant of progressive disease, and is, therefore, of bad augury. At the same time it must be remembered that a considerable degree of fever is not incompatible with gain of weight and
other signs of improvement. Moreover, after periods of severe pyrexia
the temperature may fall, and the disease enter upon a chronic phase.
Nevertheless, it may be accepted as a general principle that the existence
of marked pyrexia always necessitates a very guarded, though not
necessarily an entirely unfavourable prognosis.

A slight evening rise of temperature, with a fall to normal or slightly
below normal in the morning, is not uncommonly present in comparatively
favourable cases. The supervention of fever in the course of a mild
chronic case is often one of the first indications of renewed activity of the
tuberculous process, which may prove intractable. The absence of fever
does not in itself justify the expression of a hopeful opinion, for, as we
have seen, an apyrexial temperature may accompany advanced and active
disease. Subnormal or collapse temperatures have a very ominous
import. Emaciation signifies deficient alimentation (whether due to
insufficient feeding, digestion, or assimilation), or profound constitutional
intoxication. In the first case the cause is more amenable to treatment,
and the outlook is consequently less unfavourable. A persistently rapid
or easily excited pulse is indicative of debility, or of a state of general
nervous erethism, both of which are very undesirable features. Anemia
and debility are also an evidence of profound constitutional impression,
and must therefore darken the prognosis.

Among the more important symptoms of local disorder we must
reckon dyspnoea depending on diffuse or acutely extending pulmonary
changes. When these changes consist in disseminated miliary tubercles,
or in lesions of the broncho-pneumonic or pneumonic type, the gravity of
the symptom can hardly be exaggerated. Expectoration, profuse, puru-
lent, and containing numerous elastic fibres, implies progressive destruction
of lung. Absence or scantiness of expectoration is, at times, a marked
feature in severe cases: this is mostly, but not exclusively, seen in
children and women, who often swallow their sputum. But while no
great importance can be assigned to the quantity of the sputum, scanty
or moderate expectoration is on the whole a good sign.

The expectoration of pulmonary calculi is never met with except in
very chronic cases.

The significance of fœtor varies with its cause. When the odour has
a sickly or slightly fishy character, due to the retention of secretion in
cavities, it is of less moment than when it possesses the penetrating odour
of bronchiectasis: in the latter case, the dangers of septic broncho-pneu-
monia and other accidents are added to those already existing. The
supervention of gangrene renders the prognosis quite hopeless.

The number of tubercle bacilli in the sputum is no accurate measure
of the extent or severity of the disease, and is largely a question of dis-
charge. In some acute cases the bacilli may be very scanty, whereas in
other cases, quiescent and circumscribed, the sputum may teem with them;
complete and permanent disappearance of the microbes is a most hopeful
sign; but their continued presence in the sputum does not preclude a
protracted and favourable course.
An incessant and intractable cough, especially when it interferes with sleep and causes vomiting, adds greatly to the exhaustion of the patient. Some of the most irritable coughs depend on catarrhal affections of the upper air-passages, and can often be relieved; but cough associated with signs of persistent diffuse bronchitis is often indicative of widely disseminated tuberculous lesions.

The state of the digestion is of the greatest importance. Where the symptoms of gastric disorder, or of faulty absorption or assimilation, prove rebellious to treatment the prospects of improvement are small indeed.

In attempting to weigh the indications of physical examination of the lungs, the two chief points requiring attention are the character and the extent of the disease. An acute onset is commonly followed by progressive invasion of both lungs, and has the gravest significance. An insidious, bronchitic, or hemoptoic onset is more favourable. Rapidly extending disease is always of ominous significance. Râles and other morbid signs scattered widely over a large part of both lungs, especially in pyrexial cases, point to disseminated lesions, a most unfavourable type of disease; but similar physical signs, without much fever, may sometimes persist for months or years in cases where the disease takes the form of discrete fibro-caseous or fibroid processes. Cases with severe symptoms and relatively slight physical signs are to be regarded with suspicion, for the true extent of the pulmonary disease is generally masked by other conditions; on the other hand, the presence of marked signs of consolidation or excavation of one upper lobe is not inconsistent with a chronic and favourable course so long as the lower lobe and the opposite lung remain comparatively free.

Signs of contraction are a sure index of chronicity. Localised and stationary disease is a good element in prognosis. The disappearance of râles is, in general, a favourable feature.

After what has been said in a previous section about the stages of phthisis, it is futile to base the prognosis on considerations which are so apt to be fallacious. If, in a chronic case, we could be sure, which we cannot be, of the absence of softening and excavation, the prospects of arrest would be better than if cavities had already formed, for the existence of a cavity carries with it the risk of extension by means of inhalation of infective secretions into distant bronchi. Moreover, there is no evidence that a vomica can become obliterated by cicatrization; whereas we know that tuberculous nodules often undergo healing by encapsulation, calcification, or fibrous transformation.

Among the most ominous complications are meningitis and pneumothorax. Pleurisy with effusion sometimes appears to exert a retarding influence on the pulmonary affection. Empyema is unfavourable. Dry pleurisy is regarded by some authors as a very unfavourable sign; but this is by no means generally true. The appearance of the diffuse infiltrating form of laryngeal tuberculosis, with its tendency to produce dysphagia and stenosis, betokens a speedy termination. Oft repeated hæmoptysis depresses the patient morally as well as physically; and
under such circumstances the possibility of a sudden and fatal issue has always to be reckoned with.

Tuberculous peritonitis and intestinal ulceration cause great wasting and prostration, and generally hasten the patient's end. Tuberculosis of the abdominal lymphatic glands and generative organs tends to aggravate the general condition, and is commonly a sign of generalised disease.

 Pronounced lardaceous disease of the viscera is a most serious complication of chronic cases, pointing, as it does, to profound derangement of nutrition. A combination of diabetes and phthisis is also a most grave condition.

The presence of cardiac hypertrophy and dilatation, or of marked emphysema, justifies the opinion that the duration of the disease will be long.

The environment is a matter of much importance. A patient living in a healthy country place, under suitable climatic conditions, has better prospects than one who is compelled to dwell in a large town, especially if his life be spent in dusty or smoky rooms. Again, pecuniary means have a direct bearing on the prognosis: those who can procure, not only the necessaries, but also the luxuries of life, and can afford to rest, are in a better position to battle with the disease than those who must work hard for a living. Nevertheless, among poor hospital patients we see, not very infrequently, persons who have been suffering from phthisis for ten years or more, and who still go on working under the most adverse circumstances. A history of previous good health is a hopeful feature, as a greater capacity of resistance may be expected where the general health has not been already undermined. The influence of age has been much disputed. As a general rule, pulmonary tuberculosis runs a more rapid course in children and young adults than in older persons, among whom the chronic form is rather the rule. Cornil and Héraud suggest that tuberculosis is more chronic in old people, because heredity has already weeded out those of least resistance. Nevertheless, acute disease may occur in elderly patients, and, conversely, the phthisis of children may be chronic. Each case must be estimated on all the data; and the influence of age can only be credited with a very subordinate importance.

It has been said that the duration of the disease is shorter in women than men. If we exclude the cases associated with pregnancy and parturition, it is doubtful whether this statement be true.

The influence of heredity is undoubtedly an important one. It is a common belief that this factor determines the earlier manifestation of the disease. A strong predisposition is an unfavourable element, as in such cases there often appears to be a general lack of vitality and resistance. But, although this is generally true, hereditary influence cannot be ranked on a level with considerations derived from a careful estimation of the effects of the disease in the individual patient. The best results may be expected in cases presenting the following features:
apyrexia, or a subfebrile temperature; weight stationary or increasing; signs of disease confined to one lung or to limited portions of both lungs (especially if associated with contraction); a quiet pulse and nervous system; a good digestion; absence of serious complications; a good family and personal history, and favourable hygienic surroundings.

Treatment. — A. Preventive. — If, as our present knowledge appears to show, the sputum of tuberculous persons be the main source of the disease, it is obvious that the complete destruction or disinfection of this secretion should be our first duty. In many hospitals this is effected by means of special destructors, or furnaces, in which the sputum is burnt. In private houses, where this method is difficult of application, the expectoration, after previous disinfection, may be discharged into the drains. For general purposes carbolic acid in a strength of 5 per cent is the best disinfectant; experiment shows that the infectiveness of the bacilli is completely removed after exposure of the sputum to this solution for a short time. Sputum should not be thrown on the dust-bin, where the contents may dry and become a further source of danger. In all cases, whether in hospitals or private houses, patients should be directed to use spittoons containing a suitable disinfectant. If, in spite of advice to the contrary, patients use handkerchiefs for receiving the sputum, these should be burnt; or at any rate should be scalded before being sent to the wash.

Persons suffering from phthisis should be warned not to spit about the streets, or the house, or into any vessel which does not contain some disinfectant. Underclothing, linen, sheets, and pillow-cases should also be scalded before being washed, especially in the case of bed-ridden patients, when the chances of contamination are greater. Phthisical persons should occupy separate beds. Bedrooms and sitting-rooms so occupied must be carefully cleaned with a damp cloth, so as to avoid raising a dust; and should be well aired and exposed to light every day. Rooms that have been inhabited by such patients should be thoroughly cleaned, and, if possible, white-washed, painted, and re-papered before being used by other persons.

It is desirable that patients should be provided with separate sets of knives, and forks, and spoons; but, in default of this precaution, all table utensils, as well as plates, cups, glasses, should be scrupulously cleaned.

Milk is undoubtedly a vehicle of disease, and should be carefully boiled; particularly when intended for children. For the principles on which slaughter-houses and dairies should be regulated, and for further information on the general question of prophylaxis, the article "Tuberculosis" (vol. ii. p. 30) should be consulted.

In persons threatened with tuberculosis, and in others with a strong family predisposition, the importance of a good general hygiene can hardly be over-estimated.

Abundance of fresh air in the dwelling—especially in bedrooms—secured by suitable methods of ventilation, a large amount of outdoor
life in pure country air, a generous diet, including a large proportion of fatty constituents, daily cold sponging of the body, and the use of flannel or similar underclothing; are amongst the most necessary conditions. In the case of children the throat needs special attention; enlarged tonsils should be removed, and catarrhal affections must not be neglected. The opinion is gaining ground that the tonsils are frequently the portals by which tubercle bacilli enter the body; at any rate, in primary tuberculosis of the cervical lymph glands. The question of the removal of caseous glands, and the surgical treatment of tuberculous disease of bones and joints, are matters of great importance, but cannot be discussed here. [Vide vol. iv. p. 599.] Tuberculous mothers ought not to suckle their infants.

The choice of a profession or trade is a matter of no small consequence. Occupations in which life is mainly or largely spent in the open air are the most favourable; but, in the case of the poor, outdoor work generally implies more or less heavy labour, which is often prohibitive under the circumstances. Many people, in whom tuberculous affections of bones, joints, or lymphatic glands have been cured or partially arrested, manage to carry on successfully various sedentary trades or professions. Dusty occupations, as in the case of millers, bakers, knife-grinders, stone-masons, and the like, are fraught with special dangers to vulnerable persons. Free ventilation of dusty workshops is all-important, and serves to minimise, to a large extent, the dangers of the aforesaid trades.

There can be no doubt whatever that persons suffering from progressive disease, ought not to marry. In cases of quiescent or apparently arrested tuberculosis there is room for difference of opinion. When all symptoms of disease have disappeared, the sputum no longer contains bacilli, and the general health remains good, marriage, in the case of men, may be undertaken after the lapse of two or three years without any great risk. Women incur far greater danger in connection with pregnancy, parturition, and lactation; for it is well known that, under the influence of such conditions, quiescent tuberculous lesions are apt to prove the starting-point of active disease. If, however, the tuberculous process can only be regarded as quiescent, and bacilli continue to be expectorated, marriage ought to be forbidden in either sex. Most writers agree on this point as regards women; but some have urged that men, under these circumstances, may be allowed to marry on the ground that their lives are thereby made happier; and that, if children should be begotten, they tend to die off early, and the race does not appreciably suffer. The morality of such advice need not be discussed here; but the possibility of a phthisical husband directly or indirectly infecting a healthy wife cannot be disregarded; and the risk of adding to the already high tuberculous death-rate is one that no medical man should willingly countenance. However, as all writers point out, the question of marriage is seldom decided, solely or even mainly, on medical grounds.

B. Specific treatment.—It has been well said that where the number of
remedies recommended for any disease is large, there is good reason for the belief that none of them is possessed of much efficacy. Of no disease may this more truly be said than of tuberculosis. Tuberculin, a glycerine extract of pure cultures of tubercle bacilli, deprived of the bacilli by a special method of infiltration, and injected under the skin, was found by Koch to exert a marked influence on tuberculous lesions. The local action of the remedy consists in an inflammatory swelling and disintegration of the diseased foci. Constitutional reaction is indicated by fever, malaise, headache, pains in the limbs and trunk; and occasionally by nausea and vomiting. The effects of the remedy were most conspicuous in cases of lupus, where the changes in the skin could be readily followed.

Although Koch's statement that tuberculin had a specific influence on tuberculous lesions was speedily verified, his claim that the action was curative was not so generally admitted. Pathological evidence was soon brought forward by Virchow and others to show that the use of tuberculin was often followed by the development of acute inflammatory changes in the lungs; and that under its influence quiescent disease may spring into activity and lead to generalised tuberculosis. Space will not admit of a detailed discussion of this matter. The prevailing opinion at the present time is that the administration of tuberculin in cases of pulmonary tuberculosis is dangerous, though some surgeons still regard it as a useful adjunct to other methods of treatment in surgical forms of tuberculosis.

Klebs and Dr. W. Hunter, working independently, claimed to have succeeded in separating the fever-producing and toxic elements from the direct curative constituents of tuberculin; but their extracts failed when put to the test by other observers. Quite recently Koch has described a new method by which he was able to prepare an improved tuberculin. Dried cultures of the tubercle bacillus were thoroughly triturated in a mortar, then mixed with distilled water and centrifugalised. The sediment was again dried, triturated, suspended in distilled water, and centrifugalised. This procedure was repeated until no sediment remained. The liquid separated by the first centrifugalisation contains the active principles of the original tuberculin, while the fluid obtained at subsequent stages of the process is believed to contain the débris of the disintegrated bacilli themselves. This solution has the great advantage that it produces no constitutional disturbance beyond a slight, rise of temperature. The new tuberculin was found to confer immunity on guinea-pigs, and its use in the human subject in cases of lupus and early pulmonary tuberculosis was followed by improvement. Further experience can alone decide as to the therapeutic value of the new preparation; but it is no exaggeration to say that in a further development and improvement of Koch's method lies our best hope of arriving at a successful treatment of tuberculous disease.

The number of drugs that have been vaunted as specific for tuberculosis is legion. In recent years iodine, iodoform, carbolic acid, corro-
sive sublimate, creasote and one of its constituents, guaiacol, have been most largely used, in virtue of their antiseptic properties. These have been administered by the mouth, by inhalation, subcutaneous injection, inunction, and direct injection into the lung; and sulphured hydrogen gas has even been pumped into the rectum. The results have not differed greatly in respect of any of these methods. They have all passed through successive stages of exaggerated and hasty laudation, half-hearted approbation, and contemptuous neglect. The history of guaiacol is a good illustration: at first it was advocated as a specific; now the only claim seriously made in its favour is that it has a beneficial effect on cough and expectoration.

As we have no specific remedies, our aim must be to increase, as far as possible, the resisting power of the patient, so as to put him in the best condition to withstand the inroads of the disease.

In order to maintain the nutrition of the body at as high a level as possible, the dietary must be liberal, and should include a large amount of fat in the shape of milk, cream, butter, fat bacon, and the like, in addition to a due proportion of nitrogenous and carbohydrate constituents. A special distaste for fatty foods is manifested by some patients, but this aversion is by no means so general as certain writers would have us believe. Cod-liver oil is a valuable adjunct to the diet, but is possessed of no specific virtues. This valuable food is sometimes prescribed in a manner calculated to bring it into discredit, that is, when it is administered in too large quantities. Two to four drachms twice or thrice in the day is as much as most patients can digest, and it is frequently necessary to begin with even smaller doses. It usually agrees best when taken soon after a meal; but some people prefer a single dose at bedtime. In all cases of dyspepsia, and whenever the taste of the oil keeps rising into the mouth, it should be withheld. If persevered with under these circumstances it seldom fails to derange the digestion. It is better for the patient to enjoy his food without the oil, than to persist in its use and lose his appetite.

The taste of the oil may be disguised with peppermint, lemon juice, ginger or orange wine, cognac, liqueurs, and other flavourings. Many patients prefer to take the oil in the form of an emulsion, or in combination with malt extract. Malt is much used at present in England, and no doubt it possesses some digestive value; but it is no substitute for cod-liver oil or fats. Glycerine, in doses of three to four tablespoonfuls daily, has been recommended as a substitute for the oil by Jaccoud, but it has not found much favour with other physicians. Alcohol is not to be ordered in all cases indiscriminately; where the disease is quiescent, nutrition fairly well preserved, and the appetite good, it is not required; in conditions of debility, deficient appetite, and, above all, in pyrexial cases, alcohol is of great value.

The particular form in which stimulants are to be administered is largely a matter of taste. Ale and stout are preferred by many patients. In pyrexial cases brandy, whisky, or some form of spirit, seems often to
suit best; and the quantity that can be taken with advantage under these circumstances may be very large. Alcohol has no influence in promoting reparative sclerotic changes, as some have asserted. In cases of obstinate anorexia forced feeding by means of the stomach-tube has been found useful by Debove and others. Massage is occasionally useful, especially where debility is a prominent symptom, and is unaccompanied by pyrexia.

In addition to the utmost attention to the matter of food the rules of general hygiene must be carefully observed. The patient's house ought to be well drained, built on a light porous soil, and, if possible, it should face the south. The rooms, and especially the sleeping-apartments, must be well ventilated and suitably warmed. It is hardly possible to over-estimate the value of fresh air and sunshine. Regular exercise, walking, riding, outdoor games of the less violent kind, such as golf, cycling, shooting, and fishing, may all be practised with moderation if the patient's general condition be good, and if there be no pyrexia. Where this is not possible, the patient, in favourable weather, may go out in a bath-chair or in a carriage; or he may sit out of doors in a suitably arranged shelter. Even when he is entirely confined to bed with fever, wasting, night sweats, and symptoms of progressive disease, much benefit may still accrue from wheeling the patient's bed out of doors into a sheltered spot, or into a sunny balcony, as is done at Falkenstein and other places; bed-ridden, persons may, in this manner, spend the greater part of the day in the open air with great advantage. The clothing should be warm and yet light, and woollen garments should be worn next the skin. Comforters for the neck and chest-protectors, which encourage hyperaemia and increased sensitiveness of the skin to changes of temperature, are unnecessary and unadvisable. Woolen socks or stockings and thick boots are required to prevent the feet from getting chilled. Respirators worn over the mouth are not now so much in vogue as formerly. If the patient breathe through his nose, as he ought to do, a respirator is superfluous. When a strong, cold wind has to be faced, a light shawl or thick veil may be wrapped round the face for temporary protection.

The skin may be rendered less sensitive to changes of temperature by the daily use of the cold shower bath or douche in the morning; but in the case of more delicate patients, with feeble circulation, a warm bath followed by cold sponging is preferable. Early hours, the avoidance of crowded rooms, theatres, and smoking-rooms, a life free from excitement, and occupation for the mind, such as reading, drawing, chess, billiards, and other indoor games, are to be recommended. Instrumental music may be practised, but singing is not advisable, except for the more robust patients, in whom the disease is quiescent; though Walshe gives instances of singers continuing to take leading parts in the opera while suffering from pronounced pulmonary disease. Medical direction is most desirable; and the success of some Continental health resorts is doubtless attributable in a large measure to the careful and strict superintendence
of the physicians in charge. But the majority of English patients find such a rigid supervision irksome and disagreeable; and hitherto such establishments have not been in much request in this country.

In selecting a suitable climate we must be guided by certain general principles. Purity of the atmosphere, and especially freedom from dust of all kinds, and abundant sunshine, are the fundamental requisites. Questions of altitude, temperature, and moisture of the air, and geographical considerations in general, are still matters of dispute, and are discussed elsewhere in this work.

If the patient's surroundings satisfy the requirements just indicated, it is unnecessary, in many instances, to advise a permanent change of residence. In the case of wealthy people it may be desirable to send them away to some health resort, where they will be more ready to submit to strict medical supervision and direction than at home. When the patient lives in a large town he should be recommended to remove into purer air, if his means permit. It is useless and cruel to send patients with advanced disease to a distant health resort. To such persons the fatigue of a long journey may have disastrous consequences, and the loss of home comforts cannot be compensated even by the best of climates.

If the general health be well maintained, and the pulmonary disease be neither very active nor extensive, great benefit may be obtained by spending the winter, or better still, by continued residence at the high alpine stations, such as St. Moritz, Davos, and the like. Better results are generally obtained in men than in women, as the tastes and the habits of men impel them to take a fuller advantage of the opportunities of outdoor life and exercise presented by an alpine climate. Under similar conditions of health, emigration to Colorado, the Rocky Mountains, and the high levels of South Africa offers good prospects to young men.

Emphysemæ, laryngeal tuberculosis, and manifestations of nervous erethism are generally regarded as contra-indicating residence at the high alpine stations. Such cases are more adapted for Egypt, the Riviera, Madeira, the Canaries, or the south coast of England. For a detailed discussion of this subject the reader is referred to the article, "Climate in the Treatment of Disease," vol. i. p. 247.

C. Symptomatic treatment.—In combination with the general hygienic measures that have been briefly sketched the exhibition of certain tonic drugs is often very useful. The most valuable are strychnine or nux vomica, arsenic, and quinine. Opinions differ as to the relative value of these, but strychnine appears to deserve the first place. Arsenic has no specific influence on the disease, but it may do good service in its capacity of a nervine tonic; the same may be said of quinine. Iron has still a great reputation with some physicians; but it does not suit all patients, especially the large class that suffer from a tendency to gastric catarrh; and it has little effect on the anemia of phthisis. In persons who can take a fair amount of exercise, and have a good
digestion, a short course of iron, either alone or in combination with arsenic, is sometimes attended with good results. The hypophosphites of lime and soda have been largely tried, and are still much used in this country. They are certainly not possessed of any direct action on the tuberculous process, and their tonic effects have been greatly overestimated.

Fever.—Quinine has been extensively used for the purpose of reducing fever, especially on the Continent; but it is generally allowed that in order to obtain this result 20 to 30 grains must be given in a single dose, or divided into four or five doses, to be taken at short intervals some hours before the temperature begins to rise. Even when administered in such quantities the antipyretic action of quinine is but slight, and the stomach is often deranged by the drug. The combination of quinine, opium, and digitalis, known as Niemeyer's Pill, has long enjoyed a great reputation in pyrexial cases; but, although its general effects are sometimes salutary, it is rarely very efficient in the reduction of temperature.

Of late no small number of antipyretic drugs have been employed—salicylic acid, salicylate of soda, antipyrin, thallin, phenacetin, antifebrine, and many others. The most effectual seem to be antipyrin and antifebrine, which, when given in sufficient doses, undoubtedly effect a considerable fall of temperature. Antifebrine is a very powerful remedy, but its action is somewhat uncertain. It is never advisable to prescribe larger doses than two or three grains to begin with: in these quantities it may be repeated at short intervals till eight or ten grains have been taken. Unfortunately the reduction of temperature produced is but temporary, and no further effect on the disease is produced. Moreover, the prolonged use of antifebrine and antipyrin is extremely depressing, and causes profuse sweating. Wilson Fox believed that the continued use of small doses of these remedies and of salicylate of soda had a beneficial result on the general condition, although the range of temperature was not appreciably affected.

Tepid sponging during the pyrexial periods sometimes gives considerable relief, even if it fail to reduce the temperature of the body to any great extent. The use of the cold or tepid bath finds few advocates on account of the further depression which, in the prostrate condition of such patients, is apt to follow its use.

Sweats.—Atropine in doses of \( \frac{1}{12} \) th to \( \frac{1}{5} \) th of a grain, given at bedtime, is the most effectual agent we possess for checking sweats. A combination of extract of belladonna and oxide of zinc is also useful, but it is inferior to atropine. Picrotoxin, \( \frac{1}{120} \) th to \( \frac{1}{80} \) th of a grain (Murrell); strychnine, 10 \( \text{mL} \) of the liquor (Lauder Brunton); and agarin may also be employed with advantage in some cases. In the slighter cases, to sponge the skin with toilet vinegar and water may be sufficient. Arsenic is recommended by some authors, but when the sweats are profuse it has little influence.

Cough.—A moderate cough is the natural consequence of pulmonary
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disease, and needs no special treatment; moreover, where secreting cavities exist, effective cough is most desirable. But when the cough is very violent, spasmodic, or incessant, and the patient becomes much exhausted thereby, it is necessary to treat this distressing symptom directly. In order to do this with success we must first discover the source of the cough. The more violent the fits of coughing, the more likely are we to find that the cause is situated in the larynx or main air-passages. When the larynx is the seat of ulceration or inflammation local treatment is indicated. Among the most useful sedative remedies we may mention an intra-laryngeal spray of cocaine (2 per cent solution), an inhalation of 10 drops of oil of peppermint, or of a 20 per cent alcoholic solution of menthol in an orinal nasal respirator, and the use of 1 drachm of glycerine of carbolic acid, with 10 drops of chloroform added to half a pint of boiling water in a steam-inhaler. Creasote or carbolic acid, diluted with rectified spirits, or spirits of chloroform, may also be used for inhalation in an orinal nasal respirator. In similar affections of the trachea and large bronchial tubes the same treatment may be applied; but the cocaine spray can only reach the upper part of the trachea at farthest.

Where the cough depends on bronchitis of the smaller tubes the treatment is that of ordinary bronchitis; an alkaline mixture containing bicarbonate of soda, or citrate of potash, with a few minimis of ipecacuanha wine, may be prescribed; and, if expectoration be difficult, 3 or 4 grains of carbonate of ammonia may be added. In some cases 5 ml of antimonial wine may be substituted for ipecacuanha for a few days with excellent effect. Similar drugs may be prescribed in an effervesceing mixture. Iodide of potassium in small doses, squill, and senega may also be given when secretion is tough and difficult of removal. Warm drinks, like tea, cocoa, or milk, or a steam-kettle to moisten the air, may often be used successfully for the same purpose, and may be tried before resorting to expectorant remedies. But in many cases all the above-named measures fail to give more than temporary relief, and sedative drugs are required. A linctus containing tincture of belladonna, spirits of chloroform, and glycerine may sometimes prove useful; but in the worst cases opium in some shape is indispensable. A combination of morphia and hydrocyanic acid with glycerine, spirits of chloroform, or syrup of wild cherry, forms an effective linctus, which, however, must not be used too freely lest the digestion be deranged.

Codeia may be substituted for opium, as it interferes less with the appetite; but its sedative effects are not equal to those of morphia.

In some cases of early disease, associated with a troublesome cough, much relief may be obtained from a small blister applied to the subclavicular region on the affected side.

Expectoration.—When the expectoration is very profuse, the administration of purified creasote or guaiacol in capsules is sometimes effectual in diminishing the excessive secretion of the bronchi and
pulmonary cavities. Turpentine and various resinous drugs are sometimes given for the same purpose; but the expectoration is symptomatic of broncho-pulmonary disease, and rarely requires direct treatment.

Dyspnœa, though seldom a prominent feature, is occasionally very distressing. When due to acute miliary tuberculosis and rapidly advancing pulmonary disease it admits of little relief, and we must be content to administer stimulants; such as ammonia, ether, and brandy.

Attacks of dyspnœa, depending on violent and ineffectual attempts to remove tenacious secretion from the bronchial tubes, may be mitigated by the judicious use of expectorants, the best being ammonia and senega. Steam inhalations of carbolic acid may render good service. In some cases dyspnœa has been lessened by inhalations of iodide of ethyl, as suggested by Dr. R. E. Thompson. Dyspnœa arising from cardiac failure, with attendant œdema of the lung, must be treated by diffusible stimulants and hypodermic injections of strychnine. The dyspnœa of pneumothorax will be referred to presently.

Hæmoptysis.—The pulmonary hæmoptysis of tuberculous disease may be due to capillary hæmorrhage, ulceration of vessels, or aneurysm of the pulmonary artery. The loss of blood in the first case is never extensive, and direct treatment is not required. In the second and third cases hæmorrhage is the result of gross lesions of comparatively large vessels, consequently the amount of blood lost may be considerable; yet even under such circumstances spontaneous cure is not infrequently effected by the formation of a thrombus, which seals up the ruptured vessel.

In our treatment of hæmoptysis we endeavour to imitate nature's method, that is, to promote thrombosis by lowering the pressure in the pulmonary artery.

It is important to recognise that the faintness which often attends the attack is a symptom of cardiac depression—a condition in itself favourable to thrombosis. In all cases, whether the hæmorrhage be profuse or slight, absolute rest must be insisted upon. The patient must keep in bed in a cool airy room, and should maintain a semi-recumbent position. Talking, movement, or excitement of any kind must be avoided. Nothing more than iced milk, meat jelly, and small sandwiches of bread and butter should be given for the first few days, and alcohol in any form must be expressly forbidden. The cough, which is rarely absent, may be relieved by sucking ice; but when it cannot thus be checked some preparation of opium must be administered. Small and frequent doses of morphia may be given by the mouth, or one-third of a grain may be injected under the skin. This remedy not only exerts a valuable local effect on the injured vessel by the rest which it gives to the lung, but it helps also to allay the restlessness and agitation of the patient. At the same time, seeing that in most fatal cases of hæmoptysis death occurs from suffocation rather than from the amount of blood lost, it is clear that the indiscriminate use of morphia is not without its dangers.
When from flooding of the bronchial tubes with blood dyspnœa is very pronounced, cough should not be checked by sedative drugs. With the view of reducing the blood-pressure, sulphate of soda or magnesia, in doses of 60 grains, should be given every three or four hours to begin with. Saline purgatives cause determination of blood to the intestine, and thus relieve vascular tension, but, unfortunately, their action is rather slow. Aconite has been recommended by Dr. Andrew, on the strength of experiments by Dr. Bradford and Mr. Dean which show that it causes a fall of pressure both in the carotid and pulmonary arteries. This method seems worthy of trial, but I have not had sufficient experience of it to express an opinion as to its merits. Astringents, like gallic acid and lead, are still extensively used, but it is difficult to see what effect they can have on aneurysms or ulcerated vessels. Ergot is perhaps the most popular drug at present, but, according to Bradford and Dean, it causes a rise of blood-pressure not only in the aortic but also in the pulmonary circuit; a result which must aggravate rather than check hemorrhage from the lungs. Clinical experience shows that the effect of ergot is as uncertain and unsatisfactory as those of gallic acid and lead.

The constipation which is produced by the last two remedies must tend, moreover, to raise blood-pressure, which is injurious. Oil of turpentine in large doses sometimes does good service, probably in virtue of the cardiac depression which it causes. Nauseating doses of ippecacuanha, recommended by Trousseau, seem to act in the same way; but the risk of vomiting is a serious one, and the remedy is now seldom employed. The application of ice to the chest appears to be of very doubtful utility, and in this country is little used.

The artificial induction of pneumothorax to cause collapse of the lung and pressure on the ruptured vessel was unsuccessfully employed by Dr. Cayley in one case of persistent hemorrhage. Under similar circumstances it would be worth while, in conjunction with other measures, to try Prof. A. E. Wright's plan of administering chloride of calcium in 15-grain doses three times a day, for a few days, to increase the coagulability of the blood. In any case great care is needed in the management of the patient after the hemorrhage has ceased. The diet should be very sparing, and the patient should not rise from bed for three or four days at least. Free action of the bowels should be secured by the continued use of saline laxatives. Alcohol should be avoided altogether for some weeks.

Pleurisy.—For the relief of the pain of dry pleurisy Dr. F. T. Roberts's plan of strapping the affected side gives excellent results, and can be strongly recommended. But in cases where one lung is extensively diseased, and pleurisy attacks the opposite side, it may not be possible to apply strapping without dangerously curtailing the already restricted respiratory surface. Under such circumstances we must be content with counter-irritation, a few leeches, and poultices; if these fail, a hypodermic injection of morphia will be required.

Roberts's method is specially adapted for the treatment of pleurisy
affecting the lower part of the chest, where the ribs are more yielding and their movement easily restrained. Pleurisy in the region of the upper three or four interspaces seldom causes such acute pain, as the range of movement of the upper ribs is limited; here counter-irritation is generally sufficient. In cases of sero-fibrinous or hemorrhagic effusion paracentesis should not be resorted to unless the quantity of fluid be so great as to cause embarrassment of the respiration. Experience shows that the pressure resulting from pleuritic effusion promotes arrest of the tuberculous process in the corresponding lung. As a rule the fluid is slowly absorbed.

In the comparatively few instances of advanced phthisis in which empyema occurs, the pleura should not be opened unless the abscess point externally, or unless the effusion be so large as to constitute a mechanical hindrance to respiration. In the latter case aspiration is preferable to free incision. The empyema once opened will rarely close again; and free incision appears rather to hasten the patient's end. In cases of early or limited pulmonary disease empyema must be treated on ordinary lines. It occasionally happens that the pus spontaneously becomes inspissated, and undergoes a caseous change.

Pneumothorax.—When pneumothorax arises acutely, with severe dyspnea and symptoms of shock, stimulants in the form of brandy, ether, or ammonia should be given at once. Morphia has been recommended by some writers to minimise the effects of shock, but in the presence of marked dyspnea an opiate is contra-indicated. Where the opening is valvular, and air accumulates in the pleural cavity under great pressure, paracentesis may be necessary in order to withdraw a sufficient quantity of air to relieve the pleural tension. Sir R. Douglas Powell advises that the side be afterwards strapped to prevent reaccumulation of air. Paracentesis is sometimes followed by subcutaneous emphysema. In the event of sero-fibrinous effusion, tapping may be successfully employed, but seeing that spontaneous recovery may ensue, it is well to wait for a time before resorting to this measure. In pyopneumothorax incision is generally considered to be unadvisable, and this, no doubt, is true of advanced cases. But the practice of early drainage is worthy of further trial, where the general condition of the patient is fairly satisfactory and the lung is presumably not much affected.

Laryngeal tuberculosis.—The treatment of this complication may be general and local. The general treatment is practically that of pulmonary tuberculosis, with certain reservations as to climate. The best atmospheric conditions are a temperate climate, a moderate degree of moisture in the air, and an absence of dust. At the same time, it may be admitted that many cases do well in such a dust-laden and apparently undesirable atmosphere as that of London and other large towns. Tobacco-smoking requires a brief notice. Many patients have no desire to smoke, but some have a craving for tobacco: if so, the patient may be allowed to smoke once or twice a day after meals, provided it be out of doors or in a large, well-ventilated room; the object
of these restrictions being to prevent the inhalation of smoky air into the larynx and lungs. The practice of inhaling tobacco smoke should be forbidden. Strong alcoholic drinks, spices, and highly seasoned dishes irritate the pharynx and epiglottis, and are to be avoided. The local treatment is fully described in the chapter on “Tubercle of the Larynx,” vol. iv. p. 800.

I may say here that to palliate the laryngeal irritation we may prescribe steam inhalations containing carbolic acid and chloroform; or at other times menthol or oil of peppermint on a respirator.

To soothe the pain so often present a 2 per cent spray of cocaine may be used a few minutes before meals. The local application of menthol, in the form of a 10 to 20 per cent solution in olive oil, sometimes gives relief; or, again, the insufflation of one-sixth of a grain of morphia with a little starch powder or sugar of milk. Some patients find benefit from sucking ice. When, in spite of these measures, the patient is unable to swallow, Dr. Wolfenden’s plan may prove successful; the patient is directed to lie on his face, with his head over the edge of the bed, and to drink through an india-rubber tube. It may ultimately be necessary to have recourse to nasal feeding, which, at times, does excellent service. The operation of tracheotomy is very rarely required, the only indication being afforded by the existence of severe laryngeal stenosis and impending asphyxia. The treatment of tracheal tuberculosis can only be palliative.

Tuberculous ulceration of the pharynx, palate, and tongue must be dealt with in the same way, and with the same reservations as in the case of the larynx. Good results sometimes follow the use of lactic acid when the ulceration is localised and the subjacent infiltration is not very great. Granular pharyngitis and other non-tuberculous affections of the pharynx, which may give rise to troublesome cough and other symptoms, must be treated on the principles laid down in the article “Pharynx” (vol. iv. p. 729).

Gastro-intestinal symptoms.—Loss of appetite, cardialgia, and other symptoms of dyspepsia may be treated by alkaline and acid tonics; but for general use nothing can excel an alkaline mixture consisting of bicarbonate of soda (15 grains), tincture of nux vomica (10 minims), and compound infusion of gentian (1 ounce), given before meals. If a sedative action be desired, dilute hydrocyanic acid may be substituted for nux vomica. The good effects of this mixture are witnessed not only by increase of appetite and relief of the dyspeptic symptoms, but at the same time expectoration is facilitated, whereby the cough is indirectly relieved.

In other cases, especially where flatulence is a prominent symptom, better results are obtained by acids, with or without strychnine, given after meals. In cases marked by irritative symptoms—such as vomiting or pronounced epigastric pain and anorexia—a mixture containing bismuth, hydrocyanic acid, tincture of belladonna, or, if necessary, a few minims of liquor morphinae, given before meals, is to be preferred. Bismuth in
powder, or in an effervescent draught with hydrocyanic acid, may prove more successful in particular cases.

In all instances of dyspepsia the diet requires a careful survey. The diet should be light and digestible, and the meals small and more frequent than in health.” In the comparatively uncommon form, distinguishable by a red glazed tongue, vomiting and anorexia, liquid food, especially milk with lime-water or soda-water, koumiss, veal or chicken broth, will be required; and complete rest in bed should be enjoined.

Inasmuch as this kind of gastric disorder mostly affects patients suffering from pyrexia and other symptoms of progressive disease, the outlook is very grave unless the gastritis can be speedily removed so far as to enable the patient to digest an adequate supply of food. If the symptoms resist the measures indicated, it may be necessary to rely exclusively on peptonised nutrient enemas for a few days, giving only a little iced water by the mouth, for the relief of thirst. Fortunately this form of gastric disturbance is not of very frequent occurrence.

In most cases of obstinate dyspepsia mild purgatives are called for, such as a small dose of calomel (half a grain to a grain at bed-time), followed by a teaspoonful of Carlsbad salts, dissolved in half a pint of warm water, in the early morning. Violent purgatives should be carefully avoided altogether, owing to the risk of setting up intractable diarrhoea.

A tumblerful of hot water, sipped at bedtime for a few nights, often gives great relief by washing out the stomach and removing remnants of undigested food which are apt to undergo decomposition, and thus to aggravate the catarrhal condition of the stomach. Dilatation of the stomach occurs occasionally, and washing out may be required; though the cases in which this operation can be recommended are very few, as the disturbance caused by the passage of the stomach-tube in feeble patients may be attended with serious consequences. Gastric digestion may be assisted by the administration of pepsin or papain, but, except as a temporary expedient, little benefit is to be expected from this line of treatment.

Diarrhoea is a symptom that should never be neglected; it should be treated by rest in bed and the application of warmth to the abdomen and extremities. In many instances it depends on slight errors of diet; and in such cases regulation of the diet, and a mild purge to free the intestine from irritating substances, may be all that is required in the way of treatment. For this purpose we may prescribe 2 drachms to half an ounce of castor oil with 10 minims of laudanum, or a small dose of calomel. If the diarrhoea do not speedily yield, bismuth should be given, in 20-grain doses, with a few minims of laudanum. In the far more serious case where diarrhoea is the result of tuberculous ulceration or lardaceous disease, powerful astringents, combined with opium, are indispensable. A mixture containing aromatic sulphuric acid, tincture of opium, and decoction of logwood, or again of subnitrate of bismuth, tincture of catechu, and tincture of opium, will often suffice to keep the diarrhoea in check. But in the most severe cases we must have recourse to stronger
remedies, the best, perhaps, being a pill containing sulphate of copper (¼ grain) and opium (½ grain), given once, twice, or three times a day, as may be required. In cases where the ulceration affects the lower end of the colon the enema opii (B.P.) gives more relief than anything else. It will generally be necessary to revise the diet carefully; the most suitable food in the acute cases being milk, koumiss, or carefully prepared beef-tea; but, when the diarrhoea lasts for weeks, boiled fish and tender meat, freed from fat and pounded or finely minced, may be given in small quantities. Digestion may be aided by peptonisation of the milk, and by the use of pepsine or papain after meals. The slightly bitter taste of peptonised milk may be masked by the addition of a teaspoonful of rum, cognac, or liqueur.

If the diarrhoea be accompanied by much pain, hot fomentations should be applied to the abdomen, and the warmth of the extremities sedulously maintained. In the rare event of serious intestinal haemorrhage, an enema, consisting of a teaspoonful of oil of turpentine suspended in two ounces of starch mucilage, should be administered at once. Acute peritonitis must be treated on general principles by opium and hot fomentations. In the yet rarer instances in which perforation can be diagnosed the propriety of surgical interference must be considered; but the patient’s general condition and the extent of the pulmonary disease may not justify such measures.

In chronic tuberculous peritonitis with effusion, whether serous or purulent, incision has, in several instances, been followed by arrest or cure. A similar result may also ensue without any surgical measures; in these cases, no doubt, the effusion is serous. Some physicians believe that the application of mercurial ointment to the abdominal wall promotes absorption of the fluid.

When suppuration has occurred, incision should not be delayed. Drugs seem to be of little use in this affection.

Renal symptoms. Albuminuria, whether due to nephritis or amyloid disease, is mostly found in advanced chronic cases. In such circumstances active treatment, by rigorous milk diet and purgation, is quite out of place. If possible, milk should be taken freely; but it is not advisable to prohibit a certain amount of meat and fish if the patient can take them. The drug treatment may include digitalis and iron, mild saline diuretics, and an occasional small dose of blue pill. But treatment should be addressed primarily to the general condition rather than to the renal disease, which is a local consequence of the constitutional malady.

In the few instances where nephritis occurs at an early period of the pulmonary disease, and where the health is not seriously affected, treatment may be conducted on ordinary principles.

The tuberculous pyelitis and cystitis of advanced cases do not admit of more than palliative measures. If the lung disease be slight, surgical advice should be sought.

Nervous symptoms.—For the treatment of meningitis, tuberculous
tumours of the brain, and nervous complications in general, reference must be made to the proper articles.

It seems desirable, in conclusion, to sum up the general plan of treatment suitable for an ordinary case. In the first place, attention must be carefully directed to the rules of general hygiene; to the importance of spending as much time as possible in the open air, and the necessity of an abundant supply of food. Excepting in the most favourable instances, where the disease is quiescent, some form of tonic medicine will be required from time to time, the best being an alkaline bitter mixture, such as that already indicated. Narcotic and sedative drugs generally should be employed with great caution, because of their prejudicial influence on digestion; and complications, as they arise, must be treated on general principles.

Percy Kidd.

REFERENCES


P. K.

PNEUMOCONIOSIS

Pneumoconiosis, pneumonoconiosis, or, translated into English, "Dusty-lung-disease," is a lesion that has attracted but little attention in this country—a circumstance the more remarkable considering that Great Britain has long held the first rank in manufacture, and that a large proportion of its population is consequently engaged in dusty occupations. At the same time, credit is due to British physicians for the early recognition of inhaled dust as a cause of lung lesion; among them may
be mentioned Christison, Addison, Bennett, Corrigan, and Peacock, who taught that inhaled dust can penetrate the lung tissue, and that its presence can be demonstrated therein. This doctrine, however, had for a long time numerous adversaries, who argued that the black granules so frequently met with were derived from the carbonaceous materials of the blood; and it is only within the last half-century that this opinion has been given up. That it held sway so long was owing to the influence of the eminent pathologist Virchow. Without denying that a black colour can be derived from the blood, it must be admitted, nevertheless, that the pigmentary particles generally found in the lungs, and especially in the lungs of persons engaged in dusty occupations, are derived from inhalation of dust.

The opposite opinion derived great support from the notion long held that the orinasal passages are so perfect and efficient as dust-strainers that, in co-operation with the cilia lining the bronchi, no dust could reach the deeper lung tissue. This opinion has been disproved both by experiment and by observation, and is no longer tenable. Further, although it is true that this conservative apparatus, so efficient in itself, is greatly re-enforced by the strong expiratory act of the lung, yet it will fail to arrest the ingress of particles if dust exist in large quantity or is breathed almost without intermission; or again, if the mucous membrane have suffered damage such as to facilitate its entrance into the submucous tissue. Under such circumstances the foreign matter enters the extravascular lymph-current and lymphatics, pursues its course along the pulmonary interspaces and connective tissue, and eventually reaches the alveoli and bronchial glands. The last-named organs act as barriers against its farther progress, and in consequence they become deeply coloured and swollen, and occasionally suffer ulterior changes.

Consolidation by the excessive growth of fibrous tissue is the chief pathological feature of pneumoconiosis. The pulmonary is transformed into fibrous tissue; the extent of change being dependent chiefly upon the physical character of the dust inhaled, but in some degree also upon accidental conditions of employment. The fibrotic change is almost always associated with thickened pleura, and the degree of this change bears some relation to the extent of fibrinous production in the lung substance itself. Now and then the fibrotic change seems to start from the pleura, and to spread in a branching, vein-like fashion; or in bands across and through the lung. The ramifying lines of fibrous growth for the most part represent interlobular or interstitial spaces, and are white in colour; this, however, is no essential character, for not infrequently

1 It is very gratifying to be able to refer to so admirable a collection of specimens of Pneumoconiosis as that in the museum of the Middlesex Hospital,—the result of long-continued pathological research made by the late Dr. Headlam Greenhow, physician to that hospital. Moreover, we have the benefit of his history of many of the cases that furnished those specimens recorded in the volumes of the Pathological Society of London. In no better way can the morbid anatomy of pneumoconiosis be studied than by an inspection of this collection; I have accordingly introduced the numbers affixed to some of the specimens in the proper places of this article.
the marble-like venation is black or brown, and its colour is largely dependent on that of the dust inhaled. The consolidation in question evidently has its origin in an exudation of lymph consequent upon very chronic inflammation of low intensity, due to the passage of dust into the bronchial tubes. A very similar consolidation, though rarely so extreme, follows the inhalation of tubercle bacilli; and as there is no little resemblance in many particulars between pneumoconiosis and tuberculous disease of the lung, there has been great confusion between the two—particularly on the part of laymen, who have not inaptly called both the one and the other by the common name of consumption. At the same time, as it was observed also that consumption, in this wide sense, manifests itself pre-eminently in certain occupations, further distinctions were made between that of potters, of stone-workers, and of Sheffield grinders;—"grinders' rot," "miners' rot," and so forth.

The fibrosis in different cases varies greatly in extent, in density, and in the degree to which the bronchial tubes and pleura are implicated. The condensation in some instances does little more than destroy the sense of crepitation under the fingers; whilst in others the pulmonary tissue, losing its sponginess, is transformed into a dense mass which, in the most advanced specimens, shows no traces of normal structure, and in hardness, and often also in colour, resembles india-rubber. On section, moreover, this dense mass betrays at times an appreciable amount of grittiness, particularly if the dust inhaled be of a siliceous or metallic quality. The resemblance between specimens of "pneumoconiosis and of tuberculous disease—especially where the latter has been very chronic—is often so great that, to the eye, the two may be almost indistinguishable, and the true nature of the disease a matter of doubt. In the eyes of many persons the two diseases are inseparable, and the opinion is held that the dust-made disease does not exist apart from tubercle. That pneumoconiosis, however, does exist apart from tubercle is the conclusion of a large number of independent observers. The determination of this problem in particular cases will depend, therefore, upon the discovery of the specific bacillus.

Sir Andrew Clark uses the name "phthisis" in a wide sense, and recognises two forms of it: (i.) the tuberculous, and (ii.) the fibroid, the former being characterised by the tubercle bacilli. Dr. Thomas Harris of Manchester, in his able lectures on phthisis, makes a parallel division of fibrotic pulmonary consolidation—under the appellation of interstitial pneumonia—into "primary" and "secondary." The former is represented by Corrigan's pneumonia, which makes its appearance without evident cause; the latter by pneumoconiosis, with an obvious cause in the inhalation of dust.

The grounds for the distinction made by both these authors are well set forth, by the former on clinical, by the latter on pathological data; both, however, agree that the non-tuberculous variety is of comparatively rare occurrence. As pneumoconiosis is primarily local, and without constitutional complications, all other viscera, except the lung affected,
PNEUMOCONIOSIS

may remain normal and carry on their several functions, until indeed the long-persistent local derangement brings about secondary disturbance in one or more functionally connected organs. All such secondary disorders are of late appearance among the phenomena of pneumoconiosis; they may even extend to heart disease, with a certain amount of general dropsy, gastric and hepatic troubles, and secondary albuminuria. This being the case, the sufferer with pneumoconiosis is enabled to undergo considerable exertion for a long time, and is apt to look upon his earlier symptoms as no very important affair.

Although linked by its inflammatory characters to pneumonia, pneumoconiosis is not an example of croupous inflammation; it is rather a bilateral peribronchitis. Not infrequently it sets up a nervous disorder indicated by spasmodic breathing; hence it is also often called asthma, a name further distinguished by a noun indicative of the employment to which it is due. It is, moreover, a non-febrile malady; though there may be an intercurrence of active disease, the product of chill, or of supervening tuberculous deposit, with consequent elevation of temperature. In either case hectic symptoms may appear, local softening, and now and again a patch of ulcerative gangrenous decay. Pneumoconiosis is not a disease of children, but of adults, and these for obvious reasons are almost always of the male sex. The form of fibrosis which occurs in children after attacks of measles and whooping-cough is quite different from pneumoconiosis. Pleuritic thickening, as before said, is commonly met with; nevertheless it cannot be esteemed a necessary concomitant. The like is true also with regard to chest deformity. This last incident owns as contributory causes pleuritic adhesions and the shrinking of the lung itself as a result of progressive contraction of the fibrotic tissue diffused through its substance.

When once fibrosis has invaded the pulmonary substance, its tendency is to advance; chiefly because of repeatedly recurring bronchial attacks, due to fresh bronchial colds and the continued introduction of dust, intensifying the inflammatory action. For the most part both lungs become affected, though in varying degree. This would seem to be a necessary consequence of the entrance of the dust by the common channel of the larynx; nevertheless, some further determining cause operates to vary its diffusion, and to account for its predilection for the posterior and middle portions of the lungs. The chronic interstitial pneumonia of Clark and others is a unilateral disease generally due to a dry pleurisy.

Symptoms.—The disease is an extremely chronic one, and, beginning as a non-febrile bronchitis, it attracts little attention until an area of the breathing tissues of considerable extent is more or less disabled. The augmented bronchial secretion is at first noticed chiefly on waking, or on passing from a warm workshop into the open air. It is nothing more than ordinary mucus, with minute particles diffused throughout it, numerous enough in many instances, when the dust is of a dark hue, to give it a black colour; it is glairy, and is coughed up with some effort. As yet the affected workman does not suffer in his general health. He
eats, drinks, and sleeps well, and joins in active physical exertion. But the conditions of employment, involving continuous exposure to dust inhalation, cause recurrent bronchial attacks, each in its turn damaging the lung more and more. Months and years may pass with but a slow increase of cough and spitting, though with an amount of dyspnea exceeding that which is met with in ordinary bronchitis, and out of proportion to the severity of the evident organic changes. Constitutional symptoms are, however, still absent, and as appetite and digestion are good there is no wasting.

Continuance in the dusty occupation is soon attended by more and more copious expectoration, which gradually acquires a yellowish gray tint and the features of muco-pus. At length, however, the time arrives when impeded respiration, oft-repeated cough, loss of rest and appetite, and the discharge of muco-purulent fluid tell injuriously upon the general health and strength. The sufferer loses ground in all directions, he cannot pursue his work as heretofore, nor take outdoor exercise; he cannot even lie comfortably in the horizontal position. He seeks hot rooms and quietude, and becomes a valetudinarian, calling for medical treatment to relieve his cough and yet more to relieve his breathing. And now it is that loss of flesh and colour becomes apparent, whilst urgent and now constant dyspnea confines him to his house or to his chamber. At this stage of the malady the name of consumption is applicable enough; though, as will presently be seen, sufficiently distinctive signs between the two maladies are discernible.

The great dyspnea suggests the existence of pulmonary emphysema (1271), but the known pathology of fibrosis indicates that where emphysema exists, it is vicarious and comparatively insignificant. The dyspnea is attributable to abridged respiratory area, to the choking of bronchial tubes by secretions, and, it may be, to cardiac mischief; and, as before stated, certain dusts, by the possession of special properties over and above the strictly mechanical, still further aggravate the difficulty of breathing, and assimilate it to the asthma of emphysema: an example of such properties is found in flax-dust. Moreover, the asthmatic state is not conjoined with the barrel-shaped thorax of emphysema: on the contrary, the lungs shrink as the diffused fibrin progressively contracts, and the bronchi, as the tissues are compressed and solidified, are distorted, dilated, and thickened (1274, 1279). The movements of the chest walls are crippled by the fibrous bands which pass between the costal and pleural surfaces. By other adhesions the lobes of the lungs also are distorted, drawn together, and compressed; and as a result their freedom of action and air capacity are seriously curtailed. All these conditions necessarily add to the difficulty of respiration (1274). If pneumoconiosis be unilateral, the fellow lung expands, and indeed may become truly emphysematous.

Pathology.—That a particle of dust, when it comes in contact with mucous membrane, will cause great irritation is a fact of everyday observation in the case of the conjunctiva. The irritation is immediate,
and so severe as speedily to produce injection of the blood-vessels, and
an outpour of tears from the lacrimal glands; if the disturbing cause be
not soon removed, the phenomena of inflammation will set in with sero-
purulent discharge; thickening of the conjunctival membranes, and
effusion of lymph as a fibroid film upon the transparent cornea beneath.
All these phenomena fall within the range of unaided vision; and
we are fully justified in concluding that something of the sort occurs
when the mucous membrane of the respiratory passages is directly
irritated, modified, as it may be in some details, by the special histological
qualities of lung tissue.

The degree of irritation set up, and its consequences, will vary accord-
ing to the physical, chemical, and physiological properties of the offending
agent, the quantity introduced, the frequency of its introduction, and the
period during which its action continues. For instance, there are dusts
which are escharotic and damage at once the structures they fall upon.
This is true of dusts both of mineral and organic nature. There are
other dusts which damage by their chemical properties; and, again, others
of animal origin which may inoculate or infect the system. Lastly,
the dust of poisons may enter the body and display their respective
effects, not only locally, but on the whole organism also.

The dust that has entered within lung tissue can be detected by the
microscope; and may often be seen, under appropriate tests, to preserve
both its physical and chemical properties. For instance, particles derived
from coniferous wood have been identified by their gland-bearing fibres,
and siliceous particles by their translucent appearance and their resistance
to acids other than hydrofluoric. But the particles of dust are for the
most part amorphous, and diffused sparsely in the tissue invaded by
them (1272); or they are arranged in linear fashion, or collected in small
masses, or in vein-like form marking out the lobules and alveoli within
the interior of some of which they may be seen (1279).

The permeating dust is usually of a black colour, though where it
possesses a distinctive hue of its own this peculiar tint pervades the
altered tissue. (In specimen 1276 the lung is stained with carmine.)
A blue black is frequently seen (1271), and a yellowish or buff hue is
not uncommon; deposit of the latter colour is mostly seen in the lungs
of quarrymen, or sometimes in accumulated matter suggestive of caseous
transformation (1278). Yet even where the dust inhaled is itself of a
pale colour, the parenchyma infested by it is more or less black or slate
coloured; frequently this deep colour is not wholly derived from with-
out, but owes its origin to some material collected within the living
tissue; it is most likely a derivative from the blood.

The very clogged portions of black lung formerly met with in coal-
getters were proved by Christian and others to be due to a great
accumulation of coal-dust, the nature of which was demonstrable by
combustion.

The lung of a young child, until about eight years old, has a clear
pink colour; but about this age black pigmentary spots or lines appear
in lobar and lobular spaces, mapping out the surface in irregular areas, and producing thereby a marbled appearance. As age advances, these dots and lines multiply and enlarge, and mostly in adult life produce a generally diffused dusky colour.

In persons engaged in a dusty occupation this coloration becomes progressively more pronounced; its tint varies according to that of the inhaled dusts, but a black colour largely preponderates. In the instance of workmen exposed to the dust of oxide of iron a reddish colour prevails.

The fine impalpable dust most people breathe is not the cause of the true pneumoconiosis we are concerned with. It blackens the lungs more or less, but these organs appear to be very tolerant of it; nevertheless, it may be that a lung deeply charged with black pigment is less efficient and less able to withstand inflammatory or other disease. When, however, a directly irritant dust is abundantly inhaled during some industrial process, an active morbid process is the result, one ending in structural lesions.

The source, form, and physical qualities of inhaled dust particles have furnished the basis for the classification of ensuing morbid consequences to lung tissue. Thus authors have described the results of inhaling siliceous particles as chaliosis or silicosis; of metallic particles as siderosis; of carbonaceous particles as anthracosis; of cotton particles asbyssinosis. Other uncouth words have been suggested for different morbid varieties of the same general kind. To multiply such words is undesirable; whatever the kind of dust, the consequences of its presence are, pathologically speaking, substantially the same.

Besides the form we have to recognise other differences of inhaled particles, as, for instance, the density and the chemical qualities which affect their pathological influence. This last, in like manner, is modified by the solubility, miscibility with fluids, and cohesiveness of the dust. A very soluble dust will be got rid of by speedy absorption; a miscible one, like flour and other amylaceous substances, will collect in tenacious masses and obstruct the bronchial tubes; the fine dust of hydrate of lime, unless highly caustic and breathed freely, seems more or less to be disposed of in the parenchyma by absorption and the influences of secretions upon it; whilst that of lime salts—for instance, the carbonate in the shape of chalk, and the sulphate in the form of alabaster in fine powder—may be breathed with impunity for long periods of time.

In numerous cases mixed dusts are encountered; as, for example, in the Sheffield and needle-making trades, where siliceous and metallic dust is intermingled in various proportions. In such instances this circumstance of admixture is presumably attended by some variations in the consequent symptoms. Yet even in this matter the form and dimensions of the scattered particles play a more important part than chemical constitution.

Again, speaking generally, the ill results of dust are more serious the greater its departure from organic tissue in character.
When dust which has entered the respiratory system has reached the alveoli it attaches itself in the first place to the walls, but sooner or later penetrates them. The particles are borne along by mucous cells, which, being destitute of walls, envelop them after the fashion of amoebae; but the foreign bodies soon prove a source of irritation, which in the case of the more irritant kind advances to inflammation of low intensity, but sufficient to induce an outpour of some lymph and the generation of granular matter and exudation cells. The first effects of the foreign matter are the detachment of the normal epithelial cells of the alveoli and an attendant thickening. The accession of the new material within the alveolar cells causes their distension and functional derangement, and presently, by the action of the exuded fibrin, their obliteration. When a group of such altered cells is formed, the next phenomenon is the production of a piece of more or less solidified lung tissue, manifested in the form of a granule. The multiplication and cohesion of such granules, and the gradual condensation of the fibrin, transform the piece of lung into the fibroid texture we know as pneumoconiosis; whilst the transformation is proceeding the blood-vessels become thickened, blocked, and impervious, and the compressed lymphatics are merged in the morbid growth.

In their general features these structural changes, though chiefly peribronchial, resemble the early stage of croupous pneumonia; but their development is so slow and inactive that no marked fever attends them. However, they possess the faculty of extension; the mucous and inflammatory cells make their way along the adjoining connective tissue, and fibrosis continues to spread in the form of fibrous bands, veins, or streaks.

On section of the condensed portions of fibrotic lung the surface commonly exhibits numerous raised points which give it a coarsely granulated appearance. Most of these points, if closely examined, will be found to be small bronchial tubes, cut across, and thickened and obstructed by secretions and, frequently, by a yellowish matter, suggestive of caseation, which imparts a speckled aspect to the specimen (1254, 1255, 1256). The occlusion in its turn embarrasses the lung yet more, and retrograde structural changes are precipitated.

The morbid phenomena just described are those of dust of distinctly irritating qualities; but when the dust is of a more innocent kind, they are considerably modified. For example, where the dust is organic, but devoid of acrid qualities, as in the cases of the flour of wheat and other cereals, and of like mild substances, the primary irritation is of small account; the dust then operates chiefly as an obstructive agent, clogging the respiratory passages; if any inflammation be present it is very slow. Yet this clogging cannot go on without disabling the air-cells by pressure and otherwise. Soot and very fine charcoal, too, disturb the respiratory organs and their functions to a still less degree; and, though very widely diffused, and causing, it may be, much dyspnoea, they produce no well-marked signs or symptoms of inflammatory action and condensation.

The intimate pathological processes associated with anthracosis (coal-dust condensation) differ considerably from those dependent upon
the denser dusts of stone and metal, as is indicated by their morbid anatomy. Nevertheless, indurated lobes are met with, and pulmonary tissue may be so permeated by fine coal-dust as to be rendered friable, and to exude on section and pressure a black, inky fluid; moreover, the expectoration at the same time gets similarly coloured, and acquires the name of "black spit." In some such cases the production of fibrous tissue gives place to a process of disintegration; the pervading dust being, we may suppose, so considerable in quantity as to destroy the vascular supply and lead to a sort of necrosis (1274). In other words, the dusty irritant fails to arouse active inflammation and fibrinous exudation. Examples of the extreme forms of the disease are at the present time unknown, or almost unknown, in English mining districts, thanks to the improved ventilation of mines and the operation of the Mining Acts which limit the age of children admitted to pitwork; for the same reasons the inky expectoration, known in past days as "black spit," is correspondingly rare. It is generally true, indeed, of all dusty occupations, that indurated impervious lungs are becoming rare as sanitary construction is improved, as appliances are used in factories to convey away and disperse the dust, and as sanitary laws and observances are better observed.

The pneumoconiosis due to the coarser dusts which arise from the operations of stone-dressing and quarrying present some peculiarities attributable to the form and composition of the particles. The sawing and polishing of Aberdeen granite appear to be unattended by severe lung irritation; whilst, on the other hand, working with Edinburgh building-stone proves most injurious, and kills the workers by fibroid phthisis in relatively large numbers.

Another peculiarity of the dust of stone is that it tends to collect in masses, forming concretions (pneumoliths) which, by producing softening and ulceration around them, give rise to cavities with soft walls, though now and again lined by membrane. This state of things occasionally ends in the detachment and expulsion of the concretion, as it penetrates into a bronchus of sufficient calibre. Another feature in the lungs of stone-workers is that a yellowish or grayish hue replaces more or less the blackness seen in most other workers (1276, 1285, 1286).

Dust often reaches the pleura and lines its pulmonic surface, imparting a more or less black colour to it, and at other times collecting in nodules upon it. Its transference is effected by the lymphatics.

The ultimate goal of much of the dust inhaled is the bronchial glands, where it becomes imprisoned, causing enlargement and possible suppuration of those organs, which acquire a black colour, often of great depth.

Occasionally the enlarged and softening glands adhere to a contiguous organ, as, for example, to the cesophagus; this may be followed by ulceration through its coats, and the contents of the abscess thus discharged.

Not only do differences of dusts, in their origin, form, and composition, modify the form of the lung diseases which are set up, but they variously affect the health of the sufferers; and as the various conditions under
which labour is carried on have also their respective influences, it becomes evident that the symptoms and pathology of pneumoconiosis must exhibit a great variety. Intemperate habits in the patient, for example, precipitate the onset of the lesion, and accelerate its course.

Further, no doubt exists that persons of any age who inherit chest weakness, if they follow a dusty trade, are more liable to suffer from pneumoconiosis. One practical lesson is that such persons should not be thus employed. This inference points to the utility of certifying factory surgeons to pronounce upon the comparative fitness of all who engage in such work, especially of children and youths, and to the care they should exercise in following their occupation.

Again, the lungs of very young children, and of those born of parents with thoracic lesions and damaged constitution, suffer more readily from exposure to dusty occupations, and to heat, moisture, and confined or vitiated air, their too frequent accompaniments.

Associated lesions are found with the fibrotic change in the shape of emphysema, bronchiectasis, cell-collapse, and cavities; morbid events which are described and explained in other parts of this chapter: the first-named change is for the most part vicarious or complementary. A specimen of this vicarious emphysema is No. 1276. The emphysema is not confined to the circumference of solidified segments of lungs, but occurs elsewhere, particularly along the free anterior border; here the dilated cells are much larger, and the septa between them frequently broken through.

Bronchial dilatation or bronchiectasis [vide chapter on "Bronchiectasis" in this volume] is usually but not invariably associated with fibroid disease. As the bronchi are the first to suffer from the inhaled dust we might well expect them to be prominent in their lesions. A primary change, indeed, does occur in them in the shape of thickening of their walls, with loss of elasticity, due to the inflammatory action proceeding in and around them. Moreover, whilst losing elasticity they get plugged and distended by mucus and inflammatory products; and the growing fibrous tissue about them, in course of its contraction, drags on their walls, and causes irregularity of form and disturbance of position.

In this view of the causal relations of bronchiectasis we have the support of Dr. Coats of Glasgow and of Professor Hamilton of Aberdeen; but the former contends that both it and the formation of cavities are consequences and not causes of pneumoconiosis. The latter puts forward the following mechanical hypothesis: that "as the chest wall forms a comparatively fixed point to which the shrinking lung tissue also is attached by means of pleural adhesion, and as the tissue also is attached to the walls of the bronchi, the result of the shrinking will be that these two points will be approximated, the chest wall drawn in, and the bronchial wall drawn out. The latter, however, being the more yielding structure, will be more affected than the former. In this way we have the formation of cavities by bronchiectasis. Such cavities have for the most part well-defined walls, and are directly continuous with bronchial
tubes of which they are flask-like dilatations. It is to be remembered that the primary process, involving as it does the smaller bronchi, leaves all but these capable of dilatation."

This explanation may hold good in some instances, but cases are not uncommon where no connection by bands with the chest wall exists, and others where cavities occur in crepitant lung tissue.

Pursuing his criticism of Professor Hamilton's hypothesis, Dr. Coats affirms that in some cases cavities arise by accumulation of the secretions and inflammatory products behind an occlusion of a bronchial tube. He believes also "that cavities form by bronchial dilatation by a similar process to that which leads to emphysema without any primary disease of their wall. The dilatation, in fact, is complementary to the shrinking which has taken place in some part of the lung." Continuing these observations, he cites a case of "congenital non-inflation of the lung in which the bronchi had become converted into a series of sacs. Here the non-inflation without any active disease, implying as the chest enlarged an excessive distensible force acting on the bronchi, caused a general bronchiectasis. In a similar way in fibroid phthisis, we may have bronchiectasis and emphysema in an otherwise sound part of the lung, in consequence of shrinking in another part" (p. 124). These secondary changes are features so well marked in the later stages of pneumoconiosis as to call for our attention; but for further details as to the forms and other varieties of bronchiectasis the reader is referred to the special chapter on this disease (vide p. 53).

Cavities, not of bronchial origin, may perhaps arise in a portion of pulmonary tissue deprived of function and nutrient vascular supply; as, for instance, in a mass of condensed tissue, whether from fibrosis or from collapse. Again, the presence of a concretion may operate as a cause of softening of the tissue around it, when the cavity may contain a "pneumolith," or only pus and detritus should the stone have ulcerated into a bronchus and made its escape outward (1278, 1279). This phenomenon is most common in the case of coarse mineral dust as found among stone-workers. I have already remarked on the almost constant association of pleuritic thickening with pneumoconiosis, but expressed my dissent from the opinion of Sir A. Clark as to the direct causal relation between the two lesions. For, according to that opinion, fibroid lesion is the result of growth from without inwards. This, indeed, does take place in some instances, as seems to be illustrated by a case described by Clark; but when fibroid lung is due to dust inhalation, the development of fibrous tissue passes from within outwards, and fibrous bands do not extend from the pleura (1282). There seems good reason, moreover, for supposing that a primary inflammatory process is not universally necessary to the development of fibroid mischief; but that any condition which destroys or suspends pulmonary action for a long time, by obstructing access of air and blood-supply, will lead to lung collapse and infiltration, and eventually to fibroid degeneration. In a word, loss of function is a prelude to fibrous degeneration; and Rind-
fleisch surmises that obstruction of bronchi by accumulated cells and mucus will cause collapse of air-cells in the rear, and ultimately fibroid degeneration.

The notion that a constitutional proclivity lies at the root of pulmonary fibrosis receives some support from Clark's own statement; namely, that albuminuria is commonly associated with it. As regards the unilateral interstitial pneumonia this may be true, but as regards pneumoconiosis, in almost all the cases examined no albumin was found. Albuminuria is, of course, not unknown in such cases, but its occurrence is in no higher ratio than among an equal number of persons suffering from miscellaneous maladies. The concurrence of pneumoconiosis with pulmonary phthisis is not infrequent, but there is not pathological identity between the two maladies. Irritation may be a common starting-point in both; but, whereas in tuberculous lesion the apparent cause is the existence of tubercles and of bacilli, and the prevailing tendency is to soften, break down, or ulcerate, in fibroid disease, on the contrary, there is a chronic inflammation with fibrinous products which tend to contract and transform lung tissue into a hardened mass unfavourable to tuberculous extension and softening. For it is known that a piece of fibrous tissue in a softening tuberculous mass operates as an obstacle to the disintegrating process. Nevertheless, experience proves that the one lesion is often grafted upon the other and may replace it.

Diagnosis.—Dust-phthisis and pulmonary phthisis have been, and, indeed, still are very frequently confounded. The pulmonary fibrosis of metal-grinders, of stone-workers, of potters, miners, and some other artisans is popularly known as consumption or phthisis, and its victims are entered in death registers as dying of consumption. As already shown, the inflammatory phenomena of deposited tubercle and of fibrosis affecting the pulmonary organs diverge at an early stage; the thickened alveoli and their contents suffer a degenerative process, ending in the former case in softening and ulceration; whilst in the latter an abnormal development of fibrous tissue takes place rather prohibitive of ulceration, but directly productive of condensation and shrinking.

However, this differential feature must not be pushed too far; for, as already asserted, a tuberculous lung presents more or less condensation from excessive development of fibrous tissue, especially where life has been greatly prolonged, and opportunity given for the conservative agency of the fibrinous effusion to advance at a greater rate than that attained by the disintegrating action of tubercle (1302, 1310). A very important preliminary part of the diagnosis is to ascertain the presence or absence of tubercle in the history of the patient’s family, or of any organ of his own body. The nature of the work followed, and the conditions and circumstances of employment, will likewise, of course, be ascertained. A third point of importance is to get a correct account of the onset of the malady, its course and its duration. Pneumoconiosis is compatible for many months with capacity for physical labour and for adequate nutrition; tuberculous phthisis, on the other hand, within an equal period, will reduce
the patient to a state of considerable debility and emaciation. Tuberculous phthisis is rather a disease of youth and of early middle life, of constitutional nature, and without obvious causal connection with the breathing of dust; whereas pneumoconiosis is a local lesion of middle life, directly referable to dusty employment, and not associated with any marked constitutional bent. Moreover, it is not only most prevalent in middle life, but it is also almost peculiar to men. Nor is the previous history of dust-disease that of acute or febrile thoracic maladies, such as pneumonia or pleuro-pneumonia, but of smaller ailments, especially of winter coughs recurring year after year, and disappearing in the warmth of summer. Other divergent features are the rarity of laryngeal troubles, the comparative infrequency of diarrhoea, and the little pyrexia and sweating in the dust-produced lesion. Hæmorrhage in notable quantity is an infrequent incident in dust-disease, unless the lesion be provoked by sharp particles as of iron and steel. Green purulent sputa are also less common, though not unknown; for in the far-advanced stage they occur. The sputum for a long period is a whitish frothy mucus, which presently gets lumpy and grayish yellow, and for the most part is in smaller quantity, regard being had to the extent of lesion, than in tuberculous disease; speaking generally, the symptoms of dust-disease are more like those of chronic bronchitis, with which, in fact, it is usually confounded, than of phthisis. Fœtid bronchitis and gangrene are unusual, but not unknown results.

The temperature is less elevated than in phthisis, and hectic fever, if present, less pronounced. In the earlier stage emaciation proceeds and may for a time pass unobserved, for usually the appetite and digestion continue more or less good. Diarrhoea seldom appears.

Again, whilst anæmia and oedema are less prominent, dyspnœa is more so, and often paroxysmal. Dyspnœa is greatly aggravated when pulmonary embarrassment has long existed and secondary cardiac disease been established; and as in chronic bronchitis, the cough, expectoration, and hard breathing are increased on first rising, or on passing out into the cold outer air, and are abated by warm drinks and warm atmosphere.

Phthisis particularly affects the apices of the lung, whereas pneumoconiosis prevails rather in the posterior and inferior segments. Moreover it is distributed in separate patches, and gives rise to a greater or less number of areas of dulness on percussion. Dulness under the clavicles, when found, does not resemble that of tuberculous deposit or of pleurisy; it points rather to absence of respiration and bronchial mischief, and is less accompanied by moist crepitus and râles, or by the “cracked pot” sound of a cavity.

In those instances where pleurisy has played a prominent part the adhesions cause deformity of the chest wall and consequent displacement of the heart. The same event happens where the lung itself has become greatly shrunken or displaced by its fibrous transformation (1274).

Yet, notwithstanding differential features, tuberculous phthisis and pneumoconiosis are separable by a somewhat shadowy line (1283, 1264);
and if the presence, or absence of bacilli are to furnish the securest
foundation for making the distinction between the two, far more careful
and numerous researches are needed than have as yet been made.

The intermingling varieties of fibroid and tuberculous disease are well
exhibited by Sir A. Clark, who describes two main forms as properly
distinguishable, namely, the tuberculo-fibroid and fibro-tuberculous,—the
main difference between the two being the order of succession of tubercle
and fibroid; but a further examination of those problems would lead us
beyond the limits of a chapter on pneumoconiosis.

Of the many cases placed on record as pneumoconiosis by various
observers it remains doubtful whether all are true examples of the disease.
Some of them certainly are not, and unfortunately the opportunity for
studying the disease is to a great extent denied to the bulk of medical
men. Indeed, I apprehend from my study of his able treatise on
Fibroid Diseases of the Lung that its distinguished author lacked material
for a thorough exposition of the morbid consequences of dust-inhalation,
apart from tuberculous complication.

Non-tuberculous fibroid disease, indeed, whether due to dust or not, is
an uncommon lesion, and it is becoming more uncommon day by day by
reason of the advances of hygienic knowledge and of its increasing
practical application to those employments wherein dust is an almost
necessary accompaniment.

Prognosis and Treatment.—From the account given of pneumo-
coniosis it is evident that when once established a permanent lesion will
remain, which cannot be undone by medicinal treatment. Nevertheless,
it is capable of great alleviation; and, if not beyond a certain stage, its
symptoms will remain quiescent if the sufferer abandon his dusty occupa-
tion. Moreover, it scarcely need be said that whatever sanitary arrange-
ments can be provided, and whatever mechanical contrivances can be
invented to obviate the entrance of dust within the chest, so much less
severe and less frequent will be the disease.

Being a very chronic malady, it affords ample time and opportunities
for hygienic management, and for whatever medicinal treatment can be
suggested. The first indication, then, is to withdraw the sufferer from
his employment, or to diminish the production and diffusion of dust in
the work by mechanical and other devices to secure thorough ventilation
of shops. Respirators should be worn, the workmen themselves should
carry on the technical details of their calling so as to produce the least
amount of dust, and observe the general rules of temperance and health
in their way of living. On the part of masters it is an imperative duty
to provide healthy workshops with efficient ventilating apparatus and all
sanitary arrangements calculated to protect their work-people from the
 evils of the occupation they are engaged in.

Thus fibrotic patients may live many years, though they must be
accounted more or less invalid. At the same time it is to be remembered
that the dormant lung affection may be easily aroused into activity by
fresh exposure to dust, and become complicated or aggravated by bron-
chitis, broncho-pneumonia, and pleurisy, by depressing causes such as cold and wet, and by irregular and intemperate habits.

Prognosis becomes highly unfavourable when symptoms arise indicative of the development of tubercle in the already diseased viscus. This, unfortunately, happens not infrequently, and is less to be wondered at when we remember the prevalence of this hereditary disease, which abounds in manufacturing populations. The occurrence of hæmoptysis is thus of bad omen.

Of drugs I have found the iodide of potassium the most useful, sometimes, where additional alkali is needed, combined with bicarbonate or citrate of potash; or, where a spasmodic asthmatic state is present, with ether or the ethereal tincture of lobelia. Where great weakness exists, quinine is useful, and cod-liver oil may, when it can be borne, be administered with great advantage to prevent wasting. Terebinthinate inhalations, such as pinoil, facilitate expectoration and relieve cough, for which also vapor conii may be inhaled, or a liniment may be ordered containing a minute dose of morphia with some preparation of squills and tolu. But it is of the first importance to sustain nutrition, to encourage exercise outdoors, and to promote action of the chest muscles by regulated calisthenics. Further instructions in treatment will be found in the other chapters on pulmonary disease in this work.

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REFERENCES


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