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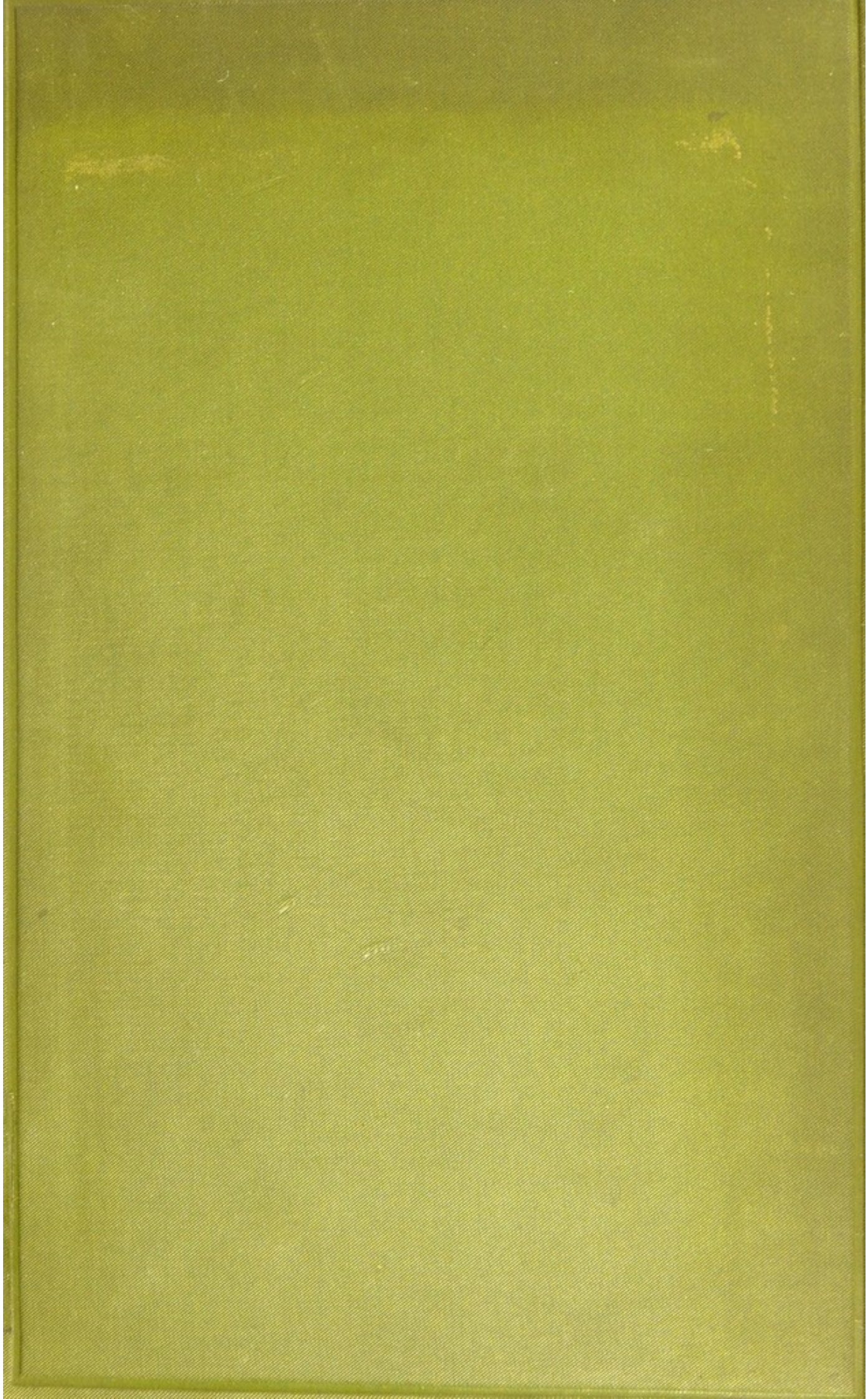
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LECTURES TO PRACTITIONERS.

LECTURES

TO

PRACTITIONERS.

ON THE DISEASES CLASSIFIED BY THE REGISTRAR-GENERAL AS

TABES MESENTERICA.

BY

W. T. GAIRDNER, M.D., LL.D.

— c

ON THE PATHOLOGY OF

PHTHISIS PULMONALIS.

BY

JOSEPH COATS, M.D.

WITH TWENTY-EIGHT ENGRAVINGS ON WOOD.

LONDON:

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PREFACE.

THE Lectures contained in this volume were delivered in the Western Infirmary, Glasgow, during the month of October, 1886. The delay of over a year in publishing has arisen from various circumstances, chiefly connected with the professional engagements of the authors, but it is believed that the lectures have not suffered, as the delay has allowed of a fuller revision of them. Dr. Coats has, with a view to completeness, added one lecture to the four delivered by him; otherwise the lectures are substantially the same as those given in the class-room.

GLASGOW, *February, 1888.*

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ON THE DISEASES CLASSIFIED BY THE REGISTRAR-GENERAL AS TABES MESENTERICA.

LECTURE I.—HISTORICAL.

The idea, or principle, involved in the name tabes mesenterica ; *origin of the name ; historical references and personal observations contrasted ;* Carreau, *as a popular and medical designation of the disease.*

THE subject of the present Lectures was suggested to me as following naturally out of a series partially published in the *Medical Times and Gazette* in 1884 and 1885, but arrested *in medio* by the extinction of that journal in December, 1885. The connection of that course with the present may not appear quite clear when I state that it was mainly devoted to the consideration of peritonitis in its various aspects; but I hope to show, in the course of the present Lectures, that the whole subject of peritonitis is inextricably involved in the symptomatology and pathology of *tabes mesenterica.*

The idea of *tabes mesenterica,* as adopted by the Registrar-General and many of the English authorities, may possibly be set before you with sufficient accuracy in the following extract from the well known book of Sir Thomas Watson, 5th ed., vol. i, p. 216 :—

" One form of scrofulous disease, common among children, is

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what is called 'tabes mesenterica.' Tabes and phthisis—the
one a Latin and the other a Greek word—signify, I need
scarcely tell you, the same thing: a wasting away, or a
consuming; and phthisis is applied to the same disease in the
chest to which tabes is applied in the belly. The common
English word is consumption, and we might very well speak
of thoracic consumption and of abdominal consumption; but
the technical name of the latter complaint is *tabes mesenterica.*
This is not only a common but a very fatal disease in children
and young persons. The glands of the mesentery enlarge, and
become charged with tubercular matter, but they very rarely
suppurate. Their enlargement is commonly connected with
tubercular disease and ulceration of the mucous follicles of the
intestines, and the little patients die because the lacteals are
no longer able to take up from the food a sufficient supply of
nutriment: they die starved. But some few do recover from
tabes mesenterica."

Sir Thomas Watson then proceeds to show from Carswell
what is now, I suppose, familiar to every pathologist of expe-
rience—that scrofulous glands, the mesenteric among the rest,
undergo a kind of cure through the arrest of all active morbid
processes in them, and the deposit of saline and earthy particles
in the cheesy-looking masses which constitute the so-called
scrofulous—or, as some hold, tuberculous—matter of the ear-
lier stages of the disease. I am not going to occupy you
much with strictly pathological dissertations in the lectures
assigned to me in this course, and accordingly I will assume
that you are all more or less familiar with the great con-
troversies that have arisen, especially in Germany, within
the last thirty years, as to the relation of this process of
caseation, in the glands and elsewhere, to tubercle properly
so-called; involving, of course, the question, long debated
by pathologists before the days of microscopic investigation,

as to the identity, or otherwise, of the scrofulous and the tubercular constitution. This subject, in almost all its aspects, was elaborately considered a few years ago by our Pathological and Clinical Society, and the discussions upon it, which were spoken of at the time as fairly representative of the state of scientific medical opinion, are open to your perusal.* I think, moreover, that it is probable that Dr. Coats may have something to say upon it further in his contribution to the present courses of lectures, and accordingly I shall content myself with a brief allusion to it, and with directing your attention to these illustrative preparations. [The preparations shown illustrated various stages and forms of chronic disease of the mesenteric glands and of the peritoneum, in combination and separately; also caseation, and, in one case, complete calcification of a considerable group of glands in the mesentery, without any known clinical history.]

It will be observed that the point of the extract given above from Sir Thomas Watson lies in the statement made (as if one of unquestionable fact) that "the little patients die because the lacteals are no longer able to take up from the food a sufficient supply of nutriment; they die starved." And there can be little doubt, I think, that this was the idea underlying the name, and giving, as it were, importance and specific character to this as the designation of a particular form of scrofulous or tubercular disease. We find, indeed, that under the slightly varying designations of tabes or atrophia or phthisis mesenterica, this form of disease is recognised more or less in many of the systematic works which appeared towards the end of the eighteenth century:

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* *Discussion on the Pathology of Phthisis Pulmonalis and its relationship to Tuberculosis.* Glasgow, 1881. (Also in *Glasgow Medical Journal* of same date.)

and yet it would hardly be correct to say that these designations were at any time universally recognised as those of a special form of disease. I have hitherto been altogether baffled in attempting to trace back the idea indicated by Sir Thomas Watson (of mechanical starvation through the interruption to the flow of the chyle) to its original source. The nosology of Sauvages gives Baglivi as the authority for the original employment of the name *tabes mesenterica* in the sense in which it has been habitually used; but, upon reference to the works of that somewhat desultory writer, I have failed to find any clear indication of the pathology in question; and although it must, of course, from its very nature, have arisen out of the great discoveries of the seventeenth century in regard to the lacteal and lymphatic circulation, I have hitherto not been able to discover any work earlier than 1750 in which that pathology is so much as alluded to. This date, moreover, is provisionally adopted, not as that of any very precise reference, but as the dividing line between the first and second half of the century. In two inaugural dissertations, published, one in Edinburgh, by Gul. Ball, in 1773, and the other in Glasgow, by A. Crawford, in 1774, *De Tabe Mesentericâ*, I find references to Juncker, Hoffmann, Stahl, Sennert, Sauvages, and to the now almost forgotten, but then probably well known, work of Richard Russell, *De Tabe Glandulari;* sive de usu aquæ marinæ in morbis glandularum dissertatio. Oxon, 1750. The Edinburgh thesist adds, however, in reference to the whole previous literature of *tabes mesenterica*, that in all these works the disease in question is very slightly treated (perpauca quidem perlegunter); and, further, that " Nulli attamen inter systematicos ex professo eundem tractaverunt. Boerhaavius, ejusque commentator Van Sweiten celeberrimi, nihil quidem de eo in medium protulerent." The works

of Baglivi are, in more than one place, occupied with discussions of what he calls mesenteric fevers; but it seems more probable, on the whole, that in these chapters he had in view something of the nature of typhoid fever rather than the chronic mesenteric disease of infants and children. Indeed, I am not quite sure if Cullen—who, in his nosology, gives by no means a prominent place to *tabes mesenterica*—was not among the first authoritatively to set forth the theory in question. In *tabes mesenterica*, he says, "the emaciation depends on an obstruction of the mesenteric glands, through which the chyle must necessarily pass to the thoracic duct." * In the excellent article on *tabes mesenterica*, written by Dr. W. B. Joy for Forbes and Tweedie's *Cyclopædia* in 1835, this theory is fully discussed; and, without being absolutely refuted, it may be said to have been set aside as inadequate, on the ground that such obstruction as would necessarily lead to the consequence referred to is quite unusual, if not unexampled. In the course of the discussion there is introduced a remarkable statement by Cruikshank, whose work on the lymphatic system was perhaps the most complete and advanced piece of anatomical research on the subject in the end of the eighteenth century (1790), to the effect that "in such enlargement of the glands, if it ever takes place, we should meet with the stagnation of the chyle in the first set of lacteals, yet I never saw such stagnation on any occasion whatever." The sceptical position indicated in this quotation is evidently shared by Dr. Joy, who adopts the opinion, then as now all but universally held, of the tubercular character of the disease in the glands, and argues that it is to the constitutional symptoms associated with tubercular disease, wherever occurring, that we have to look for an explanation of the hectic fever and the emaciation attending the so-called *tabes*

* Thomson's edition of Cullen, vol. ii, p. 562.

mesenterica. Indeed, it is rather remarkable that this scepticism appears to extend almost to the point of refusing to admit that emaciation would necessarily be the consequence of any amount of disease in these glands, or even that such disease would necessarily be followed by any symptoms whatever. The following paragraph includes statements which have been frequently quoted, and therefore perhaps demand to be mentioned here.

"Morgagni has mentioned the case of a negro cut off suddenly" (he was hanged, according to Ingrassias, the original source of the story) "in whom the mesenteric glands were found greatly enlarged and scrofulous, though he was, almost up to the moment of his death, in the enjoyment of excellent health; and Bayle records the case of a child which was burned to death while in perfect health, being fat and in good condition, though tubercles existed in the mesentery, and in some of them suppuration was actually commencing. There are probably few practitioners who have enjoyed extensive opportunities of the prosecution of pathological anatomy who cannot recall instances of young patients cut off rapidly by acute diseases in whom enlargement of these glands, which had been altogether unsuspected during life, existed." M. Guersent, in a well known and admirable article presently to be mentioned, carries this sceptical argument a little further still: for, after alluding to Morgagni's case (which he attributes to Ingrassias in the first instance) and to that of Bayle, in more detail than Dr. Joy, he gives it as his own personal experience that both pulmonary and mesenteric tubercles, if not inflamed, may not only exist, but "may arrive at the last stage of softening without notably disturbing the health, and without manifesting themselves by any pain, or by any sign that can be remarked. The persons affected by them retain their appetite and their flesh (*embonpoint*), and this fact is

important to be known also in its relations to physiology and pathology, *for it proves that the mesenteric glands are not the only way by which the chyle can pass into the blood*, and confirms, indirectly, the absorption by the veins, which is, moreover, proved (constatée) both by experiments and positive observations." (*Dict. de Médecine*, t. iv, p. 318.)

It is rather remarkable that, notwithstanding this scepticism as regards the theory of mesenteric glandular tabes, the name should have survived, in association with the theory, so long as to be thus imbedded in the classical work of Sir Thomas Watson up to its latest edition; and this, notwithstanding the fact that neither in France nor in Germany, nor yet in Italy where the theory might reasonably be supposed to have taken its origin, has the name *tabes mesenterica* been nearly so popular among nosologists as in this country, where its adoption by the Registrar-General as the designation of an important division of the tubercular or scrofulous diseases, has no doubt tended to its undue perpetuation. "Scrofula," writes Dr. Farr, "characterised by the deposit of a matter allied to, if not identical with, the tuberculous matter of phthisis, so frequently affects the lymphatic glands, that their chronic enlargement or inflammation (adenitis) is almost always considered scrofulous; the deposit of tuberculous matter in the mesenteric glands has a name (*tabes mesenterica*), as it is frequent in children." (Farr's *Vital Statistics*, Memorial vol., 1885, page 238.) And this name accordingly has been in England, Scotland, and Ireland extensively adopted as the only comprehensive designation of the tuberculous or scrofulous diseases of early age affecting the abdomen. It may be added that, according to an investigation by Dr. Greenhow, made at the instance of the Medical Officer of the General Board of Health, in 1858, the prevalence of *tabes mesenterica* as a cause of death in England and Wales was

found to be, for males, equivalent to a death-rate per 100,000
of 28, and for females, of 24; while in London the corres-
ponding death-rates for males were 44, and for females, 33
per 100,000. The variations in this rate according to dis-
tricts, &c., were minutely criticised in this report without
any indication that the name itself was regarded as an un-
satisfactory one; indeed, it is contrasted, page 110, with
the somewhat vague term scrofula, as "the well defined
form of disease called *tabes. mesenterica.*"

Personal Observations.—From an early period of my patho-
logical experience my attention was necessarily directed more
or less to this subject, although not, perhaps, with the direct
object of accumulating facts with reference to the theory
above mentioned. Had my mind, indeed, been predisposed
to partisanship, I should have found it difficult to place myself
on the side either of the upholders of the theory or the
sceptics; for, on the one hand, it soon became apparent to
me that the mesenteric glandular disease was by no means
the exclusive or even the chief pathological element in the
symptoms commonly attributed to it; and, on the other hand,
it would have been difficult to maintain, in the presence
of facts which came under my notice from time to time,
that such disease of the glands as amounted to distinct
obstruction was not capable, in the event of its being very
extended, of producing the mechanical results above adverted
to. Even the curious negative fact, apparently maintained
by Cruikshank, as to the absence of stagnation of the chyle
in the first set of lacteals, did not wholly accord with my
experience; and I am able to show you here a preparation
in which such partial damming up of the chyle, as a
consequence of disease of the mesenteric glands in the direct
course of the corresponding lacteals, appears to have taken
place. But, on the other hand, it may be admitted that such

appearances were entirely exceptional, and always partially distributed, so that it could hardly be supposed that the mechanical theory, however supported in principle by these examples, was, in fact, the chief or ruling element in the disease *tabes mesenterica*.

A Rare Case.—What, however, appeared to me still more important, as a jointly clinical and pathological observation, or rather as a pathological observation involving important clinical consequences, was that as a cause of abdominal intumescence in young subjects, recognisable by diagnosis, it very rarely indeed happened that mesenteric disease of any kind existed without complications such as would have made it entirely impossible during life, and barely possible after death, to assign to the mesenteric glands any separate share in the symptomatology of the disease. Mesenteric glandular disease, when leading up to death, was invariably complicated with disease either of the mucous or of the serous surfaces, to such an extent as to make it extremely probable that the mesenteric tumours were of quite secondary importance both in the diagnosis and in reference to the theory of the case. *Tabes mesenterica*, in short (considered in the natural signification of the words), might be said to have practically disappeared from observation altogether as an independent disease, to be replaced by other and much more complex conditions, among which tubercular peritonitis on the one hand, and tubercular ulceration of the mucous membrane on the other, were the chief and enormously preponderating factors entering into the diagnosis. Without having formulated any distinct conclusions on the subject, I had, during a course of nearly twenty years, been watching my available experience in hospital and otherwise (not specially children, however), when in the early part of 1867 a case was admitted to the Royal Infirmary of Glasgow which, throughout four months

of treatment, was very carefully watched, and in the end
terminated fatally; the *post-mortem* examination showing
what was at once recognised as a quite solitary and excep-
tional fact in my experience up to that time—viz., a con-
siderable and general enlargement, and a state of disorgan-
isation of the mesenteric glands which might have fully
entitled the case to the name of *tabes mesenterica* in the
most precise sense of the word. This case, however, was
not that of a child, and, whatever its pathology, it was not
a tubercular case. Indeed, it presented not the slightest
resemblance or even analogy to any of the cases I had been
accustomed to observe as corresponding with the symptoms
of *tabes mesenterica* in the child. There had been no evi-
dence of disease of the lungs, and the abdominal affections
were both accompanied and preceded by anasarca, so that the
physiognomy of the case was that of Bright's disease rather
than that of tubercle, and it was even with some surprise
that I found the urine to be non-albuminous. The par-
ticulars of this case are recorded in the *Glasgow Medical
Journal* for May, 1867, page 71, and it may perhaps be
sufficient to state here that after two months of treatment
" some indications of peritoneal effusion " were recognised,
" but, if so, moderate in amount." Diarrhœa and vomiting
had also begun to be troublesome, the dropsical swellings
continuing as before. Some weeks later " obscure indications
of tumour " were recognised, " which, when the swelling was
considerable, could only be felt by careful and deep manipula-
tion." The examination of these tumours on various occasions,
between 29th April and 8th May, led to the probable diagnosis
of their being mesenteric. They were found to be " of varying
distinctness—sometimes nearly superficial, sometimes over-
lapped by intestines; not capable of being identified with any
of the greater viscera, to a certain extent mobile, and chiefly

felt in left umbilical region, very dense, hard, irregular, and somewhat nodulated, altogether having much of the position and some of the characters of mesenteric glandular tumours."

I must here remark that these latter words were inscribed in the report made at the bedside during the lifetime of the patient, with the full belief that the case was an anomalous, or at least a very unusual one, and with all the reserves imposed by my own consciousness of unfamiliarity with mesenteric glandular disease recognisable distinctly as such. A few words from the details of the *post-mortem* examination will serve to indicate the special characteristics of this case, it being premised that the heart, lungs, liver, spleen, kidneys, and supra-renal capsules may be dismissed as, in general terms, practically normal :—" Mesenteric glands more or less enlarged from duodenum to ileum and from centre to circumference ; individual glands perfectly separate, varying from normal size to that of a small walnut. The mass of glandular tumours singularly hard and inelastic, giving to the mesentery and attachment of small intestine throughout a dense thickened feel. On section, individual glands solid throughout; no trace of active suppuration ; but at points a yellowish infiltrated matter, apparently supplanting and altering the glandular structure; section of glands for the most part presenting distinct hypertrophy of normal elements, with very marked variegated congestion. Intestines: Mucous membrane throughout thickened, dense, irregularly congested, the villous element highly developed, the *valvuli conniventes* well marked and continued low down in ileum; copious white mucus everywhere on surface, and towards the ileo-cæcal valve slaty discolouration and a few very superficial erosions. Peyer's glands can hardly be made out, and seem to be nearly lost in thickened mucous membrane. Omenta normal. No trace of special

disease of the peritoneal coat, with the exception of some very old adhesions of the liver to the diaphragm." Microscopic examination failed in this case to identify the structure of the diseased mesenteric glands as either tubercle or cancer, and the state of the other organs afforded no support to either theory. Emaciation had latterly become extreme, but was apparently accounted for by the diarrhœa and vomiting. There was no hectic fever of any importance, but abdominal pain had been present to a considerable extent. The patient was a carter, aged 21, who attributed his disease to cold and exposure, he having been previously of robust conformation. The case was, in my mind at the time, absolutely distinct from anything that I had previously observed as tabes or tubercular mesenteric disease; and accordingly I recorded it as being "quite unique within my experience;" adding that "though *tabes mesenterica* is a very common name, and stands for a considerable figure in the Registrar-General's returns, very few instances really occur of primary disease of the mesenteric glands. Rilliet and Barthez affirm they have never witnessed such a case, and most of the good authorities admit the extreme rarity of this condition. Bamberger, in *Virchow's Handbook*, alludes to one case only in his experience, in which (in a woman aged 60) the disease was mistaken for cancer of the stomach. The facts here recorded, therefore, deserve attention, apart from any special question of pathology or of diagnosis which might be founded upon them."

Although I am unwilling to burden your memories with a superfluity of detailed cases not actually presentable before you, this one, I think, from the point of view in which I have presented it, cannot but be interesting to you; the more so, perhaps, as I am able to connect it with another, of much later occurrence, in which an apparently closely corresponding diagnosis was *not* borne out by the *post-mortem* examination.

The success of the diagnosis in the one case, and its failure in the other, as regards the verification of the seat of disease, are of equal importance with respect to some parts of the argument about to follow, as showing the real insecurity of some of the physical signs that have usually been set forth as those of mesenteric glandular disease. This case occurred in the Western Infirmary, and is here given in brief abstract from the Journal X (Ward 6), p. 10, September to December, 1884. It was one of unequivocally malignant or cancerous disease of the peritoneum, in which paracentesis was performed about two months before death, thus giving the opportunity for careful examination in the comparative absence of fluid effusion:—

An Error in Diagnosis.—Catherine F., æt. 52, admitted 18th September, 1884. It seems unnecessary for the present purpose to adduce details up to the date of the paracentesis on 7th October; the patient had been aware since the end of July of a swelling of the abdomen rapidly increasing, and evidently ascitic, followed about a month afterwards by œdematous swelling of the lower limbs. She had been conscious of no other definite complaint; and as far as could be discovered from her statements, the solid tumours which afterwards became apparent had been wholly unknown to her previously to her admission. On the day before the paracentesis, Dr. Gairdner detected, on palpation of the abdomen, a solid resistance deep in the umbilical region, which, amid the obscurities in diagnosis caused by the fluid, appeared to be rather connected with the mesentery than with any more superficial part, and was so reported accordingly. After 149 ounces of fluid had been drawn off, the examination was renewed with the result of detecting an extremely irregular mass of tumours which, by their mobility among themselves, suggested an aggregation of greatly enlarged glands, rather

than any more continuous solid texture. This mass was completely disengaged from the liver, spleen, and right kidney, but it appeared to receive impact from the left lumbar region, and consequently its possible connection with the left kidney was reserved as a doubtful question. It is also indicated in a report soon afterwards that the question of a group of uterine fibroids had momentarily occupied attention, but had been dismissed owing to the absence of any evidence whatever of a connection of the tumour with the pelvis. To the end of the case a considerable amount of fluid continued present in the peritoneal cavity, and it appeared as if the relations of the solid tumours to this fluid were more in favour than otherwise of the theory of mesenteric glandular disease. But, before the patient died, other tumours or thickenings were detected corresponding apparently with the parietal peritoneum, both above the umbilicus, and in the hypogastric region. In connection with this it is particularly recorded that "the region proper to the omentum does not present any of this thickening, unless, indeed, the omentum can be conceived of as displaced or shrivelled up entirely into the epigastric region." The definite character of this report, made shortly before the patient's death, as bearing on the diagnosis of omental tumours, is remarkable, inasmuch as the omentum was actually found to be the chief seat of the thickening referred to, which, however, from its having underlain the fluid accumulation, and from its being extremely mobile as regards its individual parts, continued to present to the hand more the character of a mesenteric tumour than of omental disease. It will be observed, also, in the record of the *post-mortem* examination, that even had this source of fallacy not been present, it is probable that a diagnosis of mesenteric glandular disease could scarcely have been avoided, inasmuch as the mesenteric sub-peritoneal tissue (but not the

glands) was the seat of tumours which no amount of skill or *finesse* in diagnosis during life could possibly have distinguished from glandular enlargements, while the case presented in a high degree the characters of progressive and extreme emaciation usually associated with the idea of mesenteric tabes. The patient died on 10th December, in the last stage of emaciation, but without any complications indicating visceral disease. The *post-mortem* examination revealed the following facts:—

"The abdomen somewhat distended, but not very tense; a firm nodulated mass is felt floating across it in the position of the great omentum. The left pleural cavity is distended with serum, and the corresponding lung quite collapsed. The right pleural cavity contains about a pint of fluid. No cancerous nodules in the pleura. On opening the abdomen a massive pale tumour is at once exposed, while a large quantity of straw-coloured fluid escapes." [It may be remarked that the fluid withdrawn during life had been carefully and microscopically examined, but no distinctive elements, either of cancer or of tubercle, had been discovered; it was in all respects like an ordinary ascitic or serous effusion.] The connections of this tumour were briefly as follows:—It consisted mainly of the great omentum, "which is composed of a congeries of rounded tumours agglomerated together and compacted, the individual tumours being from the size of a hazel nut to a walnut. The omentum is thus converted into a solid dense mass measuring 14 inches transversely, $3\frac{1}{2}$ inches from above downwards, and 2 inches in thickness. The mass entirely covers the transverse colon, which is found behind it. It also involves the anterior wall of the stomach, which is here in one or two places continuous with the omental mass. There is also a great tumour formation in the lesser omentum. The under surface of the diaphragm is almost continuously

the seat of rounded tumours. The (peritoneal) wall of the abdomen contains many tumours; there are some in the epigastric and hypochondriac regions; but there are much more massive ones just above the brim of the pelvis on either side anteriorly. These are continuous with tumour masses inside the pelvis. The entire pelvic peritoneum is occupied by such closely agglomerated tumours, imbedding the uterus and rectum, but leaving them otherwise intact. The mesentery of the small intestines shows large numbers of rounded tumours, often almost pedunculated and like small marbles. They are most abundant over the mesenteric attachment of the intestine, and partly overlap the intestine in some cases. A few are on the peritoneal surface of the intestine. The caput cæcum coli is surrounded and partly buried in tumours. The descending colon has also large numbers surrounding it. *There is no enlargement of the mesenteric or prevertebral glands."* [Further particulars of considerable interest pathologically will be found in Dr. Coats' report, No. 1,269, Path. Register of the Western Infirmary, vol. vii; but these are all that have a direct clinical bearing on the present subject. Preparations illustrating the case are preserved in the Museum of the Western Infirmary.]

This case I present to you in abstract, not merely as an illustration of an error in diagnosis under circumstances where the complexity of the details makes such an error perhaps an excusable one, but also because I shall have to insist, by and by, upon the characters of a thickened omentum as one of the most significant *notes*, as it were, of the variety of chronic peritonitis which enters into the description of the so-called *tabes mesenterica*. It is, however, in certain rather rare cancerous cases that the characters of omental disease may be most easily studied; and one such case, as it happens, is at

present under observation in my male ward, and will be introduced to you after this lecture. [This patient had been tapped more than once for ascites, associated with great pain in the upper half of the abdomen; and after the second tapping a superficial thickening was discovered lying athwart the abdomen in the upper umbilical and hypochondriac regions, probably separate from the liver, and in front of the intestines. For some weeks after this lecture this patient was extremely ill, and was considered not likely to recover; he made, however, at least a partial and very considerable progress afterwards, and was dismissed with the tumour still quite apparent, but almost free from severe suffering of any kind.]

In France, the popular name of the so-called *tabes mesenterica* is *Carreau*, a name of rather obscure origin, but seeming to have some reference to the hard and cushion-like prominence of the abdomen. This name, of course, carries no theory whatever as to the nature of the disease, and even its association with mesenteric disorder seems to have been greatly overlooked, and its scientific character scarcely studied, up to the time of the prize essay of Jean Baptiste Timothée Baumes, of Nismes (afterwards Professor of Pathology and of Nosology, also of Clinical Medicine in the University of Montpellier), submitted to the Faculty of Medicine, Paris, 1787, and published with their approbation in 1788. A second edition of this work was published in 1806, and it has ever since been a standard authority. It is worthy of remark, however, that in the question proposed by the Faculty of Medicine for this prize the origin of the disease in the mesentery is assumed, the proposal being in these words:—"Décrire la maladie du mésentère, propre aux enfans, que l'on nomme vulgairement *carreau*, l'envisager dès son principe, rechercher les causes

qui la produisent, et exposer avec précision les moyens de la prévenir et ceux de la guérir." It is clear, therefore, that although the name *tabes mesenterica* is not used in this reference, nor much, indeed, in the work itself, the theory of a primary mesenteric lesion as the starting point of the disease had been entertained in France, and was adopted as the basis of Monsieur Baumes' researches; the loss of flesh (as *amaigrissement*) of the children and the inflation and hardness of the abdomen being directly attributed to this. " Young children," he says, " are very subject to emaciation succeeded by atrophy, at the same time that they take on a cachectic habit, and the abdomen becomes inflated and dense, afterwards indurated, and almost always painlessly. This disease has been as badly named as it has been ill described. It is certain that it has its seat in the mesentery, and taking into consideration the tumefaction and resistance (*rénitence*) of the abdomen, some have given to this affection the vulgar and metaphorical name of *carreau*; others, looking only to the principal *effects of the obstruction in the course of the chyle across the mesentery*, have named it atrophy of infants." Tulpius, Sydenham, and Lieutaud, with a number of minor authorities, are referred to, but none of them, apparently, so as very directly to bear out the theory.* From all this, as from the whole substance of the work, it is quite clear that M. Baumes was insensibly biassed to a great degree by the obstruction - theory above alluded to. The well known and classical article of M. Guersent in the *Dictionnaire de Médecine*, 1822, t. iv, art. *Carreau*, proceeds on very much the same lines, although upon a

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* Sydenham, however, is perhaps entitled to the credit of the first hint incidentally given of a connection between tabes, especially in infants, and abdominal tumours of strumous character connected with the mesentery. The passage will be found in his *Dissertatio Epistolaris de Affectione Hysterica* (1685), section 99.

much wider basis of observation; and it is remarkable that while fully acknowledging that he is not acquainted with a single case in which a child had died from an affection of the mesentery alone, and that in all the fatal cases he has observed it was combined with other diseases capable in themselves of producing this result, he has, nevertheless, insisted, more than perhaps any other of his countrymen, at once on the difficulty of the physical diagnosis, and upon the manual examination and discovery of the diseased glands as the only mode of distinguishing the mesenteric from the associated diseases. Upon this point I shall have a good deal to say in the sequel.

M. Bichat, certainly the greatest of French pathologists at the beginning of the century, in a course of lectures sketched out and probably written in 1805, expresses himself in the following terms of almost epigrammatic conciseness:—" The *carreau* is the engorgement of the glands of the abdomen. It comes on commonly in infants from the second to the eighth year. It declares itself at first by pains, disturbed digestion, frequently habitual diarrhœa, the belly is distended, there are frequently vomitings, . . . there is a delicacy of the skin, flaccidity, puffiness, heightened colour of the mucous membranes, small pulse, frequently difficulty of breathing, lactescent urine, . . . debility, feebleness in the movements, little development of the intellectual faculties. The tension of the abdomen is due to the swelling of the glands and to gaseous matters. When these gases do not exist, it is possible to feel the swollen glands, but this is the most rare case. Sometimes there is voracious appetite, at other times anorexia, abdominal pain, complication with worms. The general symptoms are the disturbed respiration, which arises, no doubt, from the consecutive enlargement of the thoracic glands; the marasmus, which some authors have given as a

characteristic sign. One might suppose at first that this (the marasmus) depends upon the *non-absorption of the chyle on account of the engorgement of the glands;* but it is only towards the later periods that this function is absolutely interrupted; and no one need be surprised at this who knows how the lung, in the later stages of phthisis, can equally fulfil its functions. At last the strength diminishes, the tumefaction of the belly is enormous, pain is excessive, and shifts its position *according as the mesentery is displaced by the movements of the patient,* diarrhœa is habitual, there is weakness and concentration of the pulse. Towards the close ascites, or infiltration of the lower limbs, supervenes. There is no disease which reduces infants to a more frightful state of marasmus." This clinical picture, the brevity and comprehensiveness of which lead me to quote it almost entire, is accompanied by a description of the various stages of enlargement and degeneration of the glands, which is, however, the less interesting to us that it has been entirely superseded by later and more exact descriptions. The association of glandular affections of the abdomen with a similar disease in the thorax is spoken of, but no hint is given of the other complications described by M. Guersent. The name *tabes mesenterica* does not occur.

Returning to the article of M. Guersent on the *carreau,* which he at once identifies at the outset with *tabes mesenterica,* he speaks of it as a vulgar name, metaphorically assigned to the tuberculous affection of the glands of the mesentery, *on account of the hardness and the volume which the belly frequently acquires during this disease.* He remarks upon other designations and particularly on the name entéro-mésentérite (evidently a Broussaisism), which he rejects as being wholly inapplicable, inasmuch as M. Petit (in 1813) had already employed that name to represent a disease (typhoid fever)

having no resemblance to the one in question. He proceeds to say that the *carreau* is not peculiar to infancy, inasmuch as mesenteric tubercles are found at all ages up to 50 or 60 years and more; and further, that even among infants it is by no means so common as has been represented. The anatomical description of the changes in the mesenteric glands is given in great detail, and its association with diseases of the mucous membrane is pointed out; this being peculiarly apt to be diseased towards the end of the small intestine. Extensive ulcerations, he says, are observed in more than half the individuals affected with this disease, but yet the connection is not essential. "The intestinal mucous membrane is often perfectly healthy in the whole extent of the canal, even although mesenteric tumours are very voluminous, and already in part softened. On the other hand, ulcerations of the mucous membrane are frequently found in phthisical persons, although the mesenteric glands are often unaffected." M. Guersent also remarks, that next to the inflammation of the mucous membrane and the intestinal ulcers, the most frequent organic alterations occurring in *carreau* are the retraction, thickening, and induration of the omenta with tuberculous degeneration, the consequences of the inflammatory affection, and of chronic peritonitis with or without sub-peritoneal tubercles; but all these lesions can only be considered as the result of the complications, more or less frequent, of chronic inflammations of the abdominal organs with the *carreau*. After a very extended description of the symptoms caused by these disorders, M. Guersent ends by the following remarkable admission, which seems of itself sufficient to suggest the doubtful propriety of the name *tabes mesenterica*. "It results," he says, "from this physiological discussion of the *carreau*, that nearly all the symptoms which, up to the present time, have been assigned to this disease

do not really belong to it, but depend upon several other
affections of the abdomen with which it is ordinarily con-
founded, or other diseases which accompany it as a rule, and
proceed side by side with it. The only pathognomonic
symptom, the only positive character by which the *carreau*
can be recognised, and this only in its last stage, is the dis-
covery by palpation of the tubercles. All the other symptoms
are more or less doubtful, and masked by those of the diseases
with which *carreau* is complicated. *Carreau* is, therefore,
one of those organic alterations which belong almost exclu-
sively to the domain of pathological anatomy. It forms in
the nosography a genus altogether artificial, to which it is
impossible for me to assign physiological characters distinct
from those of the diseases with which it is almost always
found associated, seeing that *I have never met with it in an
isolated form.*" It would appear probable that this frank
declaration has largely influenced the ideas and nomencla-
ture applied by the profession on the continent of Europe
to this affection, inasmuch as the name *tabes mesenterica*,
although, as we have seen, indicated by Guersent himself as
the scientific correlative of the popular name *carreau*, has
been practically abandoned to a great extent in France, and
also in Germany. After the detailed description, in fact, of
tubercular peritonitis by Louis, and subsequently by Rilliet
and Barthez, it is scarcely possible to point out any definite
recognition in French medical literature of *tabes mesen-
terica* as an appropriate nosological term; all the forms
of disease in question being described under the titles of
other tuberculous or scrofulous affections, such as tubercular
peritonitis and ulcerations of the intestines. In the latest
dictionary of the medical sciences published in France (*Dic-
tionnaire Encyclopédique*, edited by Dechambre), the patho-
logy of the mesenteric glands is systematically treated in

an article by Ernest Besnier, in which the secondary character of the disease is maintained, and the name *tabes mesenterica* does not once occur, nor is it to be found among the titles of the many works cited in the bibliography attached to this article. In Germany, while the name *tabes* continues to be used largely in connection with locomotor ataxia or tabes dorsalis, it seems to be entirely abandoned as a designation of mesenteric disease, so far as to be wholly unrecognised in the great *Cyclopædia of the Practice of Medicine* edited by Ziemssen, as well as in the still more recent one of Eulenburg.

LECTURE II.—NOSOLOGICAL AND CLINICAL.

The Nosology of M. Guersent involves a logical fallacy; analysis of the clinical ideas involved in Carreau. *The pathological bias. How it has come about that the conventional representations of the prognosis are much too generally unfavourable.*

IN the former Lecture we arrived, both on historical and on clinical grounds, at the conclusion that there is no sufficient basis, either in pathology or in nosology, for the name *tabes mesenterica* as applied either to one disease or to a group of diseases. This is not meant to imply that tubercular disease of the mesenteric glands is either infrequent, or of no importance when it exists; but that it is so constantly associated with and symptomatically blended with other morbid conditions as to have been incorrectly assumed as the special designation of a group. So much, indeed, is quite clear even from the article of Guersent, already quoted to you, and almost universally adopted as a leading authority upon the subject. It is notable, however, that M. Guersent's article, while fully deserving its position in medical literature as an inquiry into facts, is vitiated throughout by an error in logic, which is none the less remarkable because of the clear statement of the facts. Even while admitting that there are no pathognomonic symptoms, no positive characters by which, as he says, the *carreau* can be recognised; even while declaring that all its symptoms are doubtful, and masked by those of

the diseases with which it is complicated; nay, even while he maintains that it forms "in the nosography a genus altogether artificial," *carreau* remains for M. Guersent still identified in idea and in fact with *tabes mesenterica*, or at least with tuberculisation of the mesenteric glands. He takes a name which is confessedly a popular, and not a scientific, one; a name, moreover, which is popularly applied to conditions of the abdomen outwardly recognisable; and he insists upon applying that name, and its presumed correlative in Latin, to a disease which, as he himself says, "belongs almost exclusively to the domain of pathological anatomy;" of which, moreover, he had never met with a single isolated instance, and of whose symptoms, therefore, as a separate and independent disease he himself can give no account. This error in logic, I apprehend, is not peculiar to M. Guersent, but extends more or less to all who, at least since his time, have allowed themselves so to employ these terms. Many, no doubt, have avoided the error by practically throwing over the term *tabes mesenterica ;* as, for example, almost all the German and many of the more modern French writers on the diseases of children; but still the fact remains that the latter term continues, more or less, in use; and that wherever it is in use, it always suggests the same idea. It would seem, therefore, not to be a work of supererogation to attempt to reconstruct the nosological edifice which I have shown to be so unstable; and to show you, from the clinical rather than from the pathological point of view, what, if any, are the foundations on which it rests. This I hope to do in such a way as to lead to some practical conclusions of more or less importance as we go on.

Clinical Idea of the Disease. — We must, however, lay aside for the moment all ideas exclusively derived from pathological anatomy, as of the mesentery and its glands

infiltrated with tubercle; and on the other hand, all such fanciful notions as the purely mechanical one of obstruction to the course of the chyle, to which I have already adverted. If we take now the popular word *carreau* in its original and purely popular meaning, which, nevertheless, though metaphorical, is quite truly the expression of a fact, we shall find that the leading idea brought up by the term is the *prominence* and at the same time *induration* of the abdomen in young children. As long as this purely clinical fact was placed in the front of the picture, the describers of the *carreau* were on safe ground, clinically speaking. It was when they departed from this in search of a pathological explanation of the fact that they were led into error; and this remark applies still more to the work of M. Baumes than to that of M. Guersent. That an important and fatal disease of infancy exists, characterised by a hard and tense abdomen, and that this disease is fatal in many or most instances after the manner of a *tabes* or *atrophy* is, and remains, a fact beyond all doubt; but when we attempt to look closer at this fact, and to discover exactly what it means, we find ourselves at once involved in a difficulty from which, as long as we cling to the mesenteric theory, there is practically no escape; for although it may be true that mesenteric disease, or tubercles of the mesenteric glands, are to be found in many or most of the cases in which such hard enlargements of the abdomen prove fatal, it is impossible to maintain that such tuberculous glands are the cause of the abdominal enlargement, and it is more than doubtful (as we have seen) whether they are in most cases the cause of the fatal atrophy. As almost all modern authors admit, they are probably only the incidental accompaniments both of the one and of the other; nay, I am disposed to go even a step farther than this, and to say that mesenteric glandular disease, did it

ever exist as a separate nosological form, would in all probability not present the clinical features of the *carreau;* certainly it did not do so in the single case which I have placed before you in the preceding lecture. Now, when we consider the matter from the side chiefly of the tumid abdomen, it is necessary to make some further and some strictly clinical distinctions. The abdomen may become tumid, as we all know, from mere flatulence, with or without constipation; from mere muscular relaxation and deficient peristaltic power of the intestines; from mechanical obstruction in the course of the intestinal canal, or from scybala accumulated within it; or, on the other hand, distension may take place from fluid effusion in the peritoneum (as in simple ascites), or from cystic tumours, or even from solid tumours of the viscera other than the mesenteric glands, and especially of the ovaries, uterus, liver, and spleen. All these forms of distension, however, differ very notably from that which is implied in the idea of the *carreau;* and it is to these differences that I wish to direct your attention for a short time.

"The enlargement of the abdomen," says M. Guersent, "on the ground of which popular opinion pronounces boldly on the existence of *carreau* among children, especially when the characteristic emaciation of the limbs, and pallor of the face are united to it, is absolutely insignificant. Most children up to the age of three or four years have a voluminous abdomen; the intestinal canal is proportionally longer than that of the adult, as it is still more in the fœtus. . . . When children have a weak intestinal canal and difficult digestion, the intestines are often distended by gas; the abdomen is almost always blown up and resounds like a drum. This disposition is all the more remarkable among feeble infants having a narrow and ill-developed chest, because in them the liver is more voluminous, and tends accordingly to press down the

mass of the intestines. Rickety infants are all affected in this way, and yet very few of them present mesenteric tubercles. . . . They are much more frequently affected with diarrhœal discharges, and particularly with the mucous and sanguinolent diarrhœa which depends ordinarily on a cæco-colitis, a disease so common among young infants, that at least a fifth of those who die from the time of birth to the age of five or six years may be said to be affected with the disease either alone or as a complication." Accordingly, Guersent maintains, not without reason and in the spirit of the whole article, that the tumid abdomen is by no means characteristic of the *carreau*; and further, that it is frequently not present when tubercles of the mesentery exist in young children, and is scarcely found at all in similar cases in adults.

I am not concerned to dispute any of these propositions; and yet it may be worth while to look a little more closely at the various kinds of swollen abdomen here alluded to, in contrast with others. None of those described in this para-graph, it appears to me, are characteristic of what is popularly termed *carreau* or, according to the imperfect scientific con-ception of it, *tabes mesenterica*; for in both of these cases the idea of hardness, and even of solid resistance, forms a part of the diagnosis by no means to be neglected, and to which I am going to ask your particular attention. I shall in these remarks, accordingly, have regard not only to what is tech-nically called *tabes mesenterica* or *carreau* by the writers of the beginning of the century, but also to the descriptions of chronic and tubercular peritonitis, in those later and classical works of which the splendid French work of Rilliet and Barthez, and in our own language the one well known to you all of Dr. West, may be considered the leading examples. From the latter I would desire to read to you only a few sentences as an introduction to what I have now to say.

Speaking of tubercular peritonitis, Dr. West* writes:—"Some of you have probably been struck by the many points of resemblance between the symptoms that have just been described and those which are often enumerated as character-istic of mesenteric disease.   Nor is it at all surprising that a very close analogy should subsist between chronic peritonitis and *tabes mesenterica*, since not only are both affections the results of the tubercular cachexia, but in both the abdominal viscera are chiefly involved in the disease, and both are in consequence characterised by a remarkable impairment of the functions of nutrition.   It was natural, too, that in former times, when morbid anatomy was less carefully cultivated than at present, the attention of the observer should have been chiefly drawn to the increased size and altered structure of the mesenteric glands—appearances which must have been often discovered on an examination of the bodies of children who had died after a slow wasting of their flesh, attended with more or less enlargement of the abdomen, and disturbance of the bowels. *The physiology of those days, too, knew of no means whereby the absorption of the chyle could be effected, except through the medium of the mesenteric glands;* and the coarse appliances which then subserved the purposes of anatomical investigation did not suffice to show that, even when these glands outwardly present a considerable degree of tuberculisation, their lymphatics in many instances are still pervious.

"We know that the nutrition of children is often much impaired from other causes besides tubercular disease; and that, when the digestive organs perform their functions ill, nothing is more common than for the abdomen greatly to exceed its natural size. Our predecessors had observed similar facts; but, owing to the imperfection of their physiological

* *Diseases of Children* (4th edition), pp. 628, 629.

knowledge, they drew from them erroneous conclusions. Disease of the mesenteric glands was in their eyes the almost exclusive cause of the atrophy of children, and a preternatural enlargement of the belly was looked upon by them as an almost infallible sign that such disease had already begun. *Tabes mesenterica* was consequently regarded as a very common affection; and though its frequency is now well known to have been much overrated, yet the appearance of those symptoms that were once supposed to be characteristic of it, still excites much needless alarm among non-professional persons."

Clinical and Pathological Ideas contrasted. — The only remark I would make on these sentences, with the general purport of which I entirely concur, is that while both tubercular peritonitis and mesenteric glandular disease must necessarily come within the purview of the remarks I have still to make, I wish it still to remain in suspense whether these two pathological conditions completely occupy the field of clinical observation which I have to present to you. That they occupy a large part of it I am personally convinced, but perhaps there may be some reason for doubting whether they occupy the whole of it. Here, as in very many other instances, pathological anatomy has given us information, extremely valuable, no doubt, and accurate of its kind, but still information *with a bias.* It has told us in great detail, and with great precision, *what has happened to those who die;* it has not told us with anything like the same precision, sometimes it has not told us at all, what has happened to those who recover. What I have now to tell you applies to both; and therefore it is that I keep the clinical aspect of the case before my mind rather than the pathological.

Now, among the crowds of infants and young children who fall into disease mainly characterised by abdominal symptoms

in the first instance, it is beyond all doubt that a large proportion come under the suspicion of being tubercular; or, if you like to call it so, scrofulous. They may have had phthisical parents; they may belong to families in which deaths from manifestly tubercular diseases have occurred; the patients themselves may have had, or may have, disease of the lungs manifested either through symptoms or by physical signs; they may have had enlarged glands in the neck or in the axilla, or may present such a typically scrofulous physiognomy as I show you in this very beautiful and characteristic water-colour drawing; they may have had, or may still have, scrofulous affections of the bones or joints, associated or not with changes in the viscera, such as are now commonly called amyloid, with their characteristic symptoms; or the abdominal enlargement may have succeeded to a period of vaguely deteriorated health and disturbed digestion with diarrhœa, &c., which of itself would amount to presumption, at least, of tubercular ulceration of the mucous membrane. But on the other hand, as I have good reason to know from experience very carefully investigated and recorded, abdominal enlargements of precisely the same kind may occasionally occur quite apart from all or most of these other incidents, and therefore I wish to dwell a little upon such enlargements under their least complicated aspect in the first instance.

A child is brought to you who may or may not have been complaining for some time of the abdomen, and in whom there has occurred, perhaps within a period of days or weeks, such a notable enlargement of the abdomen as has appeared to call for medical advice. Most of you have seen scores of such cases; many of you see them every day. There may have been diarrhœa or there may not; very often there has been a period of diarrhœa controlled more or less by remedies, and then swelling when the diarrhœa has ceased; or the swelling

and the diarrhœa may go on simultaneously; or, on the other hand, there may have been no diarrhœa at all, or even obstinate constipation, or this alternating with diarrhœa. In like manner the swelling may or may not be associated with severe pain. There may have been pain and probably tenderness, and it may have disappeared before you see the patient; or pain or tenderness, either or both, may still persist; or they may neither of them have been present in any appreciable degree at any stage of the disease. If, under these circumstances, you judge that the swelling is merely tympanitic; if, as M. Guersent puts it, "the abdomen is blown up and resounds like a drum"—and this in a very young child within the rickety age, and perhaps with symptoms of incipient rickets, or even without these accompaniments—you will probably be quite correct in assuming that it is not an affair of very much importance. A few laxative doses, or the use of carminatives, rubbing with the hand, and other simple expedients, or even the lapse of a few days without remedies at all, may dispose of such a case at once, and remove all that is worth removing of the apparent disease. I agree entirely with what your experience, as well as almost all the authorities, will concur in affirming, that such a condition of the abdomen gives no evidence of organic change. But in other cases the enlargement of the abdomen is not accompanied by this complete tympanicity; the sound given forth is equivocal or of ambiguous interpretation; or, it may be, there is partial dulness on percussion over points that ought to give the clear intestinal note; and at the same time there are apparent inequalities in the resistance to palpation, which, no doubt, if the abdomen is very much distended (as in some cases of mechanical obstruction of the bowels), may be due to individual coils of the intestine filled with fluid and intercepting the clear percussion note; or even (as Skoda has shown)

to the very excess of tension diminishing or obliterating the normal resonance. The difficulty is how to deal with these ambiguous cases, and it is upon this that I hope at least to throw some little light.

Certain Aspects of Diagnosis.—I would strongly recommend you in such cases never to be satisfied with one examination. Examine the whole abdomen in detail, in various positions of the patient, with every degree of force of percussion that can be reasonably employed, and also by palpation. Note the results carefully, and return once and again to the subject, employing, if you like, such rough diagrams as I show you here, or in some cases making marks on the external surface of the patient's body to guide you in a subsequent examination. Leave as little as possible to mere recollection, and try to have at least very positive facts on which ultimately your theory of the case may proceed. If it presents from day to day a series of dissolving views, as it were, then, of course, the probability is that the conditions are evanescent, and therefore not connected with organic change. But in certain cases the facts will gradually take on order and consistency, and will, if I am not mistaken, sometimes assume one or more of the following successive aspects:—

1st. The abdomen may present all the physical character of a certain amount of fluid effusion together with tympanitic distention, the relative areas of these varying with the position of the patient. In such cases the clear percussion of the umbilical region in the recumbent posture, the dulness of the flanks and hypogastrium in the same posture, with the specially and distinctively clear gastric percussion in the epigastrium and left lateral region, usually leave no doubt as to the existence even of a moderate quantity of fluid. If any doubt remains, turn the patient over upon his hands and knees, and

observe if the perfectly clear umbilical percussion is replaced by dulness. With these signs you may, of course, have fluctuation; but I am speaking of cases chiefly in which the amount of fluid may be too small to give this sign, at least unequivocally.

2nd. The alternation of dull and clear percussion, as in the former case, may exist, but the dull percussion may not gravitate, or alter with the position of the patient. And yet there may be, in the same or other parts of the abdomen, distinct evidences of fluid accumulation and even fluctuation.

3rd. The greater part of the abdomen may be dull to percussion, or dulness may predominate largely in the umbilical region, while the percussion remains perfectly clear in the gastric region, or over the transverse colon, or in one or other groin; and in this instance, also, change of position may fail to modify the facts, or may modify them so slightly as not to be consistent with the theory of freely gravitating fluid.

4th. All the conditions, 1, 2, and 3, may appear in definite succession, and yet gradually evolved over a period of days or weeks, in such a way that an experienced physician may apprehend in advance, or even predict, the order of their appearance; or, the first stage may appear to be wanting and 2 and 3 may alone be presented to observation; or, the state observed from the first, so far as it can be distinctively ascertained to be abnormal, may correspond rather with number 3 than with any of the others.

I insist upon these details, which you will find to be all more or less carefully illustrated in cases which I have published in the *Medical Times and Gazette* (see especially 29th August, 19th and 26th September, 1885), because, according to my experience and reading, they are often neglected, or very imperfectly stated in their relations to this important subject, even in the most advanced elementary works on

physical diagnosis; and even in the best modern treatises on diseases of children, I have been led to remark a want of definiteness in their teaching on the subject. Unless, in fact, you thoroughly master these details, and thoroughly apply them in each case, you may just as well go back to the old vague description of the *carreau* as an inflated and resisting abdomen, without further distinction. But, with the aid of these details, I fully expect that you will be able to make out the following (surely very important) clinical facts:—

In many of the children who come to you with a distended abdomen you will not have much difficulty in discovering, through palpation and percussion employed together, that besides the tympanitic condition of some portion of the intestinal canal, there is distinctly evidence of fluid effusion or ascites. Now, unquestionably ascites may occur in the child from the same causes and under the same circumstances as in the adult; but this is rare, comparatively speaking. In particular, ascites in the child is very rarely determined by obstruction of the portal circulation, although I have seen a very few cases so determined. And "primitive" ascites, as Rilliet and Barthez call it, is in the child, confessedly, extremely rare. It may therefore be assumed as probable, almost indefinitely probable, in advance that infantile ascites is associated with some kind of disease of the peritoneum itself; and in the few cases in which in my hands it has been such as to require paracentesis, it has invariably been attended by circumstances showing this to be the case.

But in the great majority of cases fluid effusion, though it may be present, is small in amount, and never even suggests a question of paracentesis. Now, it is in these cases very particularly that the question arises:—Assuming that there is fluid, and yet not a great amount of fluid, and that this arises from disease of some kind in the peritoneum itself, are there

also inflammatory alterations present, or at least such organic alterations as tend to restrain the movements of the intestines floating in the fluid? If the small intestines are matted together or bound up in a mass in front of the spine, or in any way, indeed, hindered from coming towards the surface; then the gravitation phenomena almost invariably observed when a merely serous effusion, moderate in amount, is present, may be expected to undergo modifications, or even to be entirely intercepted. Hence the practical importance to you of the observations conducted under the heads 2 and 3. In point of fact, I may tell you, as my own personal opinion, that most of the cases, to which the name of *carreau* can with propriety be applied, have at one time or other presented the conditions here referred to—*i. e.*, the evidences of fluid, probably in small or moderate quantities, in the peritoneal cavity, but at the same time so disposed as not to permit of the shifting of the clear and dull percussion-area, according to position.

But, further, we have seen that the idea of *carreau* proper involved the existence of *preternatural hardness*, as well as distention, of the abdomen. Now, what is this hardness or preternatural resistance? The rickety abdomen, and even the distended abdomen which arises from constipation or tympanites, may be more or less resistent, but it is always elastic, unless from spasm of the muscular wall; and this will always disappear under anæsthesia from chloroform. The abdomen proper to *carreau*, on the other hand, is more or less permanently non-elastic, dense, and in the most marked cases giving an impression almost of a solid tumour; and yet such tumours are quite distinguishable, as a rule, from enlargements of the solid viscera. I have seen very many such cases, and have rarely felt any doubt at all as to this. You may take it as being an established fact, within my experience, that these

semi-solid and non-elastic enlargements of the abdomen stand in a very close relation to the phenomena I have already described, and that they are usually, if not always, dependent upon thickening of the peritoneum itself, or perhaps of the peritoneum and the adjacent sub-peritoneal connective tissue, and especially of that portion of the peritoneum which forms the greater omentum.

Undue Gravity of the Prognosis.—All the facts on which I have now insisted are well known as a part of the pathological anatomy of tubercular peritonitis; that is to say, they have been studied, even minutely and carefully studied, in detail, from the side of pathological anatomy, and in fatal cases. I am not, therefore, in any way pretending to teach you anything new, when I say that the diagnosis of *tabes mesenterica,* or *carreau,* is inextricably mixed up with the physical signs of peritoneal rather than of mesenteric glandular disease; and that it is even doubtful how far the latter enters at all into the diagnosis from physical signs, as commonly observed. But I am, nevertheless, clearly of opinion that the precise observation and the just significance of these physical signs *in cases which are not fatal,* but which make, at all events a temporary, and in some cases a permanent, recovery, has not hitherto had sufficient attention bestowed on it; and one consequence of this has been, that in even the most justly esteemed monographs, as well as in most of your handbooks, the *prognosis* in these diseases inclines far too much to the grave and even hopeless aspect of them, and fails to recognise the existence of more or less similar cases which would tend to qualify that prognosis. How, indeed, could it be otherwise, when it is constantly assumed that nothing of a precise nature is, or can be, known about these diseases except through pathological anatomy— *i. e.,* after the case has proved fatal? In the great work of

Rilliet and Barthez, for example, nothing can possibly be more elaborate than are the descriptions in every detail, and also the numerical data, founded upon the pathological anatomy, both of the glandular disease and of the peritonitis; but *when they come to the symptoms*, the picture at once becomes hazy and apparently altogether uncertain. " The local symptoms," it is said, " of tubercular peritonitis are sufficiently numerous, but it is often difficult to appreciate their value. We have no criterion which will admit of our recognising the disease at a period near the beginning, or which at a later period indicates, in a positive manner, its progress and its extent. We are reduced to approximative estimates based upon the more or less considerable volume of the abdomen, upon its external aspect, its percussion-resonance, its tension, its inequalities, and the tumours which palpation reveals, or the pains, more or less acute, of which it is the seat." In the discussion of each of these phenomena, nothing but praise or admiration can be, or ought to be, accorded to the authors of this great work; but still, I think I am not overstating the case when I represent them as guided mainly by the idea that the diagnosis, to be clearly established, must either be corroborated by pathological anatomy, or must, at all events, present lesions so advanced as to be certain to end in death. Their prognosis, accordingly, is discouraging to the last degree. It resolves itself into the syllogism (not, it is true, expressed anywhere in such terms):—

Certain fatal cases have had such and such symptoms;

But the earlier stages and the non-fatal issues of such cases are practically unknown to us;

Therefore, such cases are always, or almost always, fatal.

Now this, Gentlemen, is exactly the same bias that I have already referred to as influencing the progress of our knowledge in all cases in which pathological anatomy has greatly

extended, or rendered greatly more precise, our conceptions of organic disease. In respect of cardiac disease, pulmonary disease, renal disease, perhaps even cerebro-spinal disease, we have had over and over again to learn the same lesson—namely, that certain typical aspects of disease, which we have come to know accurately only through pathological investigation, have thereby come to be regarded as hopeless, or, at all events, of exceedingly grave prognosis, the milder cases and those which admit of recovery, partial or complete, being, as it were, discounted, because they have not received the verification which, according to the thorough-going pathologist, arrives only when the subject is placed on the *post-mortem* table. Now, as I view the matter, *carreau*, or *tabes mesenterica*, is at present in this, perhaps too unfavourable, position. Its diagnosis is confessedly difficult in the early stages. Its prognosis, is, under the most favourable views that can be taken of it, confessedly grave—that is, *in well established cases*, it causes a great amount of mortality; but are we therefore to conclude that all the cases which recover, or appear to recover, are cases of a different order entirely? Such is not the conclusion to which I have been led in the course of my experience.

You will now see why it is I have insisted so much, in the earlier part of this lecture, upon the necessity of keeping our eyes fixed upon the clinical, rather than upon the pathological aspect of this subject. Not by any means that we are to cast aside, as valueless, the great and fruitful labours of the pathologist, but that we are to use them in the spirit and after the manner of the physician, who aims at curing his cases rather than completing his diagnosis of them by pathological anatomy. We must, in other words, absorb the facts of pathological anatomy into our diagnosis; but we must avoid, if possible, the fatal bias which arises from the patho-

logical method of contemplating these facts. Hence, although it may be admitted, and freely admitted, that many cases of chronic peritonitis, or of glandular disease, are tubercular, and that many of these die; yet it by no means follows from this that cases of apparently the same order, but which recover, are not cases of mesenteric disease, or of chronic peritonitis. We may admit that a certain amount of doubt necessarily attaches to the diagnosis: but we must not allow this doubt to carry us so far as to affirm positively the extremely dangerous, or hopeless, or necessarily fatal character of all such cases. I suppose there is not one of my audience who has not witnessed one or more recoveries under circumstances which, had the issue been death, tubercular peritonitis or *tabes mesenterica* might have become the assured diagnosis. My argument is, that, in some of these cases at least, the diagnosis was probably justified, even in the face of the recovery.

LECTURE III.—DIAGNOSIS AND PROGNOSIS.

Carreau and Chronic Peritonitis; association of the latter with Tubercle by Louis, and unduly grave prognosis accordingly.

FOUNDING on the facts which I have already submitted to you, I think we are fairly justified in affirming, that while the existence of *tabes mesenterica* as a distinct nosological type—*i. e.*, as characterised by disease chiefly or exclusively of the mesenteric glands—can hardly be maintained, there is, nevertheless, a definite group of diseases corresponding in general, clinically, with the suggestion of the popular name *carreau*—viz., that the dense resistance of the abdomen as well as its fulness, and the alterations to percussion as well as to palpation, are characteristic of something more than merely flatulent distention. Structural changes of some kind are undoubtedly present in these cases. In not a few of them the physical alterations in the abdomen render it very probable, and in some quite certain, that fluid effusion is present at certain stages of the disease; in others, this evidence is wanting; the disease, so far as observed, has not been characterised by a stage of fluid effusion. But even in these cases, or some of them, it is necessarily open to doubt whether such a stage may not have preceded the first actual careful observation. Cases every now and then occur in which it is perfectly certain, from the history and symptoms actually observed, that patients in this disease may pass

through the stage of fluid effusion without considerable suffering; and accordingly, unless the mere bulk of the abdomen is such as to bring such cases under medical observation, they may easily escape notice. In some cases, again, where the amount of the fluid is moderate or small, and where it is so disposed as not to gravitate, or rather where the intestines are so disposed as not to levitate, fluctuation also being absent or not definably present, questions of the utmost difficulty arise; and he would be a very rash diagnostician who should pretend to a clear opinion in all such cases as to the precise nature of the changes entering into the abdominal swelling. All that can be fairly stated as matter of evidence in many of these cases is, that there are structural changes of such a kind as to intercept the normal intestinal percussion note; from which it is reasonable to infer that the intestines are, in some way or other, withdrawn from the anterior abdominal wall under circumstances when, if the parts had not been thus altered, they would certainly have given a tympanitic note. But as pathological anatomy and clinical observation concur in teaching us that in many of these cases the structural changes are in the peritoneum rather than in the mesenteric glands, and that, even when these latter are affected, the former is apt to be equally or more affected, the diagnosis which is on the whole safest *a priori* in any individual case is also that which most frequently concurs with the physical conditions observed in the majority of cases—namely, thickening of the peritoneum, with or without the effusion of fluid. Cases will occur, no doubt, in which any attempt at precise diagnosis will occasionally give rise to error. An omental tumour, as in the case of C. F. (see Lecture I), may be mistaken for glandular enlargement in the mesentery; or, *vice versa*, the latter for the former; but in the majority of cases, when the resistance is great, when the dull percussion

extends over the greater part of the umbilical region, and -when, notwithstanding this, there is no evidence of very large fluid effusion actually present, although there may have been such effusion at an earlier stage, the probabilities are greatly in favour of chronic peritonitis, whatever may be the symptoms, or whatever may be the accessories tending to show the tubercular or scrofulous constitution of the child. In by far the greater number of such cases, the facts are such as to corroborate this diagnosis; and, as regards fatal cases, almost every one is aware of the opinion, entertained since the time of Louis, that chronic peritonitis is always tubercular. This opinion, it is true, has been brought into question; but it still holds the field in many of the best informed text-books, especially as regards young subjects; excluding, of course, from consideration cases of circumscribed peritonitis, such as occur when adhesions are formed over the liver, spleen, or at some particular point in connection with morbid alterations in the viscera of the abdomen. Bauer,[*] who has treated the whole subject of diseases of the peritoneum in a very comprehensive manner, refers to Toulmouche (*Gazette Médicale*, 1842) and Galvagni (*Rivista Clinica di Bologna*, 1869) as two of the most trustworthy authorities on the subject of chronic non-tubercular peritonitis; and he further details two cases from the late Professor Lindwurm's clinique in Munich. He

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[*] Ziemssen's *Cyclopædia*, vol. viii, p. 292, on "Chronic Diffuse Peritonitis." He deals with the opinion above referred to as follows :—" Louis, notwithstanding his valuable work, has brought a certain amount of confusion into the subject by making the statement that every case of chronic peritonitis is tubercular. Up to the present day the effect of the statement has not been altogether eradicated; and hence the existence of a primarily chronic simple peritonitis is by some observers entirely called in question." He admits, however (p. 300), that "the diagnosis of chronic non-tubercular peritonitis is very difficult"; from which it may fairly be inferred that in cases not fatal, or ending in complete recovery, the diagnosis may often remain impossible.

recognises three forms:—1st, A chronic stage, supervening on an attack of general acute peritonitis; 2nd, Chronic peritonitis, arising in the course of old-standing ascites; 3rd, More or less copious effusion, latent as regards its origin, so that the commencement of the attack can scarcely be defined. This last form he regards as very rare, so much so that its existence has been altogether denied by medical men of experience. One case, however, given in illustration, is completely demonstrated by the fatal result and *post-mortem* examination—an attack of small-pox having carried off the patient while under treatment in hospital. In this case, the large amount of effusion found was nearly serous; but nothing is stated in detail as regards the physical signs during life, except that there was increasing distention of the abdomen, " with a considerable quantity of very movable fluid and slight tenderness on pressure " (p. 298). It is sufficiently evident that in cases of recovery it would be quite impossible to distinguish this variety of chronic non-tubercular peritonitis from ascites, and scarcely less difficult, unless from the mere fact of recovery, to distinguish it from the much more common, or at least more commonly fatal, tubercular peritonitis. The apparently exhaustive analysis of the facts in Bauer's memoir justifies both of these assertions; but I will not detain you by going into details. Practically, it might almost be held that cases of apparent ascites which get well are more likely than not to have been cases of this form of chronic peritonitis. And this would hold especially of infantile cases in which, according to Galvagni, chronic peritonitis, with mainly serous effusion, has a rather favourable prognosis; while, on the other hand, true ascites, as we have seen (arising from portal obstruction), is both extremely rare and very unlikely to end in recovery. As regards tubercular peritonitis, its gloomy prognosis, as described in systematic works, is notorious.

There is scarcely a single exception, among the successors of Louis, to the statement that such cases are all but invariably fatal; and, perhaps, the only isolated fact tending in the opposite direction, which can be appealed to as of the nature of a demonstration, is one that has been now repeatedly referred to, having been in the first instance, I believe, set forth in this connection by my colleague, Professor M'Call Anderson, in a lecture on the subject.* This was a patient of Sir Spencer Wells, aged 22, in whom a small incision was made below the umbilicus for the surgical relief of what was supposed to be a "thin non-adherent unilocular ovarian cyst." "On opening the peritoneum," we read, "a large quantity of opalescent fluid escaped, and then the whole of the peritoneum was seen to be studded with myriads of tubercles." "Some coils of small intestine were floating; *but the great mass was bound down with the colon and omentum, all nodulated by tubercle, towards the back and upper part of the abdomen.* The uterus and ovaries were felt to be of the normal size, but their peritoneal coat was very rough."† The singular importance of this case can scarcely be denied, whatever doubts may be entertained as to its true pathology. A sceptical inquirer, indisposed to admit the cure of tubercular peritonitis, will, of course, demur to accepting an isolated case like this, in the absence of a thorough investigation as to the precise nature of the morbid deposit in the peritoneum; a follower of Koch would naturally demand at least one *instantia crucis*, wherein the tubercular bacillus shall have been shown to have been present in the fluid. Be this as it may, the fact remains that a condition of the peritoneum evidently attended with very appreciable

* "On the Curability of Attacks of Tubercular Peritonitis and Acute Phthisis" (1877), p. 14.

† *Diseases of the Ovaries* (1872), p. 135.

thickening, binding down the mass of the intestines with the colon and omentum, in the midst of a large effusion of opalescent fluid — chronic peritonitis, in short, of the most typical kind, and attended with all or most of the unfavourable local conditions found in cases of tubercular peritonitis — was not inconsistent with an apparently good recovery. This patient not only so recovered, but we are told by Sir Spencer Wells that she afterwards married.*

Prognosis of Chronic Peritonitis.—My own observations for many years before this had led me to believe in the occasional permanent recovery from tubercular, or at least chronic, peritonitis. It was, however, exceedingly difficult to place the facts of such cases in a point of view such as not to be open to challenge. I have remarked to you, in last lecture, that pathological anatomy, from its very nature, teaches us but one side of the case. At all events, it teaches us the fatal prognosis of such cases with such an overwhelming number and variety of instances, that, as I have said, the pathological mind receives an inevitable bias thereby. The purely clinical observer, on the other hand, is hampered in his inquiries by the fact that in cases which apparently recover he is rarely, if ever, able to adduce unexceptionable evidence as to the nature of the lesion. And in connection with this there is yet another difficulty, in the case particularly of very young children—namely, that unless through the microscopic evidence of the tubercular bacillus, the line even of anatomical demarcation between tubercular and non-tubercular lesions is very far from being strictly

* Sir Spencer Wells has quite recently told me in conversation of a still more remarkable, and indeed marvellous, fact in his almost unique experience—viz., a case in which the abdomen was opened as a *dernier ressort* for the evacuation of fluid, the peritoneum being found occupied universally by growths altogether resembling cancer ; and in which, notwithstanding, the patient made a good recovery.

defined. In the so-called catarrhal pneumonia, for instance, especially when it occurs in the apices, and when it tends to lobular condensation, and to what I have elsewhere called bronchial abscesses, these often undergo caseation, and are accompanied by glandular enlargements; and under such circumstances it may well be (apart from the bacillus) absolutely impossible to define what is tubercular and what is not. Even in very much older subjects, before Koch's discovery, I have again and again seen cases in which the recognition of tubercular lesions in the dead body was only a matter of inference, and, in some of them, of doubtful inference. It is well known, moreover, that pathologists so generally accurate, so advanced in theory, and so well versed in microscopic morphology as Cohnheim, have entirely repudiated morphological tests as not being final, and have argued that the only perfectly unexceptionable method of demonstrating the tubercular character of a lesion is by inoculation, under selected conditions, in a living animal.

Notwithstanding these grounds for scepticism, I have for many years held that tubercular peritonitis, speaking of it as including, if not accurately corresponding with, chronic peritonitis generally, ought not to be regarded as having the gloomy prognosis commonly assigned to it; or at all events, that cases which are clinically undistinguishable from tubercular peritonitis do get well, or apparently well. A statement of this kind must necessarily be made with reservations, but it appears to me to follow, as a necessary corollary, from facts observed at different times over a long series of years under the following heads:—

1. Cases manifestly tubercular—*i. e.*, with what are usually considered tubercular lesions in more than one organ or part, and often also with hereditary antecedents pointing in the same direction, in whom abdominal affections, characterised

by all the signs of chronic peritonitis, have undergone apparent cure for the time being.

2. Cases similar in all respects, in which a variety of tubercular lesions in various organs associated with chronic peritonitis have resulted in death; but the death has not been from the peritonitis, but in the ordinary course of pulmonary or of cerebral tubercle.

3. Cases of this kind, in which the progress of chronic peritonitis has been carefully watched during life, its symptoms passing into abeyance for a longer or shorter period, while the progress of other lesions was equally watched over a period of months or years, and in one or two instances the previous existence of old tubercular peritonitis was established on a *post-mortem* examination.

From such facts, it has appeared to me not unreasonable to infer that tubercular peritonitis, or some disease clinically very exactly resembling it, may occasionally undergo a practically complete cure, and may also, not unfrequently, undergo temporary or partial improvement to such an extent as to form an apparent cure. The cases which I have narrated to you, already published in the *Medical Times and Gazette,** and all occurring in the course of one summer and autumn in the Western Infirmary, are more or less illustrative of all these positions; and, but that I am unwilling to weary you, I could adduce a number of additional instances. At the same time, I feel bound to admit that there is still a *hiatus* in our knowledge of the subject; and this *hiatus* will not be filled up until pathological anatomy shall have completed the other aspect of its own biassed information, by show-

* See, also, a very interesting lecture by Dr. Gee (*Lancet*, 1st January, 1881), which only became known to me after the cases here referred to had been published; and, indeed, since the present course of lectures was delivered.

ing us, in detail, what are the changes undergone by cases of the kind referred to, in their progress towards healing. One case, such as that of Sir Spencer Wells, if submitted to *post-mortem* examination, the patient having died at a remote period from some other cause than peritoneal tubercle, would probably do much towards solving the difficulty.

In speaking of peritonitis of pelvic origin, I have alluded to the apparently extraordinary character of the recoveries frequently observed in the puerperal form of the disease. One case in particular (*Medical Times and Gazette*, 5th July, 1884, p. 5), observed and placed on record both by Dr. Matthews Duncan and myself, showed that an abdominal infiltration, perimetric in origin, but causing dense impaction with dull percussion of the whole abdomen from the umbilicus to the hypogastrium and flanks, accompanied, moreover, by fever extending over many weeks continuously, and closely resembling, in some of its characters, sub-acute tubercular fever, was followed in the end by a recovery so complete as to allow, not only of life being prolonged and health maintained for years, but also of renewed child-bearing. I do not maintain this to be a tubercular case, being, indeed, strongly persuaded of the contrary; but as regards the local changes, and looking to the remarkably complete resolution implied in the restoration of function above alluded to, the case may well take rank among the marvels of clinical experience, even although we may be compelled to admit that its pathological record has not been completely worked out, and is even, to a certain extent, inexplicable. In some such category I have been accustomed to place the clinical evidence, to my mind unassailable in fact, as to the cure, or apparent cure, of chronic, and even of tubercular, peritonitis.

As to the cure of mesenteric glandular disease, there is,

even from the pathological point of view, no reason to doubt. The conversion of caseous and scrofulous glands into masses of calcareous deposit is so well established as to require no further proof. The only question is how far the detriment to function, in any particular case, may be inconsistent with adequate nutrition, and therefore with prolonged life. On this point, it seems only necessary to remark that large masses of these glands in the mesentery, converted completely into a calcareous condition such as I show you here, not only demonstrate the healing process pathologically, but indicate that in some way or other there is compensation for the loss of function, whereby alone life could have been prolonged so as to permit of the retrograde metamorphosis necessary for pathological healing. I shall, therefore, have no hesitation in assuming that *tabes mesenterica*, in every aspect of it, is susceptible of curative processes, the precise nature of which may be admitted to be imperfectly known; and, on this assumption, I shall treat, in my concluding lecture, of the preventive, hygienic, and curative treatment of this disease.

Lecture IV.—Practical Considerations; Prevention and Cure.

Diagnosis with a view to Prognosis—twofold division of cases, of more and of less favourable prognosis. Carreau indolent and Carreau inflammatoire. The earliest period of infancy; Dentition and Weaning. The "weaning brash," or Atrophia ablactatorum. Entero-colitis—its relation to climate and locality. Food of early infancy—hygienic details. Diet and regimen, &c., of older children. Therapeutics.

I WILL now attempt to trace out for you such a narrative of the origin and progress of these disorders of the abdomen, whether in children or in adults, as may guide you with respect to their prognosis, and also their management in detail. Two distinct classes of cases may be adverted to:—

In the *first*, notably illustrated by the case of L. C. (see Appendix) the abdominal swelling occurs as either absolutely or very nearly the first symptom of ill health; it may be without appreciable pain or uneasiness; or, at least, with pain so slight as to be practically overlooked. There may be no diarrhœa at any time; there may even be constipation. The tongue may be clean; the appetite fairly good. It is, however, not usual for the disease to last long under such circumstances, without a notable change in these respects, and without an appreciable degree of emaciation. The temperatures may present every possible degree of variety; they may even be entirely undisturbed throughout. More usually, however, the evening temperatures on the whole, are relatively elevated as compared with the morning, be it ever so

slightly. In some cases, there is a pretty regular diurnal oscillation of one or two degrees or more, the evening temperatures being to this extent in excess of the morning, which may or may not slightly exceed the normal. In such cases, and when the strength and bodily condition are well maintained, the prognosis may be pronounced relatively good, even when the physical lesions in the abdomen are quite well defined. And if under these circumstances evidences of moderate fluid effusion occur, followed by increased resistance and dull percussion over the middle abdomen, still there is no reason to despair of effecting at all events a practical, if not an absolute, cure. It is quite unusual for the amount of fluid to become such as to raise any question of paracentesis. Indeed I can only remember, in eleven years' experience in the Western Infirmary, two or three cases where the question of surgical interference presented itself in a practical shape. In two of these, at least, the operation appeared to be urgently demanded; but the results were not encouraging; and, although I am far from believing that the condition of these patients was made worse by paracentesis, yet, on the whole, the effect upon my mind was to produce an impression that, unless absolutely demanded by urgency, it is better to abstain from the use of the trochar. In the majority of cases the fluid effusion spontaneously subsides within a reasonable period, and nothing is gained by the attempt to hurry the process.

In the *second* class of cases the abdominal distention has either been preceded or is accompanied by other disorders, the significance and the gravity of which may vary almost indefinitely. In some cases there is pretty severe pain, with tenderness on pressure, such, in fact, as to suggest a sub-acute, if not an acute, attack of peritonitis. In others there is diarrhœa more or less long continued. In some, but by no means

in a large proportion, there is vomiting; in the majority of
cases, however, vomiting is either not present, or is altogether
occasional and accidental. The physical alterations in this
group of cases, to percussion and palpation, may differ but
little from the former; the prognosis, however, is distinctly
less favourable. And, in not a few of these less favourable
instances, the temperatures are persistently elevated; diarrhœa
is more or less constant, and accompanied by pain; the tongue
becomes coated, or perhaps still oftener red, with markedly
injected papillæ; and the emaciation is considerable from the
first. The essential difference between the two groups as here
briefly indicated is, that in the former there may be no disease
of the mucous membrane, while in the latter ulceration, more
or less considerable, of the glands of Peyer, or of the solitary
glands of the ileum and colon, has preceded or accompanied
the abdominal swelling. The presence or absence of mesen-
teric tubercles makes very little difference as regards the
symptoms. In the majority of the latter group, and in
almost all the fatal cases, they are present; but so inextri-
cably involved in the other lesions, whether of the mucous
or serous membrane, or both, as to lead to the probable
inference that they are of quite secondary importance. At
all events, there is no reasonable ground for adopting the
rule of many of the French authorities, of confining the use
of the name *carreau* to the cases of mesenteric glandular
tubercle. It is not improbable, however, that a distinction
insisted on by M. Guersent, and, on the other hand, disallowed
by Rilliet and Barthez, may be considered as corresponding
generally, though by no means in every detail, with the
symptomatic differences of these two groups. "Mesenteric
tubercles," writes M. Guersent,* "present themselves under
two very distinct aspects, which must necessarily have a

* *Loc. cit.*, pp. 316, 317.

very different influence on the abdominal organs, and consequently on the vital phenomena thence arising. The tubercles may be without any kind of inflammation of the surrounding parts, or they may be accompanied by a true phlegmasia of the glands, and sometimes even of the intestinal mucous membrane in the neighbourhood of the diseased glands. In the first case, they (*i. e.*, the tubercles) are indolent; in the second, they are ordinarily painful." And he proceeds to show, accordingly, that between the *carreau indolent* and the *carreau inflammatoire* there is all the difference between a disease with hardly any distinctive symptoms and one having the ordinary and, to a certain extent, well known symptomatic characters of the *tabes mesenterica.*

The error here is, if I mistake not, what I have already pointed out in a preceding lecture—*i. e.*, that M. Guersent, while admitting in detail that mesenteric tubercles have no distinctive symptoms at all, still adheres, most illogically, to the old tradition which identifies them exclusively with the *carreau,* and considers all other lesions as purely accidental. MM. Rilliet and Barthez, following in the same track of tradition, but describing, with the greatest care, the anatomical condition of the diseased mesenteric glands, maintain (no doubt quite correctly from this point of view) that it "seems impossible to find, in the pathological anatomy, a point of departure for the division of the *carreau* into two species—painful and indolent. Pathological anatomy," they remark further, "does not justify this manner of looking at the subject, and symptomatology is still more opposed to it."* But by separating entirely, in two distinct chapters (xv and xvii), the tuberculisation of the peritoneum (*phthisie péritonéale*) and the tuberculisation of the mesenteric glands

* Vol. iii (2nd edition), page 810.

(*carreau*), MM. Rilliet and Barthez have, in effect, thrown
away the opportunity of presenting a complete nosological
picture of disorders which are so constantly seen in com-
bination that it is practically impossible to dissever them.
The real basis of the distinction of the *carreau* into two
varieties is to be found, if at all, in the difference above
mentioned.

The clinical distinction between the two classes or groups
of cases above described (which, however, must be considered
as passing into each other by all sorts of intermediate
gradations) is sufficiently manifest. In the former, the
prognosis is relatively good; but, on the other hand, it is to
this group that the remarks chiefly apply which I formerly
made to you as regards the difficulty of defining their strict
pathological position, and especially in determining how far
they belong, or not, to the tubercular class. All that can be
affirmed with certainty in the meantime is that such cases
occasionally, and by no means infrequently, get apparently
well. And, moreover, their antecedents, hereditary and
other, are often such as to justify the hope that at least
in some of them the cure may be permanent. On the
other hand, much of what I have to say as regards the
preventive, hygienic, dietetic management of such cases,
applies less to this group than to the other and more formid-
able one; because a disease emerging, as it were, directly out
of a state of apparently good health scarcely admits of the
application of such anticipatory treatment. Such cases may
perhaps belong to the variety described by Bauer * as *chronic
diffuse peritonitis*, in its variety of *latent general peritonitis*
resembling ascites; but, if so, the information before us as
yet gives no insight into its causes, such as would suggest any
practical distinction of it from the corresponding class of

* *Loc. cit.*, pp. 316, 317, *et seq.*

tubercular cases. "The symptoms which are present in the beginning of the disease (according to Bauer), are, as a rule, very trivial, anything the patient complains of being general and undefined. Dull pains occur in the abdomen, which are increased on pressure and on bending of the body. The patient's general health is out of order; he feels tired, and his appetite is bad. Going up stairs, especially, is frequently the source of an unpleasant sensation in the abdomen. Months may intervene between these primary, scarcely heeded, symptoms and the complete development of the disease; in other cases this stage, which Galvagni describes as prodromal, is shorter." . . . Even in the stage of effusion, "the phenomena are in no way very urgent, and are such as might, for the most part, be quite as fully produced in simple mechanical ascites. . . . There is something remarkable in the insignificance of the pain in many, and the complete absence of it in some few cases of chronic peritonitis. The temperature is often found to be quite normal; in other cases there occur evening exacerbations of a moderate degree. The pulse, on the other hand, is almost always quickened."* It is needless to point out the close resemblance between Bauer's description of what he regards as a very rare disease, and that with which we are at present concerned. Upon the whole, without excluding from view in further remarks the first group of cases, it is the second that we must keep chiefly in view in all that concerns treatment, and especially prophylaxis; it being understood, however, that the principles of management applicable to the second group are also to be kept in view in the first as far as may be.

If an infant, or a young child at any age, appears to be out of health, or emaciating, or disturbed in any way as regards

* Ziemssen's *Cyclopædia* (American Translation), vol. viii, p. 299.

the abdomen, it will always be proper to suspect, at least, the beginnings of this disorder. MM. Rilliet and Barthez, like almost all the other writers on the diseases of children, insist on the differential diagnosis between simple enlargement of the abdomen and that of tubercular peritonitis. And Guersent, as we have seen, has also described the enlarged abdomen of debilitated or rickety infants as being quite distinct from that of mesenteric disease, or *carreau*. The distinction may be admitted as a pathological fact, and yet some of the grounds of the diagnosis cannot be accepted without demur. "The consideration of the age alone," say MM. Rilliet and Barthez, "would suffice to prevent mistake: peritoneal phthisis being extremely rare among young infants, while, on the contrary, rickety distention is frequent at this period of life" (page 791). A similar remark is made by Guersent as regards mesenteric disease. But these statements are scarcely borne out by the facts; for in the article on causes (p. 794), MM. Rilliet and Barthez adduce no fewer than 11 fatal cases of tubercular peritonitis as occurring between 1 and $2\frac{1}{2}$ years, as against 26 between 3 and $5\frac{1}{2}$ years; 40 between 6 and $10\frac{1}{2}$ years; and 9 between 11 and 15 years; thus showing that the conditions which dominate tubercular peritonitis, if not the actual fully developed disease, must, from the practical point of view, be reckoned with as in operation from the very earliest ages. For it is hardly necessary to point out that the 11 infants who died within the first three years of life, and the $11 + 26 = 37$ in whom the disease reached its fatal term within the first six years, were in all probability subject to the causes of it from a very early age; if not from birth, at least from the commencement of dentition, and therefore quite within the period when no practical distinction can be established on this ground between the rickety and tubercular forms of enlargement. Moreover, in seeking to lay down

prophylactic rules it would be impossible, or at least would be highly inexpedient, to limit these to the developed disease. Statistics founded on fatal cases are therefore only applicable when we give due consideration to the fact that these fatal issues are the sequel and last extremity of a more or less protracted course of impaired health, the first stages of which it is very desirable, if possible, to intercept. The same remarks apply to the statistics by Rilliet and Barthez of mesenteric tubercles (*loc. cit.*, p. 820). These were found in 27 cases at from 1 to 2½ years, as against 41 cases from 3 to 5½ years; 55 cases from 6 to 10½ years; and 21 cases from 11 to 15 years. Here again, therefore, although the greatest predominence of fatal results is between 6 and 10½ years, the numbers given at earlier ages necessarily carry us back to the very earliest infancy, if we are to entertain at all the idea of prophylaxis. And it is further to be remarked that, at all the ages given, but much more at the earlier ages, the numerical preponderance of the male sex is very notable, amounting to about double in the very earliest ages, and from this to about a third greater in numbers as childhood advances. I have not found any corresponding statistics of the rickety distention of the abdomen, for the obvious reason that this is rarely fatal; but it is generally admitted that rickets, while occasionally congenital, and not unfrequently developed during the first six or twelve months of life, attains its greatest frequency, and possibly its highest active development, during the second year, or, speaking broadly, during the progress of the first dentition, while the authorities differ widely as to its relative frequency in the two sexes.* It is, therefore, obviously impossible to found on mere considerations of age and sex in establishing such a differential diagnosis as shall be of practical utility in the early stages of either disease.

* Ziemssen's *Cyclopædia* (Senator), vol. xvi, pp. 172, 173.

This point I regard as one of great importance ; because it has been usual to assume (in my opinion without any sufficient foundation) that all enlargements of the abdomen, and most of the catarrhal affections of the mucous membrane occurring before or during the first dentition, are to be taken as non-tubercular; and even that there is a kind of antagonism, or mutually exclusive element of difference, as between the rickety and the tubercular constitution. Thus, M. Guersent declares that rickety infants are very rarely the subjects of mesenteric disease or *carreau*. The statistics of MM. Rilliet and Barthez are entirely opposed to this assertion. I am strongly persuaded, on the contrary, that *rickety infants who die* would, like other infants, be found in a large proportion tuberculous. It is sufficiently obvious that those who recover, or who present at more advanced ages the permanent rickety deformities, are precisely those who may be said to have escaped death *because* they were non-tuberculous, or, at all events, not tuberculous in a high degree.

Whenever, accordingly, in a very young child there is a manifest disturbance of health affecting the general nutrition, especially if this is associated with diarrhœal disease, or with colicky pains in the abdomen and habitual feverishness, if not explained by any accidental cause, or by any endemic or epidemic condition generally prevailing, it is, at least, safe as well as expedient to adopt measures founded on the presumption that such manifestations may be the harbingers of organic changes which may issue in *tabes˜ mesenterica*, or in some of its congeners. In the well known essay of Dr. Cheyne on the Atrophia Ablactatorum, or "Weaning Brash," * there is not, indeed, a well ordered account of the pathological anatomy, nor is there any clear recognition of tubercular

* *Essays on the Diseases of Children, with Cases and Dissections.* Essay II. By John Cheyne, M.D. Edinburgh, 1802.

disease concurring with the lesions described; which, indeed, the author's theory leads him to associate more with hepatic derangement than with the changes in the mucous membrane of the intestines. But in one at least of his dissections, Dr. Cheyne has especially figured "the glands in the root of the mesentery much enlarged" (page 49). This child was under 13 months old; was weaned at 11 months, and about a fortnight after weaning, diarrhœa came on. The fatal issue was due to convulsions; the purging having ceased, owing to the administration of remedies. And the author remarks that "the original disease had by no means arrived at so great a height as I have seen it. The emaciation was not so great as usual, nor the purging nor derangement in the alimentary canal so determined" (p. 47). It is quite obvious that the disease in this case was rather acute, and, in all probability, was not definitely tubercular. This may, indeed, be the usual fact in cases of this kind; and yet it is surely reasonable, in view of the facts stated above, to argue that when, from hereditary causes or otherwise, young children are predisposed to tuberculous forms of disease, the first foundations of such diseases may often have been laid at the time and in the manner here set forth.*   In like manner

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* "The mesenteric glands are enlarged," writes Dr. Cheyne, in giving the results of his general experience in fatal cases of the "weaning brash;" "nay, in some instances, inflamed. May not this proceed from the acrid nature of the alimentary matter to which their absorbing mouths are exposed? May not the *tabes mesenterica* often arise in this way?" The remarks which follow, at p. 28 (note), show conclusively that, without having definitely adopted a tubercular pathology of these disorders, and having, moreover, peculiar and individual views as to the relation of the biliary secretion to the *atrophia ablactatorum*, Dr. Cheyne was, nevertheless, fully impressed with the probable pathological continuity of the more acute disorders of early infancy described in this essay, with the more chronic and scrofulous ones of childhood generally. Case III, intended to demonstrate this, is too long to quote here, but may be referred to at pp. 28-30.

it is difficult to believe that the various forms of diarrhœal disease in young infants, especially those that are characterised as *inflammatory diarrhœa* or *entero-colitis*, are not more or less closely associated as causes, if not as coincidences, with the derangements of health that, at somewhat more advanced periods of childhood, bring about deaths that are frankly tubercular. It is not necessary, in order to maintain this position, that we should assume the existence of tubercle as a demonstrable fact in many of the fatal instances of these early acute forms of diarrhœal disease. No doubt, innumerable cases of simple diarrhœa, of epidemic diarrhœa, or of cholera infantum, and of entero-colitis, attended by destructive lesions, chiefly of the large intestine, may occur altogether apart from tubercle; and yet, as we have seen, abdominal tubercle is very far from being unknown in early infancy. Scarcely any one doubts the influence of certain acute fevers in arousing the latent proclivities to tubercular and scrofulous diseases of the chest—*e. g.*, measles, hooping-cough, and epidemic influenza. Why, then, should it be doubtful that the acute and sub-acute disorders of the intestinal canal which occur during, or even previous to, the first dentition, may reasonably be supposed to have a like influence as regards abdominal tuberculosis? In the valuable chapter on entero-colitis in Meigs and Pepper's *Practical Treatise on the Diseases of Children*, many interesting details will be found as to the lesions in question, and their association with climatic and epidemic conditions. It will be sufficient for my purpose to refer you to this easily accessible work.

It follows, then, that in all cases in which, from hereditary antecedents, or from delicacy of constitution acquired soon after birth, a child may be reasonably suspected of a tubercular predisposition, the utmost care should be taken

that, if possible, during the specially perilous season of weaning, and during the whole progress of the first dentition, nothing should be done to bring into activity the latent defects of constitutional habit which might afterwards assume the form of some of the diseases with which we are now concerned. The food of the infant, its clothing, its surroundings as regards pure air, &c., ought to be the subject of the most anxious consideration. Dr. Meigs points out, by means of a very striking table obtained in Philadelphia, and presenting, in an intensified form, data which are equally well known in connection with the diarrhœal death-rates of this country, that, in July and August, and in a less degree throughout the summer and autumn months, diarrhœal diseases assume a prominence amounting in some cases to not much short of a hundred times as many deaths as they cause in some of the colder months: indeed, that a mean temperature above 70° is absolutely certain to bring these diseases in its train, and to a greater degree in proportion as the temperature is higher. Drs. Meigs and Pepper, therefore, recommend that, under these circumstances, children who are suffering from diarrhœal disease should be removed as much as possible from the town into healthy and cool stations during the whole of these hot months. And this recommendation is powerfully aided by the parallel they are able to draw between the diarrhœal diseases of infants and those observed among adult soldiers during the American war, when it was found absolutely necessary, and in fact the only effective method of treatment, to remove the soldiers affected with disease from their unwholesome surroundings in the South, and to send them to hospitals specially provided in healthy localities in the Northern States. It has also been regarded as highly expedient, in most cases, to avoid weaning a delicate child at the dangerous

season of the year; * or, if weaning is absolutely necessary under such circumstances, to take extra pains to secure proper food which has not undergone decomposition, or otherwise been contaminated by noxious effluvia. The elaborate and well considered chapter upon the food of infancy in the excellent work already referred to † may be recommended to all of you, as also the quite recently published little work of Dr. Arthur V. Meigs on *Milk Analysis and Infant Feeding*, which may be said to contain the latest data, at once scientific and practical, upon the chief food of infancy. At page 410 of Meigs and Pepper's work will be also found certain interesting statements regarding the putrescibility of milk under various atmospheric conditions, and the probability of bacterial infection as a cause of diarrhœal disease, which, although very important, I have no time to enlarge upon. Two "broad generalisations," however, at page 418, may be here cited :—

"1st. An unhealthy food—one incompetent to furnish to the body what it needs for the purpose of nutrition, as farinaceous food or unhealthy milk, is sure to produce the disorder we are considering, no matter how favourable may

* This very important recommendation extends back to the time of Dr. Cheyne, and is strongly corroborated in the excellent and thoroughly practical American work of Dewees, when remarking on Cheyne's statement, that "delicate children should in the autumn season be kept a month or two longer at the breast than might be thought necessary at any other, rather than be exposed to the aches and hazards which never fail to accompany this distemper" (the "weaning brash"). On this passage Dr. Dewees observes :—"This advice is in strict conformity to the usage of this country : it is so well understood by everybody here that a physician is rarely consulted upon its propriety. During the months of June, July, August, and frequently September, weaning is never performed as a matter of choice, let the age of the child be never so proper, unless it has completed the cutting of its first set of teeth. The dread of our females in this country is the " second summer."—Dewees *On the Physical and Medical Treatment of Children* (1826), p. 331.

† Meigs and Pepper, seventh edition, 1883, page 301.

be the circumstances, in all other respects, in which the child is placed.

"2nd. The best breast milk in the world, or the most correct artificial diet, will not save a child from this disorder who is located in an ill ventilated house in a dirty and filthy quarter of a large city during hot weather."

It would be difficult to place the whole subject of the hygiene of early infancy in a clearer light, or in a more comprehensive form of statement than this.

But we have seen above that, in not a few instances of disease resembling *tabes mesenterica*, it arises without much notable diarrhœa in the first instance, and certainly not as the sequence of obviously epidemic or endemic conditions. In such cases, as in all cases, it is of course necessary to treat the disease actually existing, either without reference to its antecedents, or with only such reference to these as is possible under the circumstances. Certain points may here be stated which are of quite general application. Warm underclothing and dress loosely fitting to the body should be usually adopted, unless the temperature is very high, when even the lightest and thinnest flannel is borne with difficulty, which can rarely happen in this country. But when thus protected the child should not be too closely confined to bed, or even to the house in good summer weather; as a rule, unless, indeed, the intensity of the fever, or extreme bodily weakness, or the frequency of the discharges make this imperative. Bathing in water moderately warm, and, in the great majority of cases, inunction of the abdomen with cocoa nut oil (preferable, as a rule, to cod liver oil which, however, is also often recommended for inunction), will be found very advantageous. The temperature of the bath may vary, according to the state of weakness or chilliness of the child, from 60° or 70° up to 90° or 95°. It is rare, comparatively speaking, for the mere intensity or con-

tinuity of the pyrexia to require the absolutely cold bath as an antithermic agent; but no doubt the same principles as would hold in typhoid fever may be considered as applying to the disorder now in question, under the like circumstances. My colleague, Prof. M'Call Anderson, has published several cases in which the application of ice-cloths to the abdomen as a substitute for the cold bath has, apparently, proved useful under very unpromising circumstances, both in tubercular peritonitis and in acute phthisis. The method of application is as follows :—" The night dress is pulled well up over the chest so as to avoid any possibility of its being wet, and, for a similar reason, a folded blanket is placed across the bed under the patient's body. The usual bed-clothes are arranged so that they reach up to the lower part of the chest only, which latter is covered with a separate blanket in order to prevent unnecessary exposure while the iced cloths are being changed. Two pieces of flannel are employed in the process, each being sufficiently large, when folded into four layers, to cover the whole of the front and sides of the abdomen. One of these, wrung out of iced water, is applied, while the other is left in a large basin filled with iced water at the side of the bed. The pieces of flannel are changed every minute, or so often that they still feel cold when they are removed. The changing of the flannel, especially if two persons are in attendance, one to remove the bed-clothes and the flannel, the other to apply the piece which is freshly iced, can be effected with great ease and rapidity, and without exposing the patient to any injurious extent, if the preliminary arrangement of the bed-clothes is made in the way I have indicated. I have thought it right to mention these apparently trivial details because I have often seen the process carried out in such a way as to be perfectly futile, and because I have been frequently interrogated on the subject. But I think it right to

add that, in the treatment of acute phthisis, I do not wish to
lay too much stress upon the value of iced cloths by them-
selves, but to attribute the success of the treatment to the
combination of measures employed. Of course the same
precautions must be taken in the use of iced cloths as in
the employment of the cold bath, and the cloths must
be at once removed if there is any tendency to coldness or
collapse." *   This method is certainly convenient; but it
may reasonably be doubted, I think, whether too much is
not claimed for it. According to all my experience hitherto,
the use of cold externally applied as an antithermic relieves
or palliates, it is true, the individual symptoms for the time
being, but fails entirely in arresting, or even in most cases
in subduing, the essential morbid process. I am not here
arguing the case of hyper-pyrexia, or even of typhoid fever
or pneumonia with temperatures continuously maintained
for days at such a level as must necessarily prove extremely
debilitating. In the affections we are now considering, this
state of the temperatures is rarely present; and, while not
discountenancing the use of external cold, or even ice, in
such cases as may appear to require it, I am of opinion
that the majority of cases do equally well or better with-
out this. I have also frequently used quinine, and of late
years antipyrine, in these cases. The latter remedy has, on
the whole, answered well so far as bringing down the tem-
perature is concerned; but its action is very fleeting, and it
is extremely doubtful whether more is not lost than gained
by the repeated introduction into the stomach of substances
which are non-nutritious, and which, on the whole, tend
to disturb digestion. It should never be forgotten that
the paramount object of treatment is to preserve as far as
possible the remains of the digestive and assimilative powers;

* *Clinical Lectures, &c. Op. cit.*, pp. 35, 36.

and large doses of quinine, or antipyrine, or any other substance of this nature, are scarcely consistent with following out a regimen of this kind.

In dieting patients beyond the age of the first dentition, it has been a not uncommon practice on the Continent to employ raw meat. This, as a food for infants deprived of their mother's milk, or for weaned children, was recommended as long ago as 1840 by Weisse, of St. Petersburg, and has been highly praised by Trousseau and other eminent authorities on the Continent. The meat is to be cut very fine, pounded in a mortar, and strained through a sieve or cullender. The pulp, thus separated from the cellular tissue of the texture of the meat, may be rolled into small balls in salt or powdered sugar. This, given in very small quantities at first, and gradually increased, is said to arrest diarrhœal diseases, and to bring about restoration of health. Generally speaking, so far as my experience extends, it may be said that Continental physicians are far more inclined than those in this country to advocate a diet of meat even for very young children; and this, in circumstances under which few physicians among us would consider it suitable.* I have no doubt

* The controversy, however, is by no means one of yesterday, or even one between British and Continental physicians, as I find from the following note in Dewees' celebrated *Treatise on the Physical and Medical Management of Children*, 1826. The author, as representing American opinion in his day, is criticising (with much appreciation, however) the essay of Dr. Cheyne, cited above :—" We were not a little surprised," he writes, " to find the following observation of Dr. Cheyne, on the subject of proper diet for children—namely, that 'an animal diet produces less irritation than one which is solely composed of vegetable matter' (p. 36). It is certainly contrary to our experience, and we had like to have said to that of everybody else. We have even found reduced milk, rennet whey, gum arabic water, thin sago, tapioca, or arrowroot, vastly more proper in all the complaints of the bowels, where nourishment may be safely permitted, than any animal juice, however much diluted. But we are persuaded that during the acute form of the disease, where purging is necessary, where there is a pain and a fever, the less the child

that raw meat is under such circumstances found more digestible, and therefore less injurious, than some other forms of it; but, in one very striking case, under the care of Dr. Goodchild, of Bordighera, I had occasion to follow, with the interest of an intimate friend as well as a medical man, all the details of treatment under a variety of medical authorities in Paris and elsewhere for years. The case is one in which a young extremely delicate boy, now about eleven years of age, had been for a very long time subject to diarrhœal and digestive disturbances from the very slightest errors of diet, and not unfrequently with threatenings either of chronic peritonitis or, at all events, of some disorder involving considerable swelling of the abdomen, with foul breath, enlarged papillæ of the tongue, and at times very decided feverishness. The general impression left on my mind is, that the meat diet allowed, or enjoined, under these circumstances, by foreign physicians, especially in Paris (where his parents reside) has had again and again to be abandoned in favour of a diet either absolutely, or almost absolutely, composed of milk, when the symptoms were most threatening; and, that it has been only with the greatest reserve that a return even to the lightest farinaceous articles along with milk could ever be allowed, and to meat not at all. Such, at all events, was the result in Dr. Goodchild's mind from the most careful and

takes into its stomach the better. . . . The only thing proper at such times is a little of the mother's milk, if the child be not weaned, or if the milk is known to agree with it, or the occasional use of thin gum arabic water." But Dr. Dewees seems to have forgotten for the moment that a diet of milk and rennet whey, &c., is *not* "solely composed of vegetable matter," such as is denounced by Cheyne. Dr. Dewees' own opinion is very much in accordance with that of his legitimate successors in Philadelphia in the present day. "During the continuance of this disease, we strictly forbid animal food or juices under any form. If the child be at the breast, let it be confined to it, if the mother has a sufficient supply; if she has not, let it be in part supported by reduced milk and a little sugar."

detailed watching of this interesting case.* Still, in some cases, very moderate quantities of meat, or of meat juice, or still better, of meat-peptones, such as Carnrick's, or the peptonised jelly prepared by Benger, or the meat juice of Valentine, may be found useful, but only in older children.

It is very doubtful how far counter-irritation, in any extended sense of the word, should be employed, even in cases where there are physical alterations attended with considerable pain, and symptoms of inflammation locally. The addition of iodine, or of iodine ointment, to the ointment of cocoa nut oil above mentioned, is a constant resource; and, as it can be employed in every proportion, answers every purpose without the necessity of breaking the skin. Hot linseed meal poultices give relief when there is much pain, but if extended over the whole abdomen are unpleasantly heavy, and tend to produce sweating in excess. Dr. Eustace Smith recommends, accordingly, smearing the surface with a salve composed of extract of belladonna and glycerine in equal proportions, and covering this with a thick layer of cotton wool. A very old external application, recommended in the last century by Russell, in what he called "glandular tabes," is sea water; and perhaps it might be re-introduced with advantage in the form of Tidman's sea salt, used as a very strong brine warmed, and applied locally by means of flannels or spongiopiline; but I have no experience of this. Some eminent authorities recommend the application of mercurial ointment or ointments of the oleate of mercury; but this too has not entered into my practice. Opium, where either pain or intestinal

* The very last intelligence I have of this child is that he is now, after years of most anxious watching, and several winters and springs spent on the Riviera, so far recovered as to be able to vary his diet without injury, and practically well and in good bodily condition.

irritation are present to any great degree is, according to my experience, an indispensable remedy, and may be administered with considerable freedom, due regard being had to the age of the child. Bismuth, even in large doses—*i. e.*, up to 20 or 30 grains, is sometimes very useful when there is obstinate diarrhœa. But, of course, these and all other like remedies should be limited to the critical conditions which precisely demand them. Under opposite conditions, when pain is not great and when the bowels are quiescent, cod liver oil is a valuable medicine, and when digestion is feeble, it may be aided by the pancreatic emulsion of Savory & Moore, or the *liquor pancreaticus* of Benger, which may also be given independently with good effect. Cream may be given in place of cod liver oil, or the malted cream lately introduced by Lœflund, which, however, is often found too luscious, as are, indeed, the most of the malt extracts manufactured in this country. In some cases a very little stimulant, brandy or whisky being the best, from a few drops to a teaspoonful at a time, appears to aid the digestion, restrain diarrhœa, and conduce greatly to the comfort of the patient; but it ought by no means to be converted into a routine treatment.

By these or similar means I have been led to think that the dark prognosis of what has been termed *tabes mesenterica*, and the still gloomier picture given in systematic works of tubercular and chronic peritonitis, may in some, even not a few, cases be relieved of its darkest shades. What has chiefly interfered with this conviction becoming more general than we find it among highly educated and experienced physicians, is the unconscious bias to which I have already alluded, derived from pathological anatomy. Having suffered from this bias myself in the earlier stages of my career, and only emerged from it very gradually

through the clinical experiences of more than a quarter of a century, it has seemed to me a not superfluous task, even now, to lay before you the sources of the error as they appear to my own mind, and to call upon a younger generation for still further researches with respect to it. You will already have apprehended that I have no specific, and scarcely anything that can be called novel, to set forth in the way of treatment; but even well recognised facts and principles may operate in a different manner on minds prepared to receive them by sound pathological and clinical experience, as compared with those that are misled by names, and by the overweening influence of great pathological authorities.

[No attempt has been made in the preceding Lectures to discuss the surgical treatment of tubercular peritonitis by free opening of the abdomen, washing out of the effused products, and subsequent drainage. The debate in the Clinical Society of London, which appears while these pages are going to press (*Lancet*, 5th Nov., 1887, p. 917), presents many interesting topics for consideration, and I should not be indisposed to adopt this plan of treatment in certain cases; but, notwithstanding the marvellous success claimed on behalf of some operators, it appears not improbable, in the light of the preceding inquiry, that not only have operations been resorted to unnecessarily, where a spontaneous recovery might reasonably have been expected; but also that errors of diagnosis have had a considerable share (as in Sir Spencer Wells' case, referred to above), in leading up to operative procedure. At all events it may be affirmed without hesitation, that in any future discussions on the subject it will be necessary to give effect, in some degree, to the views here submitted as to the prognosis apart from operation.]

APPENDIX.

CASES IN ILLUSTRATION, WITH REMARKS.

THE four cases which follow were published in the *Medical Times and Gazette* of August, September, and October, 1885, as following a series of observations on acute peritonitis in the preceding year; and it was intended to have followed up the subject in succeeding papers, to somewhat the same effect as in the preceding Lectures; only preserving more than has been done in them the form of clinical instruction addressed to students of medicine. The *Medical Times and Gazette*, however, was brought to an abrupt conclusion at the close of 1885, and hence it became necessary to reprint these cases in their present connection, as a part of the evidence of what has been submitted in a more doctrinal form. Each case, from this point of view, may be regarded as a type from which a definite lesson can be drawn. And it has been considered better to present a few facts, very carefully observed, than to multiply instances. With the exception of Case IV, none of the others can be said to be nearly unique in the experience of the author.

CASE I. (*Medical Times and Gazette*, 29th August, 1885, page 273.)—*Peritoneal disease with fluid effusion, in a child; evidences, in all probability, of chronic peritonitis, and, at a later period, of thickening of the great omentum.*

*Symptoms exceedingly mild, with entire absence of organic
complications, and little, if any, fever. Cure apparently
complete.*

Jane M., æt. 8, was admitted to the Western Infirmary
on 10th April, 1885, and dismissed, apparently well, on
14th July. During this three months' residence she was
repeatedly and carefully examined by a considerable number
of persons, and owing to the clinical examinations for the
degree being in progress at the time, she was repeatedly
employed as a typical case—*i. e.*, as illustrating the physical
signs of fluid in the abdominal cavity, with certain peculiari-
ties of local distribution. The description in the *Medical
Times and Gazette* gives, in more detail than is perhaps
necessary here, the progress of the case and the precautions
employed in the diagnosis. It may suffice to record at
present that the child was stated to have been "always
somewhat swollen in the belly;" but that towards the
end of March an increase of tension, with some pain,
caused a degree of alarm. The bowels had been regular,
except during an attack of measles at the beginning of the
year, when they tended to become loose. The appetite was
much diminished, but there was no vomiting. A slightly
rickety condition, soon passing away, at two years of age,
and previously to this whooping-cough, seemed to comprise
the entire morbid history of any consequence; and it may
be mentioned that the circumstance of an elder sister
suffering from what was called "consumption of the bowels,"
had led her parents to look very carefully for the slightest
traces of any similar complaint in this girl.

Within a fortnight from her admission on 10th April, it
was ascertained that the circumference of the abdomen,
evidently too great from the first, had increased from 25
to 28½ inches, and the signs of fluid accumulation, at first

not evident, became such as to leave no doubt whatever, and even to give the characters of what might have been a well marked ascites, were it not that during its whole progress the *levitation* of the intestines in the umbilical region was markedly interfered with. "The inference I drew from the whole facts at the time was that this fluid effusion was not a mere ascites, but some more or less fibrinous exudation which hampered the free movements of the intestinal coils among the fluid, and prevented them from following the physical law which in moderate ascites, or serous dropsy of the peritoneal sac, usually causes them to float up towards the higher levels, according to the position of the body at the time." Palpation also gave slight indications of increased resistance in the umbilical, as com- pared with the sub-umbilical, regions of the abdomen. The abdominal superficial veins, too, were decidedly increased in volume at this date.

Notwithstanding these somewhat ominous physical signs, however, the general condition of this little patient was extremely favourable. There was no acute suffering of any kind. The utmost that could be said was that the child was rather indisposed for play and for active movements, though quite able to sit up without fatigue for several hours each day. The temperatures indicated only slight febricula (max., 100·4° F.) The period most characterised by these slightly abnormal temperatures was from 19th to 26th April; both before and after this period the differences between the morning and evening temperatures were insig- nificant; and after the 1st May no abnormal temperatures at all were noted.

An increase in the body weight of 3½ lbs., concurring with the physical signs above referred to, was discounted as probably due entirely to the fluid accumulation, possibly

even concealing a real loss of tissue weight. The urine, relatively scanty during the period of active effusion, increased from a daily average of 17 ounces to 24 ounces after the close of this period. The lungs and all the organs, so far as they could be investigated with the utmost care throughout the illness, presented no signs of disease. The bowels were regular, and the tongue absolutely clean.

Four days before she was dismissed from the hospital, apparently quite well, a most deliberate and minute survey of her condition, for purposes of clinical instruction, was entered in the journals, and formed the basis of remarks to be found at length in the *Medical Times and Gazette*. The practical result of the whole was that almost every trace of fluid effusion had disappeared, the circumference of the abdomen declining to a certain extent as compared with the maximum above-mentioned, but being, on the other hand, manifestly reinforced by the considerable increase of muscular flesh and fat in the abdominal wall, "so that this child is now only $\frac{7}{8}$th of an inch less in girth than when admitted on the 10th of April with an obviously enlarged abdomen." In other words, with a circumference of 25 inches on admission, the child was quite manifestly too large and distended as regards the abdomen; four days before her dismissal, with a girth of $24\frac{1}{8}$th inches, she was not distended at all, the abdomen being almost, if not quite, in due proportion to the rest of the body. Between these periods the girth, as stated above, had been $28\frac{1}{2}$ inches.

The point of greatest interest, however, in this final examination, was the evidence obtained, on very critical palpation and percussion of the abdomen, tending to show that an appreciable, though slight, amount of thickening of the great omentum still persisted after the disappearance of the fluid, and after the return of the child to apparently

good health. The complete details of this evidence will be found in the *Medical Times and Gazette*. It was considered as not only demanding careful exposition as a clinical lesson in itself, but as entirely corroborating the previous impressions derived from the state of the physical diagnosis during the period of effusion. Taken in connection with the gain in weight (2¾ lbs. since the absorption of the fluid ceased to be a counterpoise to the putting on of flesh), the obviously improved general condition, and the absence of all symptoms of disease, it was not considered to be a state of matters interfering with the reasonable hope of a practical recovery.

This hope was fully justified; for, nearly two years afterwards, I heard of this patient as still practically quite well.

The treatment was exceedingly simple; it consisted of inunction with cod liver oil, the use of extr. hordei, pancreatic emulsion, and careful regulation of the diet. Cold compresses were employed up to 25th April, but were then found to be unnecessary, and the oleaginous application was substituted. Gastric tonics, &c., did not appear to be required at any time.

In reporting this case for publication in a medical journal, my object was that it should, as far as possible, tell its own story, independently of technical nomenclature. But the reader of the preceding pages will have no difficulty, I think, of bringing the case into relation with the general subject of what has been called *tabes mesenterica* or *carreau*. As regards the question of its tubercular origin, the evidence is (happily) wanting. All that can be said is, that in a sister of this little girl the symptoms of a disease in some respects similar had actually been interpreted as those of "consumption of the bowels." The doubt which attaches necessarily to the pathology of the cases which get well,

as remarked in the previous chapters of this memoir, hangs over this case to the full extent. Not so with the following case, in which there was a fatal result, but no *post-mortem* examination.

CASE II. (*Med. Times and Gazette*, 19th September, 1885, p. 389.)—*Typical case of tubercular peritonitis, with co-incident disease of the intestinal mucous membrane, and probably also of the mesenteric glands. Gradual emaciation, with many fluctuations, and ultimately death, with all the features of* tabes mesenterica, *as described.*

Mary Jane S., æt. 10, admitted to the Western Infirmary 13th March, 1885. When this girl was first seen, it was felt to be impossible to handle or disturb her much. Her condition resembled a good deal that of a case of typhoid fever, with marked abdominal complications; it was, in fact, not quite certain that this was not so, although there was no characteristic eruption. There was great tenderness of the whole abdomen, which was much distended, and the superficial veins were very much enlarged. Had it been a case of typhoid fever, I should have been apprehensive of perforation, either actually present or impending. The face was flushed and bedewed with perspiration. The temperature, however, did not exceed 101·6°, and I noticed that she had not the peculiar aspect of acute peritonitis from perforation; nor had she the disposition to draw up the knees and to lie in a constrained position on account of the pain. The physical signs at this time were (so far as they could be gone into safely) very much the same as those in Case I at a somewhat advanced stage of its progress, and this although the whole of the symptomatic phenomena and the urgency were so different in the two cases. There were signs, also, of a mild general bronchitis,

but without any expectoration. The percussion of the chest was normal.

The history was, briefly, as follows:—She had passed for a healthy child up to three weeks before admission. Then began a series of symptoms closely corresponding with those of *tabes mesenterica*, or "consumption of the bowels;" flatulence, with pain, tumid abdomen, diarrhœa, some cough, emaciation, and probably more or less of fever. It was the diarrhœa, gradually increasing in severity, which finally drove her away from school, which she had been attending up to a week before admission. Vomiting had been an early, but by no means a persistent symptom, being brought on, apparently, by some powders prescribed for her. There was nothing to lead to the suspicion of any serious illness previous to the present.

The treatment at once adopted, even before all these particulars were fully ascertained, was by opium in $\frac{1}{3}$ gr. doses, with gr. ij of quinine three times a day. Fomentations were applied to the abdomen, evidently with much relief. On the third day it was possible to make a more detailed examination of the abdomen, and although it was still necessary to use great caution, I was able to demonstrate to several members of the class facts which were at the time roughly indicated in a diagram ; but, as the attempt to reproduce this by a woodcut in the *Medical Times and Gazette* was not very satisfactory, it is omitted in this reprint. Generally speaking, the right side of the abdomen and the umbilical region were almost everywhere more or less dull to percussion, the left side much less so. The gastric region, and some of that of the transverse colon, were normally clear ; and the left iliac and hypogastric regions were at least not notably dull. It was also observed that the dulness was throughout *easily penetrated—i. e.*, by employing a slightly

increased strength of stroke; and this relative dulness on percussion corresponded well with the tactile examination in giving the signs of thickening of the parts overlying the intestines, without fluid effusion; and, in particular, thickening, at least on the right side, of the parts anatomically related to the great omentum. The details of this examination, as it was recorded for clinical purposes on the 16th of March (three days after admission), are commented on at length in the *Medical Times and Gazette*, although, as already stated, the diagram in illustration is so unsatisfactorily brought out as to be of no use to the reader.

The remedies employed from the first very greatly relieved the pain, and also completely controlled the diarrhœa, so much so that two days after this, constipation (evidently caused by the opium) was the predominating condition. The abdominal tension was much reduced; the temperature became normal; pulse 96; tongue nearly clean and natural; so that in four days this girl seemed to have passed from a prospect of immediate death to a state of almost complete convalescence as regards the acute symptoms. Even the details of the physical examination underwent a corresponding modification; and after a short time it was found possible to intermit the opium and quinine, and to substitute very gentle friction of the whole abdomen with cocoa-nut oil, for the fomentations. Under this treatment, for some weeks at least, the progress appeared to be that of an uninterrupted convalescence. In the course of the month of April, however, the improvement came to be regarded as more doubtful, although there was no return of any of the more obviously unfavourable symptoms. On 6th May, she had been losing rather than gaining in weight; the temperatures, which had continued normal up to 16th April, were again more or less disturbed, and in ten days in

succession showed a mean of 98·5° morning, and 100·4° evening, with a maximum of 102°. But a still more patent fact in the direction of an unfavourable change was the existence of manifest signs of a considerable amount of gravitating fluid in the abdomen which, concurring with the above symptoms, certainly appeared to indicate a new period of sub-acute morbid change. During the entire months of May, June, and July, there was a progressive loss of body weight which, although not more than 6 lbs. in all, was not fully accounted for by the absorption of the fluid effusion. All the facts now corresponded with a downward progress, though perhaps not a rapid one; the increasing languor, the occasional pain, the dispirited look and manner, were very striking; although the appetite was well maintained and there was no diarrhœa. At a still more advanced period (in September), although there was no pain at all, and almost no active symptoms of any kind, there was slight febricula (maximum 101·2°) and in this state the case was reported as one probably of tubercular disease, more or less widely distributed in the abdomen, perhaps also in the thorax, so as to include the mesenteric glands with the peritoneum and the mucous membrane. This character the case retained for a month or two longer, when the death of this child was reported to me as having occurred in the East Park Home, without any essentially new symptoms. No opportunity occurred of completing the knowledge of the facts by a *post-mortem* examination. The treatment latterly need hardly be discussed, as it was of little use.

CASE III. (*Med. Times and Gazette*, 26th September, 1885, page 423.)—*A case with several incidents suggestive of tubercle, and apparently taking origin from a caseat-*

ing femoral gland, the result of a blow—Great improve-
ment under simple treatment, but evidence of omental
thickening, and disease of one pulmonary apex still
present after three months.

John P., an emaciated boy, eight years of age, was admitted
to the Western Infirmary on the 18th January, 1884, and
dismissed on the 20th of April in the same year, so much
improved in all respects that, whether as regards the general
condition or the local phenomena, it might very truly have
been said at the time of his leaving us, that " you would
hardly have known him." Like the other cases detailed,
he had for his chief or most obtrusive symptom on admis-
sion a swollen abdomen, which measured 29½ inches in its
extreme circumference on the 20th of January (two days
after admission), and afterwards declined so rapidly, that
exactly three weeks afterwards (9th February) it was 4
inches less; the other changes noted in great detail in the
journal (but of which I will spare you the particulars)
showing that in the interval a considerable quantity of
fluid, shown to be present on admission both by the
percussion signs and by a very manifest fluctuation, had
been nearly or completely absorbed, exactly as in the case
of the little girl of like age, Jane M. Moreover, there were
some further points of resemblance between these two
cases, the statement of which here will save me, to a great
extent, from the necessity of going over the same ground
again in this brief narrative. As long as fluid was present
in appreciable quantity, it was impossible to be quite sure
of the changes in the wall which became apparent after-
wards; only it could be remarked very easily and certainly
that the physical changes were not those of simple ascites
to a like amount, the dulness on percussion over the whole
right side of the abdomen, and even on the left side in the

umbilical region, being inconsistent with the perfectly free movement or floating up of the intestines, as we have already said. Only when the fluid effusion was nearly or altogether gone did it become evident that there was an area of relatively dull superficial percussion extending across the abdomen in the umbilical region, and with this (at date 9th February, twenty-three days after admission) "a feeling of decidedly increased resistance on the right side of the abdomen especially, and even a certain amount of irregularity as of a transverse induration just about the level of the umbilicus and for an inch and a half above it, corresponding exactly with the type of a thickened and retracted omentum; and it is noteworthy (the report proceeds) that the dull percussion, though perhaps everywhere more apparent on the right side (it had been according to a previous report dull all over on the right side) is at this level decidedly greater—*i. e.*, less easily penetrated." (You will remember the significance that I attached in the other cases to this fact.) "The left hypochondrium and epigastrium are now more approximately normal than formerly (*i. e.*, than eight days before, when it was doubtful if the percussion even over the stomach was quite normal), and it is only from about $1\frac{1}{2}$ inches below the hypochondrium downwards, that delicate manipulation makes out abnormal percussion superficially, which continues from this point to the lower abdomen, being, however, very easily penetrated a little to the left of, and below, the umbilicus. On this side also, there is a feeling of undue and unequal resistance, which may very well be conceived to correspond with a thickened great omentum, but by no means so definite or so limited in area as on the other side." What gave these facts a quite peculiar interest for the clinical class at the time was that if they were not declared in advance, they

were at least sought for as the probable and (as it were) normal results of the antecedent conditions, and accordingly every care was taken, by repeated comparison with healthy subjects and with the previous reports, to guard against every possible fallacy in observation.

Thus far, then, the case of John P. corresponds so much in its details with that of Jane M., that we may consider the local details of the physical facts in the abdomen as sufficiently clearly indicated. There were two other points of resemblance between these cases. This little boy, like Jane M., suffered, as we were told, no pain in his abdomen throughout; indeed, he suffered less pain, if possible, than Jane M., though he was much more of an invalid, and, as we shall see, much more severely *ill*, altogether. Then, in the family history there was one other fact common to the two cases; this boy had, among six brothers and sisters, one who was said to have been ill of, and in fact to have died of, "a wasting of the bowels." I give the popular expression for what it is worth, in both cases: we know, and are likely to know, nothing more of the facts, or of the family history, in either case.

But with these facts resembling, or common to the two cases, there were also very considerable differences. One was as regards the temperatures, which in the former case were scarcely ever more than just appreciably abnormal, and this only for a very brief period, while in John P.'s case they once reached 102·6° F., and were on repeated occasions at upwards of 101° within the first three weeks after admission, the temperatures so elevated being almost always the evening temperatures, while the morning temperatures were approximately normal, and the daily oscillation therefore amounted to two or three degrees of Fahrenheit's scale (one and a half to two or more C.),

TUBERCULAR PERITONITIS.

CHART OF TEMPERATURE.

JOHN P., ÆT. 8.
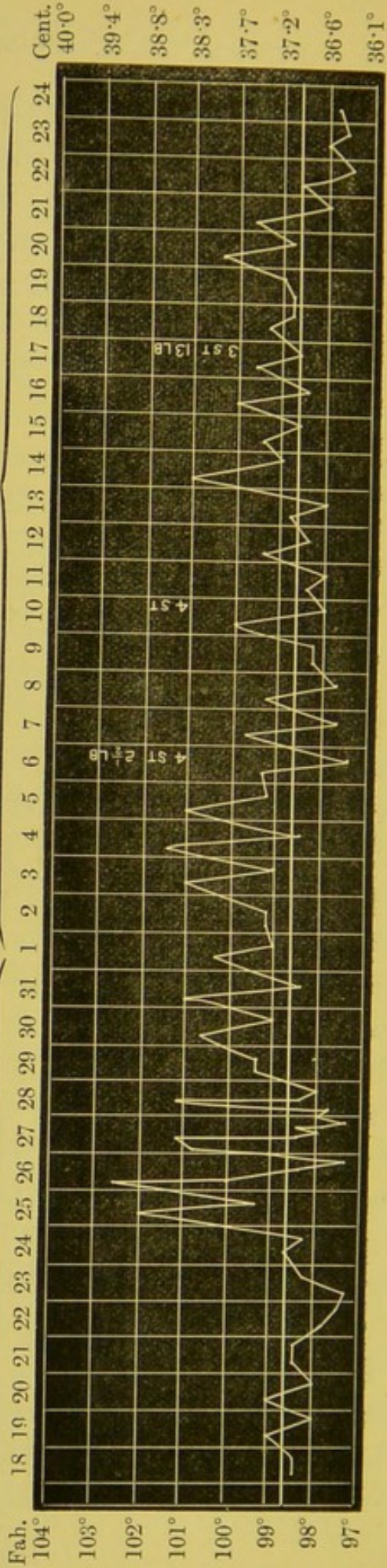
there being in one instance a fall of 5·4° F. during a single afternoon and night—*i. e.*, from 102·6° to 97·2° F., or from a very decidedly feverish to a very decidedly subnormal temperature. These are typical facts for a large proportion of cases of this kind, and accordingly you might almost take the temperatures of five or six weeks in this case, here recorded on a chart or diagram, as being a clinical working model for what is to be expected in a well defined case of chronic or sub-acute peritonitis in a child, when it is only moderately severe—so severe as to justify apprehension, but not so severe as to exclude hope of great and marked benefit through care and treatment. In this case, the abnormal temperatures gave way after six weeks' residence, and all the other symptoms underwent a corresponding improvement; so much so that, but for the very decisive local lesions, you would almost have been justified in doubting the serious or dangerous character of the case. But although, as we have said, a typical case so far, it is also true that in many other diseases of the infantile period, you may have temperatures not essentially differing from those of chronic peritonitis; and it is also probable that you may have (though rarely, I believe) a chronic peritonitis in which the temperatures are never appreciably disturbed at all.

There were, however, other circumstances in the case now reported to you, which gave to it a much graver aspect in my eyes at the time than the case of Jane M., and thus enabled me to feel a corresponding sense of relief, as from something happily occurring and yet unexpected, when the course of the symptoms showed so decidedly, after six weeks, that we were on the line of a proximate, if not an absolute *cure* (popularly so-called) of this disease. One ominous-looking fact was that the serous or sero-

fibrinous effusion was not limited to the peritoneal cavity, a very considerable amount of dropsical swelling of the penis and scrotum having taken place about a week after admission ; this being, too, altogether without swelling of the feet, or any of the other concomitants of general dropsy, but still, on this very account, an exceptional fact, and one suggestive of lesions in the venous and lymphatic systems of the pelvis. Another fact was the presence of diarrhœa (as in the case of Mary Jane S., but not so severe) during the second week of the abdominal swelling. A third fact was the presence of a lesion, very distinct in its physical signs, and not without significantly ominous symptoms—" cough, unattended by expectoration, and sweating about the head, especially in the night-time, which had accompanied the progress of the more obvious abdominal signs from the first—*i. e.*, for about three weeks before admission." The physical signs in question were :— " slight dulness on percussion at the left apex, over the two uppermost ribs in front ;" respiratory murmur in this situation " loud, harsh, and with a slightly tubular quality " (as compared even with the normally puerile R. M. all over); this abnormal quality extending slightly to the apex behind, but without dull percussion, or any râle either behind or in front. All the other organs, so far as they could be observed accurately, were normal to physical diagnosis ; but a difference like this in the respiratory murmur of the two apices is so significant of constitutional infirmity, that even if we had no other sign of disease, and no appreciable symptoms at all, we should be justified in looking upon it as a ground for suspicion of more or less chronically impaired health, especially in a child or young person.

But perhaps the most exceptional of all the points of

difference between the case of John P. and that of the girl
of the same age, Jane M., who made such a good recovery,
was one fact in the previous history which I have reserved
to the last, because it involves a doubtful question of
ætiology, and also one of pathology. In Jane M.'s case,
beyond a vague statement that she had always had rather
a large abdomen, there was nothing at all to indicate any
local lesion, or any well-established cause of possible injury
to the constitutional health, up to the invasion of the
disease for which she was admitted. In the case of John
P., on the other hand, his mother distinctly attested (as
she believed) the origin of the child's ill-health from a
local injury quite outside the abdomen, and only connected
with it indirectly through the vascular and lymphatic
systems. He was a very healthy boy, she declared at our
first interview with her, till four months ago, and had no
illnesses beyond what are common to most children. "At
this time he received a blow on the upper part of his
right thigh, which was very soon followed by a round
and painful swelling, the size of a walnut, among the soft
parts in the femoral region ; but after a few days the pain
became less, and the swelling also diminished. It has since
remained quite stationary and free from pain. After this
injury, the boy never seemed to recover perfect health.
He lost appetite, became paler, weak, and emaciated. These
symptoms went on till about three weeks before admission,
when a general swelling of the abdomen was observed,
which has gone on increasing until the present date (20th
January, two days after admission). This swelling was
not accompanied by any abdominal pain, but there has
since been increasing weakness and emaciation, &c., the cough
and hectic fever being presumably of the same date, and

probably of identical origin with the abdominal disease." Taking these facts in connection with the rather unusua fact above referred to, of dropsy of the scrotum and penis, suggestive of local venous obstruction in the pelvis or lower abdomen, it is difficult to avoid the conclusion that an inflamed and caseating gland at the seat of the blow had been in some way or other the starting-point of all the subsequent lesions; a theory, as you know, which has been in great favour in Germany for many years past as bearing on the causation of tuberculosis; and which, although I do not adopt it in the extreme sense of Niemeyer, for example, as regards phthisis pulmonalis, is certainly well fitted to explain, so far, the order of events in this case. Here is the actual description of the facts, as they were observed, in connection with the preceding history, a few days after admission.

January 20th.—"The swelling described as the first incident of his illness is rather less than a walnut in size, situate in Scarpa's space on the right side, about two inches below Poupart's ligament. On handling, it gives at first the impression of being soft aud elastic, but deeper palpation leads rather to the belief that it consists of a solid base, the more superficial part being so highly elastic as almost to suggest fluctuation. The skin over it does not differ in appearance from the neighbouring skin. In the right inguinal region several of the glands, chiefly belonging to the chain running in the direction of Poupart's ligament, are distinctly enlarged." And, again, on February 1st, "An examination of the usual sites of glandular enlargements shows an entire absence of these in the neck, axillæ, and left groin. In the right upper femoral region there is a soft, elastic, not decidedly fluctuant swelling, between

the size of a hazel-nut and that of a walnut, fairly mobile as regards the superficial textures, and presenting no pointing or other sign of an abscess, but seeming to be attached to the deep fascia with considerable firmness. It is painless on handling, but the history quite definitely leads to the belief that in the first instance it was caused by a blow, was painful, and considerably larger than it is at present. Dr. Gairdner finds a difficulty in determining how far this swelling is glandular, but several of the glands over Poupart's ligament are slightly enlarged, without any inflammatory thickening of the surrounding textures or pain on pressure." As this swelling, whatever it was originally, did not frankly suppurate during the whole time this patient was under observation (three months), it is certainly most probable that a caseating process had taken the place of active suppuration, if indeed the latter at any time existed.

The latest information I have to give you as to the progress of this case under observation, and, I may say, under a very simple treatment, much resembling that of Jane M., is contained in the following report of 8th April, which I think it worth while to present to you exactly as it appears in the journal, and which I think you will agree with me in considering, under the circumstances stated, very satisfactory.

"Since the 1st February, at which date a general survey of the temperatures was recorded, there have been on eight evenings temperatures noted extending up to, or over, 100°, the maxima being on February 4th, 101·5°; on 14th, 101°; and since this barely exceeding 100° in any instance; while the great majority of the temperatures, especially since the first week of February, have been

subnormal. A very distinct, though gradual, gain in weight, and a very marked filling up of the features as compared with his state on admission, has been observed throughout this period. The urine, moreover, has undergone very decided and apparently permanent increase as compared with the state on admission and during the period of febrile temperatures. The last ten daily collections recorded from 21st to 31st March give 28 oz. as a mean. This mean quantity was even exceeded about the middle of February for some days together, 31 oz. mean being recorded in six days from 21st to 26th February, but on the whole the above may be considered to present a fair average for the last six weeks. The last weight recorded (April 7th) was 4 st. 4 lb. The boy seems to suffer no discomfort at all either from the abdominal symptoms or from the swollen gland in the right groin, which seems to be slowly maturing towards abscess, and presents a very distinct red blush at the most elevated point of the skin.

"The conditions in the abdomen as last described are not very essentially altered, except that the distinct feeling of transverse, almost solid impaction to the right of the umbilicus, has given way to a more diffused and less definite sense of increased resistance, with superficial dull percussion, however, almost equally characteristic of a thickening extending over a transverse zone about three inches in vertical diameter, half of this being above and half below the umbilicus. A nearly corresponding dulness on the left side is also much less in degree and in limits. Tongue perfectly clean. There has been no diarrhœa at any time. There is still distinctly impaired percussion in the left pulmonary apex, and the R. M. is relatively less full

than in the opposite; but the alteration in quality is so slight that but for the other facts it would certainly be disregarded."

On the 20th of April, 1884, as above stated, John P. was dismissed from the Western Infirmary very much improved in health and in general condition.

When Cases II and III are carefully considered in connection with Case I, it will be found that, although differing in their prognosis and probable issue,* they are cases, in many respects, of the same order. In all of them there was observed the presence of a very notable quantity of fluid effusion in the abdominal cavity, with physical conditions differentiating the case from simple ascites, and leading to the inference of lymph effused so as to restrain the free movements of the intestines; while in two of these cases at least the physical signs after the removal of the fluid were exactly those of thickening of the great omentum. Each case, in fact, is a typical illustration of one or other feature in the above diagnosis; and while in Case I, taken by itself, the complete and satisfactory curative result might induce a doubt as to the pathology, and lead to much hesitation in admitting the case into the category of a *tabes*, the other two cases, in various degrees, may be said to fully deserve this designation. The case now to be submitted (Case IV) is a much more remarkable one than any of the preceding; and how far it can be said to fall within the description of a *tabes* may be held over for further discussion.

* I have not been able to trace the boy John P., although inquiries have been made, by post and otherwise, at his former residence. The facts recorded must be taken, therefore, for what they are worth as indicating a possible recovery. It is to be feared that the improvement may have been only temporary.

CASE IV.—(*Medical Times and Gazette*, 17th October, 1885, p. 525.**)—Very well-marked thickening, amounting to tumour of the great omentum, gradually resolved in two years, with extremely insignificant symptoms. Crepitus in right pulmonary upper lobe, arising under observation, and persisting during the entire course of treatment with little modification—Symptoms scarcely appreciable, and general health well maintained.*

Lizzie (or Elizabeth) C. was 9 years of age when she was first brought under my notice on the 7th of May, 1883, on account of a swollen abdomen; which, it may be said quite unreservedly, was at that time her only symptom, and, even after most careful examination and enquiry into the facts, was almost the only thing that could be ascertained about her suggestive of organic disease at all. Now, a swollen abdomen in a child may proceed from many causes, and is often regarded as a very trivial and unimportant disorder, *per se.* I do not say that you will be right in so regarding it; I would rather lead you to be very careful about all such cases, and not to dismiss them in the summary way they are often treated, with a little grey powder or a few doses of castor oil. No case could have been more trivial than that of Lizzie C., if judged by the amount of local uneasiness or of constitutional disturbance; for, from a note extending to more than seven pages in the Hospital Journal (Ward 6, T., pp. **222**, *et seq.*), and mostly made by myself in presence of the

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* This case, from its great importance, was recorded in the *Medical Times and Gazette* with the most anxious care to convey, if possible, the exact verbal impressions, corresponding with a great number of separate clinical demonstrations and lectures to successive classes of medical students, during the two years she was more or less under observation up to October, 1885. It is, therefore, considered expedient to preserve here the very words of the original publication, without any attempt at condensation, and omitting only an introductory paragraph.

class within two days of her first admission to the Western
Infirmary, we can assure ourselves even now that the most
thorough investigation was made at that date, and that
neither in respect of complaints, nor in respect of physical
and physiognomic signs, was there anything to beget
even a suspicion of grave disease, except the physical
condition of the abdomen; no hereditary syphilis, no
scrofulous indications, no rickets, no chest symptoms of
any importance, unless it be the cicatrices of a very old
and obviously artificial eruption which may have pointed to
some chest disease in early infancy, treated with antimonial
or other ointment, but in the interval of years practically
lost sight of and forgotten. She was said to have been
always a healthy child, and her appearance confirmed it:
she was, indeed, the very picture of health, nay, even of
superfluous or "rude" health (as judged by her colour
and general appearance) at the time of admission. She
herself had first drawn attention to the swelling and hard-
ness in the abdomen about three months before, and she
admitted that she had felt it "slightly sore." Her mother
affirmed that, a few days after the swelling was first
noticed, the girl was troubled with diarrhœa, at first pretty
severe; but this had ceased after a fortnight, under merely
dietetic treatment and without medical advice, and it had
not recurred. Appetite and digestion, according to her
mother's testimony as well as her own remained unimpaired;
and I may add that our experience in the ward tended in
all respects to confirm these statements, in respect that her
temperatures, carefully taken for weeks together, showed
no signs of fever; and her body-weight, by no means
apparently deficient even at first, went on increasing after
admission.

And yet, with all these facts in her favour, more strongly

so even than in the case of Jane M. (the first of this
series that I placed before you), there was no doubt at all,
in Lizzie C.'s case at this time, of the existence of a distinct
tumour in the umbilical region, corresponding exactly, both
as regards percussion and palpation, with the characters of
a thickened great omentum as already detailed to you.
The report, which goes into every element of the diagnosis
as demonstrated to the class on 9th May, is open to your
inspection; and I spare you the details here, simply because
there is so much besides in the case to occupy us. The
tumour was distinct and palpable, dense to the feel and
superficially dull over the entire umbilical region, so that it
could not have been either fæcal accumulation, or merely
inflated intestines. The epigastric, hypogastric, and both
iliac regions were comparatively soft and yielding, and
quite normal to percussion. There was not, in fact, a single
element in the diagnosis of thickening of the great omentum
that was wanting in this case, and all the physical signs
were present in even a more definite form in Lizzie C.,
than in any of the others; but there was no evidence at
all of fluid effusion at the time we first saw this girl, nor
at any time afterwards. We judged it possible or probable
that there might have been such effusion at an earlier
date; but if so (as in the case of Jane M.), it had been
absorbed so as to leave no traces to physical diagnosis.

Had the case ended favourably at this point, and with-
out any observations of interest other than those above
mentioned, the details would have been mainly a reproduction,
or rather an anticipation, of the facts presented to you in
Jane M., with some quite non-essential variations. Had
this been so, I should probably have been satisfied with
alluding to it briefly as such. But there is much more of
clinical instruction in the case than this. The great

probability, from the pathological point of view, of such a lesion as existed in Lizzie C. being of tubercular origin led to a more than usually, indeed an anxiously careful exploration of the lungs at the very first examination (May 9th). The result of this was that there was some ambiguity as regards the auscultation and percussion signs, respectively, of the two apices; but on the whole we thought the right apex showed "*a distinct deficiency, both as to quantity and quality,* of the R. M., and even, perhaps, *a little irregularity amounting to jerking.* At the back," the report continues, "percussion of the right apex appears relatively impaired, and here also the R. M. is *inferior in fulness to that of the left, but without râle or other alteration in quality.*" I emphasise one or two points in this report for a reason that will presently appear. Taken by itself, it amounts to a suspicion, but no more, of disease, probably tubercular, in the right apex.

Just a week after this, however (16th May), I was again examining this patient, after noting carefully her satisfactory condition since admission as to the general symptoms. On this occasion my attention was attracted for the first time by "a fine and slight crepitus, probably of the mucous order, developed in the right apex since the previous careful observation, and audible very distinctly down to the second, and less so down to the third rib, accompanied everywhere by wheezing râles, but of no great intensity." The observations, in other respects, as to the relative fulness of the R. M., &c., on the two sides, were confirmed on this occasion.

I purposely caused a large number of persons to listen to these sounds, because, although it was possible that they might have been merely occasional and transitory, and therefore of no great importance, it was also possible that

they might have a bearing on the diagnosis and prognosis of the case as one of tubercular disease both in the abdomen and the chest. The observation being made thus early and thus studiously with reference to this point, was followed up with the more freedom, because the state of the patient admitted easily of frequent examination. She was gaining in weight; she was free from fever and from pain; she had no considerable cough,* and no expectoration. Yet from that time onwards I can absolutely affirm that during the whole summer, when she was under daily observation up to the end of September, 1883, there was not a single occasion when the râle above referred to was found absent from the right lung in front, or even very difficult of detection. It had always nearly the same characters—a very fine clicking or crepitant râle, always much more distinct during inspiration, and sometimes only audible with forced inspiration, without any appreciable amount of tubular or other altered quality of the R. M. in the right lung; but with, as above stated, a somewhat depreciated relative amount of fulness, and this both at apex and base. It perhaps differed from the crepitus of the early stage of pneumonia, but very slightly so, in being more of a moist character; and certainly much more in its long persistence without other alterations. All this was frequently the subject of clinical commentary, and of very repeated observation, not only by myself and my assistants but by some of the other physicians of the Western Infirmary, and by occasional strangers. In not one instance did the râle altogether fail to be observed, although its area and its

* The precise fact, as noted, 16th May, is as follows :—" The ward sister, on being appealed to, has noticed a short cough without expectoration, but not such as to molest her, or to have attracted the child's own attention, which, therefore, may have been indefinitely present before admission."

amount both varied. It was chiefly heard over the upper lobe, and never on the left side.

During her residence, on this occasion, of nearly four months, Lizzie C. gained in weight, in all, 25½ lbs., having been on admission 3 st. 2 lbs., and at her dismissal, 4 st. 13½ lbs. The condition of the abdomen very notably improved, without its being able to be said that the physical signs mentioned had altogether disappeared. Her cheerfulness, and her sense of well-being, were unimpaired throughout, and no local symptom seemed ever to cause her any uneasiness. Her cheeks were almost preternaturally rosy and fat, and it was only by becoming satisfied of the entire absence of fever that we acquitted her at times of a morbid flush; but I afterwards came to know that this peculiarity, and her altogether exceptionally good physique, had procured her an engagement at a theatre as a subordinate in a pantomime, and were by her mother and all her friends considered to be exactly in accordance with her condition from infancy onwards. I may add, that although partial sweats about the head and face appear once to be mentioned in the report, it was during warm weather, and was not considered, ultimately, to have been a fact of any importance.

Here, then, we have a combination of details, in the case of Lizzie C. which I have given you, so far, in a much condensed form, or abstract, from journals T and U of the female ward, inviting you to refer to these journals for yourselves, in case you should be of opinion that all the evidence of importance is not fully before you. I will merely say here that there is not a single point, in my opinion, in which the evidence of detail bearing on the peritoneal disease was not as carefully recorded, and as faithfully considered in reference to possible fallacies, as in

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any of the other cases here cited to you, or, as in the
case of Jane M. and Mary Jane S., which have been mainly
under your own observation. And you are not to suppose
for a moment that any of these details are at all new to
me, as I may presume some of them are to you.* On the
contrary, cases of this kind in a general way (though that
of Lizzie C. is certainly an exceptional one) have been con-
stantly occurring to me for more than thirty years in
hospital and dispensary practice, as well as in consultations
among the more affluent classes; and the impressions about
them that I wish to leave on your minds might be almost
indefinitely illustrated out of the fifty or sixty journals
which a decennium of the Western Infirmary has accumu-
lated for our wards, as well as by reference to older
experience in the Royal Infirmary here, and in the
Edinburgh Royal Infirmary. What I wish to do now,
however, is not to generalise prematurely, but rather to
place before you the lessons of these four cases, thus suc-
cessively presented in series, as elements out of which you
may learn something more for yourselves. And it is to press
home the lessons of Lizzie C.'s case that I now invite you
to read with me more continuously the later reports, made
when she could be hardly spoken of as a patient, but rather
when she was sent for at my request, in order to take stock
(as it were) of her physical condition and progress.

On 1st February, 1884, I sent for this girl (she lives at
Paisley, seven miles from this), and in the journal of Ward
6 (V, p. 169) we have a note which, as it is not long, I will

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* Although in teaching clinically, I usually make but little reference
to authorities, in dealing with demonstrable facts, it would be easy to
show that in most of the text books the diagnostic characters and sig-
nificance of thickening of the great omentum have been strangely over-
looked; although the mere anatomical fact has long been known. Hence
the prominence given to these points in the preceding papers.

place before you entire:—" Speaking generally, it may be said that all the facts noted last summer are more or less apparent, the abdomen being soft and elastic, but decidedly abnormal to percussion over the whole omental region ; and the true intestinal note (*i. e.*, on superficial percussion) being only procurable towards the hypogastrium. The râle observed in the right apex is still appreciable, and perhaps more clicking and hollow in character, but still. not so definitely altered as to suggest any well marked new physical changes. The mother's account is as follows :— Lizzie complains of nothing, is always light hearted and fit for all games with her companions, rising at eight in the morning and going to bed at eight in the evening without any appearance of undue languor, sleeps well at night, takes her food well, is never sick, has no diarrhœa and complains of no pain. As regards pulmonary symptoms, her mother remarks that she may take a cold occasionally, and did so three weeks ago ; and as the result of this a very little cough may be admitted as occasionally audible, but not so as to make the girl herself complain. [See footnote above, indicating exactly the same facts as at date 16th May, 1883.] She has preserved to the full the rosy appearance she had in summer—so much so, that the only question that can be raised is whether it does not incline to lividity. Her mother thinks her decidedly less fat than when dismissed last summer, but she has never noticed any feverishness nor sweating."

Again, on the 11th of October, 1884, I sent for Lizzie C., and on this occasion, and some later ones, much longer and more elaborate reports will be found in Journal X, p. 37, *et seq.*, the substance of which (condensing them very carefully to save repetitions, but omitting nothing essential) I will now endeavour to convey to you.

It appears that Lizzie C. suffered somewhat from headaches during the autumn of 1884 ; and as they disturbed her sleep she was on this account withdrawn from school in September, and put under medical treatment.   There was, however, no uneasiness as regards the chest or abdomen till the beginning of October, when she complained of pain in her right side, and on strict enquiry it appears she was possibly a little short in the breathing, but it was by no means a prominent symptom, and in the main, her mother adheres to the report made on 1st February (see above). She may have been a little feverish with the headaches, but otherwise there was no evidence of any constitutional disturbance.   The physical facts were exactly as before, with the exception (which, however, was in exact accordance with the observation of 16th May, 1883) of a certain amount of distinctly wheezing râles audible on the right side.   (It was presumed that these were on this occasion of recent origin.)

On the 11th October, 1884, Lizzie C. was found to weigh 5 st. 2½ lbs., as against 3 st. 2 lbs. at our first observation of her in May, 1883, and 4 st. 13½ lbs., at her dismissal in September ; so that although she had not lost weight (but in fact gained about 3 lbs. between September, 1883, and October, 1884) it might be fairly maintained that there had been either an absolute or a relative loss of weight during some portion of the latter period, corresponding, in all probability, with the illness above mentioned.   It was, however, by no means very apparent to even a professional eye that she was notably thinner or less in good health than on previous visits.

On 5th January, 1885, this girl was readmitted at her mother's request, chiefly on account of a "shortness of breath," with wheezing, especially at night.   At this time

she had gained 3 lbs. in weight since October, and looked certainly to my eye as well as ever (as a somewhat critical note made the next day informs me); the trace of "lividity" above incidentally alluded to being discounted (as it were) in consideration of previous knowledge of her appearance. The pulse was 80, regular, and of fair strength. The respiration barely 16 in the minute, and absolutely tranquil. Apart from the mother's statement, there was nothing to lead to any apprehension of renewed chest disease. It was, however, ascertained more fully than previously that Lizzie's father died of something that was called "bronchitis with inflammation of the windpipe" at the age of 30. He was a very intemperate man. Her mother also thought that there were traces, at least, of bronchial affections in the family.

The abdomen was found to have a maximum circumference at this time of $24\frac{1}{2}$ inches, *i. e.*, one-and-a-half inches less than at the time of first admission, and almost four inches less than the absolute maximum noted on 28th August, 1883. Allowing for the developmental growth and improved condition of the child generally, this was considered a highly satisfactory note, and it was borne out in detail by a most critical estimate of the physical facts, which, as it was practically superseded at a later date by a much more compendious statement with almost identical results, I will not trouble you to read over. The râle, so often previously observed and discussed, was at this date made the object of a formal lesson at the bedside to the clinical class, and in the very ample report framed for the purpose of detailed instruction, and resting on all the previous observations, the following statements emerge :—
"Dr. G. remarks, with a view to the classification of this râle (1) That during the whole period of its singular

permanence it has been heard, perhaps not quite exclu-
sively, but always in an immensely preponderating degree,
with the inspiration; (2) That while it has at different
times and in different degrees suggested so much of the
moist quality as to make it impossible to demarcate the .
râle from the moist râles, there has been no such con-
sistent progress from dry to moist as is implied in Dr.
Walshe's description of the dry and moist crackling rhonchi
respectively (see 4th edition) *Diseases of the Lungs, &c.*
pp. 326-330; therefore, it was argued, there had been no
evolution of the phenomena in the direction proper to,
and according to Dr. W., eminently characteristic of, the
progress of tubercle in the lung towards softening; (3) It
appears, both to Dr. G. and Dr. Middleton, that there has
been a little (but still only a little) increase in the tubular
or bronchial quality of the R. M. in the right apex since
the first observation; and this mostly, if not exclusively,
in the region of the sterno-clavicular articulation; (4) It
is difficult to say whether there has been any change in
the percussion since the first observation in May, 1883."

On 4th February of this year (1885) we were able to
record the results of a renewed daily observation of this
case for a month in hospital as showing once more "quite
satisfactory progress" in respect of the removal of the
slight complaints indicated on her re-admission. "She says
she has now no headaches. The pain on the right side is
gone, and she appears to be quite unconscious of any
wheezing or other notable symptom of pulmonary disease.
Appearance perfectly good; temperatures subnormal rather
than otherwise, but cannot be said to show anything
worthy of remark. Apart from the history, there would
be no reason at all for anxiety about the patient, or for
detaining her in hospital. The abnormal facts in the

abdomen are still just perceptible, but with so great an amount of difficulty that in the absence of previous history they would certainly be disregarded. It seems safe to say that the palpation is that of a normal abdomen in a plump, well nourished child, with a good deal of fat in the abdominal wall. [The percussion still, however, appreciably abnormal.] The circumference is now 26⅓ inches, being two inches of increase since she was last admitted on 6th January, but this increase is probably commensurate with an increase in the general bulk of the body; at all events, there is nothing whatever to lead one to suppose it abnormal. [The circumference in February, 1885, was in fact just over what was recorded as that of an abnormal or swollen abdomen in May, 1883, the difference being that normal development had taken the place of disease.]

As regards the chest, it was observed on 4th February 1885, that the same ambiguity which had been noted in May, 1883, existed in the percussion of the two apices. "The well known râle was again found in the right apex in even a larger and more diffused form than ever before, and, though it is difficult to put into words changes of so extremely slight a character, the râle is certainly more of a moist order, and with larger bubbles than on most previous observations, and this is the more remarkable, as it is only a few days since Dr. G. and Dr. Beveridge, examining together, were struck with the paucity of the râle, and with its being rather difficult to catch, except at some points and on very full inspiration. To-day, the râle is distinct at all points down to the third rib, and even occasionally a little below this, but during the examination by the class it becomes much less abundant and more circumscribed, and is chiefly audible in the sterno-clavicular region. Wheezing is scarcely if at all appreciable. [The

other characters of the R. M. did not differ from previous examinations; indeed, could not be said to indicate any advance in the disease even as compared with May, 1883.] Sputum has been entirely absent since admission."

On 7th February this girl was dismissed for the second time, after exactly five weeks' residence, greatly improved and practically well. The whole period of observation and treatment, taken together, in this case may be said to extend over more than two years, and it is likely, I hope, to be much further extended, for I have good hopes now Lizzie C. may attain to womanhood, or even to mature age; and, having watched her case with so much interest thus far, I shall endeavour, if possible, not to lose sight of her entirely in the future.

[On a recent occasion, being in Paisley, I had an opportunity of examining Lizzie C.; and although there was not, perhaps, any essential change in the facts, it was evident to me that she leads a life of considerable privation and perhaps of some hardship. I shall, therefore, in all probability, cause her to be admitted again to the Western Infirmary, if so disposed, in the course of the winter. The râle in the right lung, however, was less pronounced than it has frequently been in the course of this report.]

ON THE PATHOLOGY OF PHTHISIS PULMONALIS.

By JOSEPH COATS, M.D.

LECTURE I.

THE TWO TYPICAL FORMS OF PHTHISIS.

Introduction. Definition of phthisis pulmonalis. The forms of phthisis.
*(1) The caseous form: general appearances; the advancing lesion
centres in the bronchioles; the presence of tubercles, and of caseous
necrosis; the formation of cavities. (2) The fibroid form: general
appearances; the advancing lesion centres in the bronchioles; the
presence of tubercles; extension to lymphatics; the fibroid change;
occurrence of bronchiectasis, emphysema, and cysts in pleura; the
pigmentation. Comparison of the two forms; both are tubercular.
Clinical cases.*

THE subject of phthisis pulmonalis is a very extensive one
whether we view it from the purely pathological or from the
clinical side. In the present course of lectures it is not my
object to give a complete exposition of the whole pathology
of phthisis. I am addressing practitioners who are actively
engaged in the observation and treatment of disease, and
my object is to give such an exposition of the subject as
will fit into your experience and assist you in the solution
of the problems that must daily present themselves to
you in a class of diseases which forms a large part of
your practice. While, therefore, the course will not be
a rigidly systematic one, I believe that we shall have

under consideration most of the important questions connected with the pathology of this disease.

In regard to my own personal position, I may be allowed to say that in the course of my career as Pathologist, I have had consciously before my mind most of the leading problems involved in the pathology of phthisis pulmonalis, and have brought the various views given out during that period as much as possible to the test of my own observation. My position in regard to many of these views has been that of imperfect conviction. About the time I began work as a pathologist an attempt was being made to eliminate a large proportion of the cases of phthisis pulmonalis from the domain of tuberculosis, and, being naturally impressed as a young man with what was most novel, I sincerely endeavoured in the *post-mortem* room to discriminate between tubercular and non-tubercular phthisis—without much success indeed, and with a growing consciousness that the essential pathology of the disease had not been attained. It is only of late years that I have been able with some degree of confidence to see my way through the complications of the subject, and it is, perhaps, for this reason that I have chosen this disease for discussion in the present course of lectures. I may perhaps be allowed to add that, although chiefly engaged in the observation of morbid anatomy, I have always recognised the paramount importance of taking cognisance of the clinical as well as the anatomical aspects of the cases. In my official capacity of pathologist I have made it a part of my duty to see that, along with the account of the *post-mortem* examination an abstract of the clinical history shall be entered in the Pathological Register. The *post-mortem* records are thus kept in close relation to the clinical cases, and further reference to the

Ward Journals is rendered easy. Believing also that pathological problems have always their clinical side, I have throughout retained appointments in the Infirmary which have brought me into relation with patients and afforded me a certain amount of clinical experience.

The opinions which I have to offer you here are based mainly on the *post-mortem* examination of numerous cases, in the Royal and Western Infirmaries, with due regard to the clinical facts. I have gone over our Pathological Registers in the Western Infirmary, in which we are now met, and from the records of my own examinations I have selected 186 cases, in which the accounts are sufficiently complete to give a satisfactory basis for tabulation of results. In any statistical statements which I shall make these cases will form the basis.

DEFINITION.—It is very difficult to give a satisfactory definition of phthisis pulmonalis, if due regard be paid both to the origin of the name and its current meaning in the present day. In its original use the term Phthisis had reference to a general wasting of the body, irrespective of the cause of the wasting. The term was qualified by an adjective when it was desired to connect the wasting with disease of one or other of the internal organs; in this way the terms phthisis renalis, phthisis abdominalis, phthisis laryngea, phthisis pulmonalis came into use, and to some extent these terms still survive. In its modern use the term phthisis has a more definite and limited application. It is not merely a wasting of the body associated with some affection of the lungs, that is designated by the term, but its use is commonly limited to a particular disease of the lungs. There can be no doubt that in the great majority of cases of wasting of the body due to disease

of the lungs, the lesions in these organs have a number
of common features, which afford a certain basis for group-
ing them together. The name phthisis pulmonalis has
therefore, by a natural process, come to be applied to cases
in which the lungs are affected by a progressive lesion,
the ordinary and regular result of which is the destruction
of the lung tissue and the formation of cavities. The idea
of wasting, originally applied to the body in general, is
thus extended to the lungs, and, as the wasting of the
body is obviously a secondary phenomenon, it does not enter
into consideration as an essential element in the pathological
process. As a matter of fact it is not infrequently absent.

Assuming, then, as our definition of phthisis, that it is a
progressive disease of the lungs whose ordinary and regular
result is the destruction of the lung tissue and the forma-
tion of cavities, it will be our duty in this course to take
a general survey of the disease, and to consider the whole
phenomena from the first morbid appearances on to the
formation of cavities. We shall study the structural
changes in the various stages of the process, the nature of
the process, and the various secondary consequences which
result, in the lungs and elsewhere.

THE FORMS OF PHTHISIS PULMONALIS.

It is possible, in going over a considerable number of
records such as those to which I have referred, to distin-
guish two groups of cases which have a tolerably broad
line of distinction between them. These two may for
convenience be termed the caseating and the fibroid forms
respectively, and it will be convenient to begin here with
a description of each of these forms in their naked eye
and microscopical characters.

I. THE CASEOUS FORM.—The commonest case is that in which the upper lobe of one or both lungs is the seat of one or more cavities of very irregular outline. The cavities are of various sizes, but very often there is a large one divided by partial septa and evidently formed by the coalescence of several smaller ones. The cavities may contain a curdy pus, or they may be comparatively empty, but their internal surface is usually coated more or less
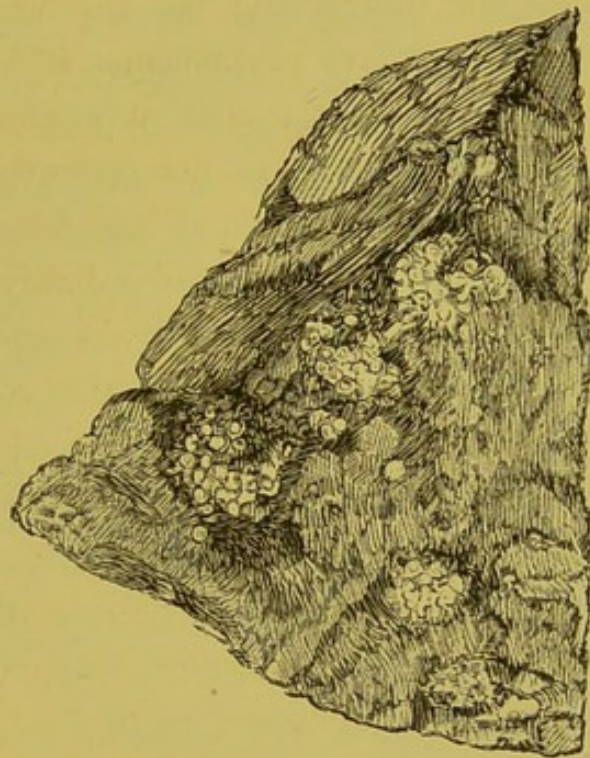

FIG. 1.—A portion of lung showing the naked-eye appearances of the initiatory lesions in caseous phthisis. There are rounded isolated condensations indicating the lobular extension. Natural size.

with a yellow curdy matter. They may involve the greater part of the upper lobe and even a portion of the lower lobe, and the tissue immediately around them, especially the older ones, may be condensed and pigmented.

As a general rule we have to look away from the cavities into the midst of the more sound and crepitant tissue in order to observe the characters of the process in its earlier and more distinctive stages. One can nearly always

distinguish in the midst of crepitant tissue isolated areas of condensation such as those illustrated in Figure 1. They are usually more or less rounded in outline, and can be felt as solid masses in the midst of the soft crepitant tissue. On section they often present an appearance suggesting a central stem and bodies grouped around it as grapes in a bunch, or perhaps more correctly as the carpels of a berry. This appearance was long ago figured by Carswell,* but in such an exaggerated form that certain observers, and among them Hilton Fagge, have denied its existence altogether. I must say, however, that these isolated condensations nearly always have to my eye a suggestion of such a grouping around a central stem, although the appearance is not always diagrammatically similar to that of a berry. The central parts of these areas are usually whitish or yellowish and opaque, and this appearance may involve almost the whole area. Generally, however, the peripheral parts have a grey translucent character. It is hardly necessary to say that the white or yellow opaque structure is what used to be designated yellow or crude tubercle.

· Whilst these isolated areas may be found in the midst of comparatively healthy tissue, there are usually more continuous condensations, evidently formed for the most part by the coalescence of smaller areas, although sometimes there is evidence of an almost simultaneous involvement of a considerable portion of the lung.

As the smaller areas of condensation form the advancing outposts of the disease, it is by the study of them, and indeed of the smallest of them, that we may arrive at some conclusion as to the course of the pathological

* Carswell, *Pathological Anatomy: illustrations of the elementary forms of disease.* London, 1833-38.

process.   I have placed on the table microscopes under
which are sections illustrating most of the points to which
I have now to refer.   It will be found that the most
uniform commencement of this form of phthisis is, that
the smaller bronchial tubes become plugged with what is
at first simply an inflammatory exudation.   This is illus-
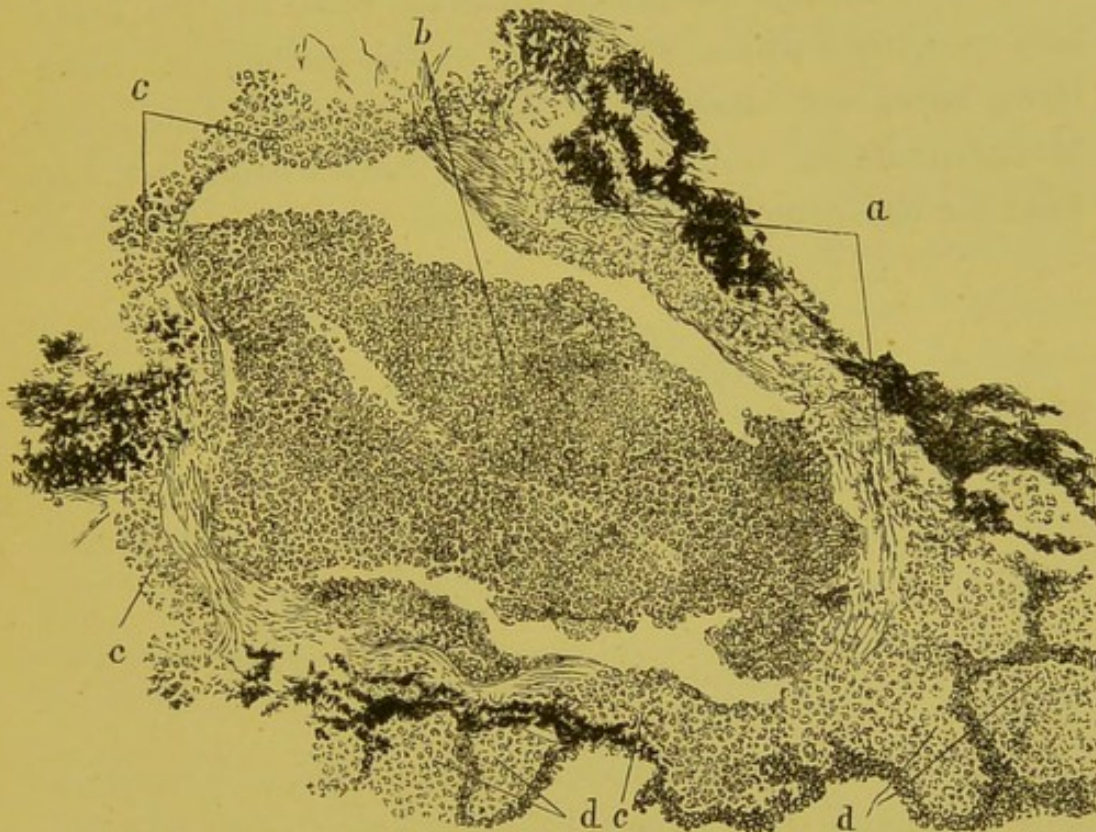trated in Figure 2.   The outline of the tube is preserved,

FIG. 2.—Caseous phthisis; recent centre.   a. Wall of bronchus with pigment externally.
b. Plug in bronchus.   c. Round cells infiltrating wall of bronchus.   d. Alveoli filled with
blood and catarrhal cells.   ×60.

but its wall is considerably infiltrated with round cells,
the products of inflammation, while its calibre is occupied
by desquamated epithelium mixed with round cells.   The
plugged bronchial tubes form the central stems and branching
twigs of the areas under consideration, but the lung-alveoli
in the neighbourhood of the affected tubes are likewise
involved, and here also we have the effects of inflammation.
It is sometimes possible to catch the process at the early

stage represented in Figure 3, in which the epithelium lining the alveoli has undergone a very marked enlargement. This epithelium consists normally of very delicate flat cells, but here it is converted into a substantial layer of nucleated cells. As a general rule these cells have already, at the time of observation, passed into the central parts of the alveoli, where they form groups of large cells, which are usually designated catarrhal cells, and are generally recognised as the derivatives of the epithelium. Sometimes these are accompanied by the ordinary round cells of inflammation, and in very acute cases these latter may even be the principal constituents at least in some of the
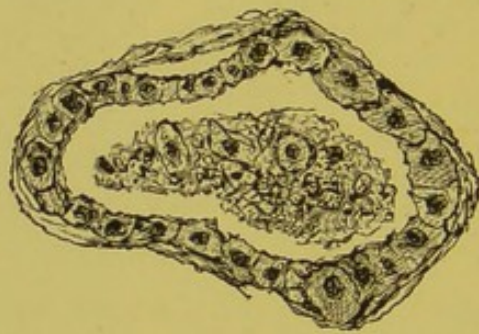
FIG. 3. Alveolus near affected bronchus. The epithelium is enlarged, and there is some desquamated epithelium in its calibre.

alveoli. I have also sometimes seen fibrine with round cells occupying the alveoli as in ordinary cases of acute pneumonia.

We have here, then, as the primary lesion an inflammation centring in the finer bronchial tubes and extending to the proper parenchyma of the lung, and hence often described under the designation broncho - pneumonia. The products of the inflammation vary with the chronicity of the lesion, consisting usually of the derivatives of the epithelium, but frequently mixed with ordinary round cells, and sometimes even with fibrine, these affording evidence of an acute character in the lesion.

But there are two elements which render this inflammation peculiar, and separate it from ordinary inflammations; these are—*the presence of tubercles and the occurrence of caseous metamorphosis.*

The wall of the affected bronchus is always infiltrated with round cells, so that its normal structure is greatly obscured. This infiltration extends to the surrounding connective tissue, and to the walls of the neighbouring alveoli, so that the external outline of the tube is also obscured, the whole being involved in the round-cell infiltration. In the midst of this, however, we often find those rounded aggregations of cells to which it is customary to give the name of tubercles. As a histological entity the tubercle is a small collection of cells assuming a rounded form. The cells of which it is originally composed are somewhat various in character, but in a perfectly fresh tubercle it is possible to distinguish small round cells having the ordinary characters of leucocytes, larger cells which resemble epithelial cells in structure, and very large cells, called giant cells, which when present often form very prominent objects. These giant cells are irregular in outline and contain numerous nuclei which are frequently arranged in a row along the periphery of the cell. While these are the typical characters of tubercles, they are in actual cases very frequently of much less definite structure. In the first place their cells are very prone to undergo caseous metamorphosis, which, as we shall see immediately, has the effect of greatly obscuring structure, and, in the second place, being situated frequently in the midst of tissue infiltrated with round cells, their external outline is often very indefinite. Hence it not infrequently happens that the giant cells are the most prominent objects in the tubercles, and may even be almost the only recognisable elements. The giant cells sometimes take into their substance granules

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of black pigment such as we find abundantly in the lungs of all adults, and this may render them the more obvious.

Caseous metamorphosis, or, as I prefer to call it, caseous necrosis, soon overtakes the affected structures. This consists in a necrosis or death of the structures accompanied by the production of finely granular fat. As this change reduces everything which it affects to a homogeneous granular

Fig. 4.—Further stage of caseous phthisis. *a.* Caseous centre including plug and bronchial wall rendered indistinguishable by the caseation. *b.* Alveoli scarcely distinguishable. *c.* Alveoli filled with inflammatory exudation and with walls infiltrated. ×60.

condition, it greatly obscures the structure, and renders the identification of the individual elements very difficult. It occurs in all the structures already described as affected by the inflammation, the plug which fills the bronchus, the bronchial wall, the contents of the alveolus, the alveolar wall, the tubercles. If the disease be not very advanced, as in the specimen from which Figure 4 has been taken, then

it may be seen that the caseous necrosis has evidently its starting point in the bronchus, the outline of the tube being still vaguely visible towards the centre of the affected area. In this illustration the change has extended to the alveoli immediately around the bronchus, and their outlines are obscurely visible in the midst of the general granular appearance. Outside these again there is the regular round-cell infiltration involving the connective tissue and walls of the alveoli, while the alveoli are filled with catarrhal cells. In the large areas where condensation is older caseation is so complete that very little structure can be detected.

The comparison of the material which results from this process with cheese is not an unhappy one. It has the yellow colour and somewhat brittle consistence of cheese, and it owes these characters, just as cheese does, to the presence of finely divided fat contained in a dry nitrogenous basis-substance. It need hardly be repeated that the caseous matter which we have here been considering is that which has frequently been designated by the terms yellow and crude tubercle.

There seems to be no reason to doubt that the caseous matter which results from this change is really dead matter, and the process is one of death or necrosis, the affected structures being utterly incapable of recovery. The dead matter may lie for a long time and do little harm, just as any inert dead animal matter may. It may even undergo a partial process of absorption as also happens with dead animal matter in general; or with partial absorption there may be combined a process of infiltration with lime-salts, so that a cretaceous mass remains imbedded in the lung. These processes, however, imply that the disease has ceased to be active, and will fall to be considered afterwards in treating of the healing of phthisis. Much more common is the

process of softening or breaking-down of the caseous matter.
Indications of this are often visible to the naked eye in
the central parts of the areas already described, especially
if the process be an acute one. Under the microscope it
can be frequently determined that the softening usually
begins in the situation of the bronchus, which thus again
proclaims itself the centre of the process. In Figure 4, already
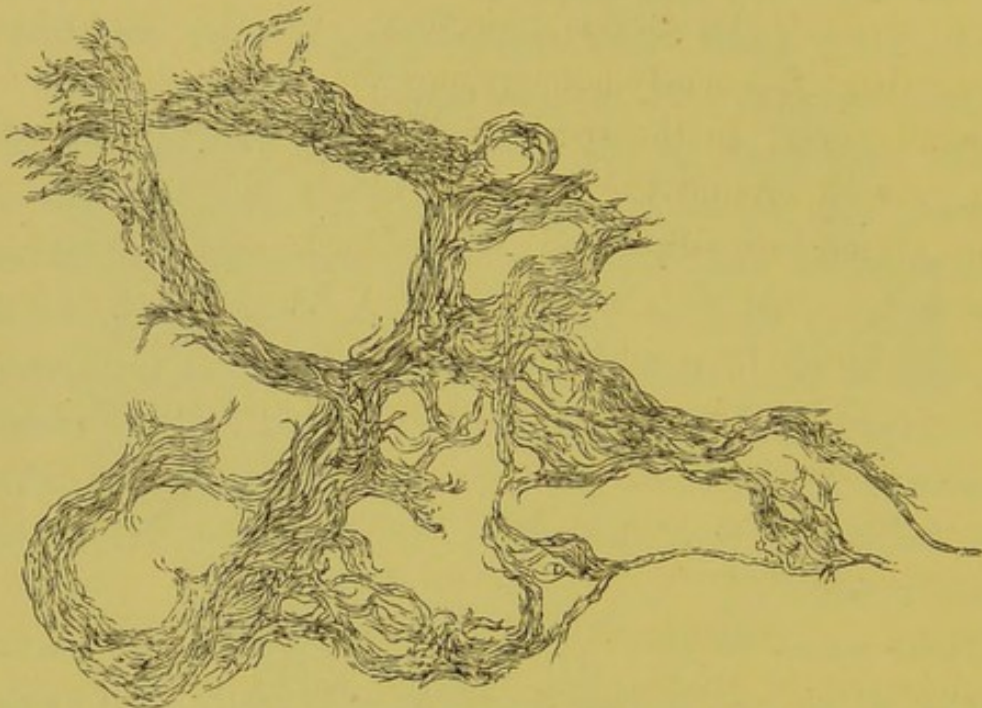referred to, there are indications visible as if the caseous

FIG. 5.—Lung tissue from the sputum in phthisis. The sputum was digested in caustic soda
according to Fenwick's method, and the sediment examined microscopically.   ×350.

matter were crumbling and separating from the surrounding
structures so as to leave cracks or fissures. It need hardly
be said that this process of softening implies *the formation
of cavities*, and that each cavity thus formed represents the
death and destruction of a certain portion of lung tissue,
usually involving a bronchus and surrounding alveoli. The
softened caseous matter is spit up, and it is well known
that the more resisting elastic tissue of the lung may be
recognised in the sputum, as shown in Figure 5.

This process of separation of the caseous matter is not accompanied by hæmorrhage, for the reason that in the preliminary caseous necrosis the blood-vessels are involved as well as the other structures. The necrosed vessels are incapable of carrying the blood, and there is consequently no blood circulating within the caseous area. Some authors regard the obstruction of the vessels as the cause of the caseation, but, as will be afterwards pointed out, this view is liable to serious objection. On the other hand hæmorrhage frequently accompanies the earlier stages of the original lesion. In the specimen illustrated in Figure 2, the lung alveoli around the bronchus nearly all contain blood. The alveoli are distended with blood, in the midst of which catarrhal cells are visible. A hæmorrhage such as this is of very frequent occurrence in this early stage, and is, perhaps, to be referred to disturbances of the circulation due to the exudation pressing upon and obstructing the vessels.

Before leaving this subject, it ought to be said that the changes described occur with very varying degrees of acuteness. Sometimes extensive tracts of lung will be almost simultaneously affected; and to the naked eye there may be little trace of the bronchial arrangements, although, under the microscope it will usually be apparent that the disease is advancing by the processes which I have described. Again, the processes of caseation and softening may rapidly follow the original inflammatory phenomena so that little interval may exist, and there may even be a softening which looks more like ordinary necrosis or sloughing than the more gradual caseous change. These all indicate differences in the acuteness, or virulence of the disease.

II. The Fibroid Form.—This form presents considerable differences both in the naked-eye and microscopic characters

to that already described.   At the *post-mortem* examination
the lung is found more firmly adherent than in the caseous
form over the affected part, which nearly always includes
the apex.   You often remove with difficulty a dense leathery
layer forming a kind of cap to the apex of the lung. (See
Figure 6, *a a.*)   On cutting into the lung you may find
cavities, but they are not usually very large, and the
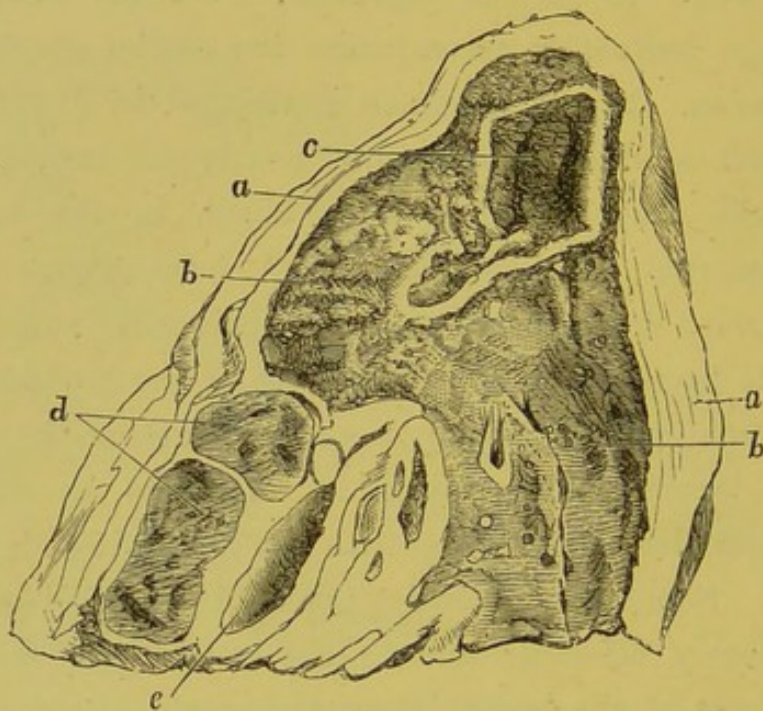internal surface is mostly clean and moderately smooth.

FIG. 6.—Upper lobe of lung in fibroid phthisis. *a a.* Greatly thickened pleura. *b.* Condensed
and pigmented tissue. *c.* A cavity with distinct lining. *d.* Bronchial glands enlarged
and pigmented. *e.* Main bronchus. The nearness of the bronchus and glands to the
apex indicates the shrinking.

The lining consists of a distinct membranous layer.   (See
Figure 6, *c.*)   The tissue outside the cavities is of a deep
slatey colour (*b b* in Figure) in which you may find here
and there opaque white spots, but these are sparsely dis-
tributed; it is also very dense.   The dense pigmented tissue
may involve a considerable portion of the lung, and the
affected part is shrunken and contracted.   Figure 6, for
example, represents of the natural size the greater part

of the upper lobe of the lung, the position of the root, with bronchus (e) and lymphatic glands (d) indicating, when we consider its proximity to the apex, the great shrinking of the tissue.

Outside the condensed parts, and in the midst of the crepitant lung tissue, you do not find in this form the rounded areas already described as characteristic of the caseous form, but instead there are hard dark bodies
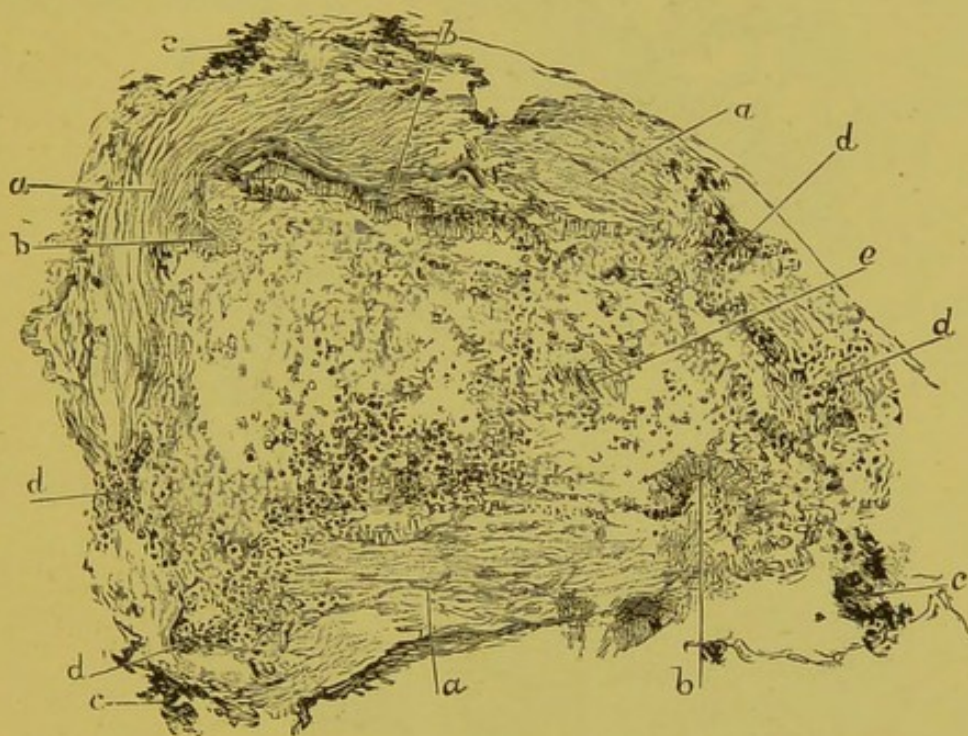

FIG. 7.—Bronchus in early stage of fibroid form. *a a.* The bronchial wall. *b b.* Projecting parts still covered with epithelium. *c c.* The pigment surrounding bronchus. *d d.* Round cells infiltrating bronchus. *e.* Round cells and epithelium in calibre. ×60.

scattered through the lung tissue. On running the finger over the cut surface of the lung we feel these bodies, and they stand out to some extent above the general level of the cut surface. These form the outposts of the disease, and it is by the examination of them that the earlier stages of the lesion are to be studied.

In these hard isolated bodies it will be found that, as in the caseous form, it is a plugged bronchus which is again

the centre of the lesion. This is shown in Figure 7, which
is from a case of this kind. The bronchus contains, as
before, inflammatory products—namely, round cells and
desquamated epithelium. The wall of the bronchus is also
infiltrated with round cells, and in the wall and in the
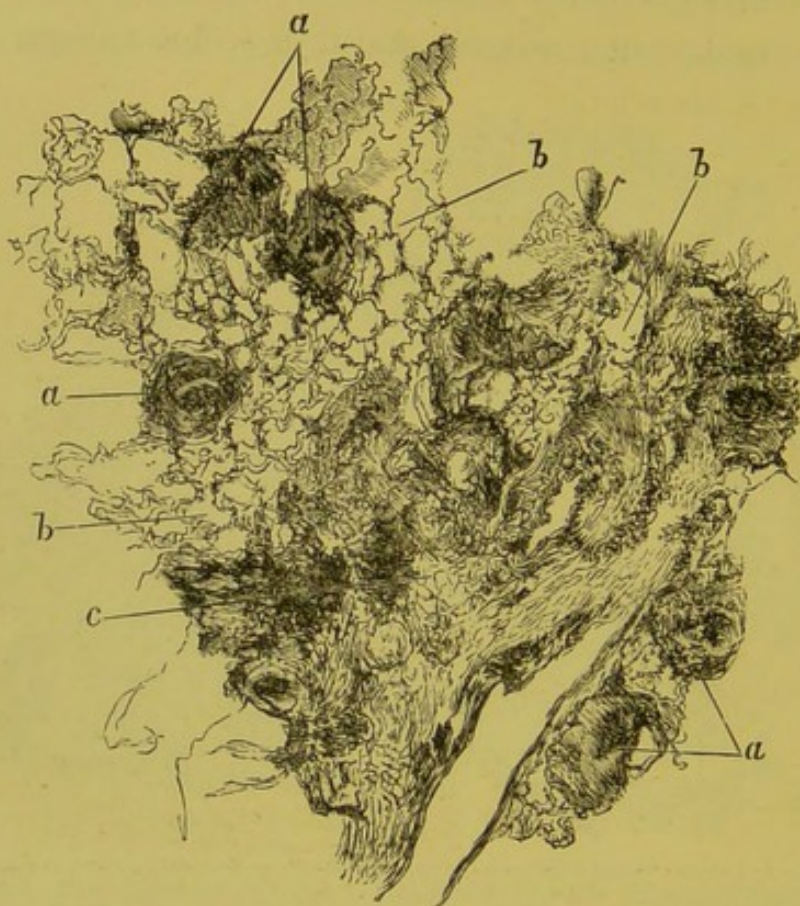connective tissue around you will commonly find tubercles.

FIG. 8.—Fibroid phthisis. *a a.* Tubercles in the connective tissue around a bronchus; the
darker centres indicate caseation. *b b.* Emphysematous lung alveoli. ×20.

There is, however, very little appearance of inflammation
in the lung alveoli; they are not filled, as in the other
form, with the products of inflammation. *Tubercles* are a
peculiarly striking feature in this form, and they are often
grouped in the neighbourhood of the bronchial tubes or
in their walls as shown in Figure 8. In the earlier periods
these tubercles present the usual structure, and there are

frequently several giant cells in the midst of the nodules.
From the arrangement of these tubercles and their relation
to the bronchial tubes, it may be inferred that many of
them have their seat of origin in the lymphatics, the
process travelling from the fine bronchi outwards into the
connective tissue of the lung by the lymphatic vessels. It
is not uncommon in this form as well as in the other to
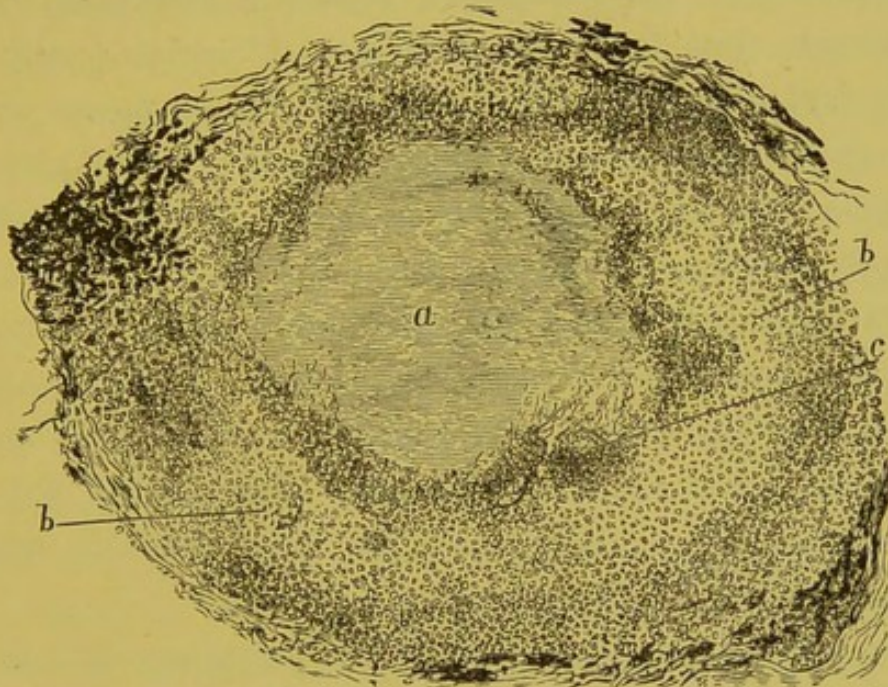find blood in the lung alveoli, and it is sometimes present in
considerable abundance.

Fig. 9.—Fibroid phthisis. *a*. Bronchus plugged and caseous. *b b*. Wall of bronchus
infiltrated with round cells and tubercles. *c*. A tubercle with giant cell.

It will be seen that in many of its details this process
closely resembles that in the other form. It begins like it
in a bronchitis, affecting the finer tubes, and it is accom-
panied by the formation of tubercles. We often find also
that the plug in the bronchus becomes caseous. This is
shown in Figure 9 which represents a bronchial tube from
a case of fibroid phthisis. In the centre (*a*) the plug has
become quite homogeneously granular from the occurrence

of caseation, while the walls of the tube are extensively infiltrated with round cells (*b b*). In some places, as at *c*, the cells are in the form of rounded aggregations or tubercles. Caseous necrosis is also frequently manifest in the tubercles as shown in Figure 8.

While presenting these features in common we find that the two forms have certain peculiarities which are sufficient to give them separate characters. One of these is that in the fibroid form, as we have just seen, the process spreads much more to the connective tissue of the lung by the lymphatics, than to the lung alveoli. Associated with this is the fact that, although caseation is by no means absent, it is quite subordinate to another change, which gives its character to the lesion. This is a process of *fibroid transformation*. It affects both the tubercles and the general connective tissue of the lung. The tubercles are converted into clear structureless bodies, in which all the elements of the tissue are lost except occasionally one or two giant cells which are partially transformed, but still recognisable, sometimes containing a considerable number of black pigment granules.

The connective tissue around the bronchus and in the neighbourhood partakes in the fibroid transformation, and the result is the formation of dense fibrous structures which tend to shrink, and in their shrinking to cause atrophy of the delicate alveolar structures.

The shrinking of the tissue, associated as it is with adhesion of the pleura, causes the drawing in of the chest wall which is sometimes such a characteristic feature in these cases. It also causes two other changes in the lung tissue which are often associated—namely, *emphysema and dilatation of the bronchial tubes.*

BRONCHIAL DILATATION or BRONCHIECTASIS is, in this form of phthisis, the most active factor in the formation of cavities, and this result is usually brought about by a process which, in some of its details has been well described by Prof. Hamilton, of Aberdeen. As the chest wall forms a comparatively fixed point to which the shrinking tissue is attached by means of the pleural adhesion, and as the tissue is also 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 BRONCHI-ECTASIS. Such cavities have for the most part well defined walls like that shown in Figure 6, c, and they are directly continuous with bronchial tubes of which they form flask-shaped dilatations. It is to be remembered that the primary process involving, as it does, the smaller bronchi, leaves all but these capable of dilatation.

While this is in many cases the process by which cavities form, it must be said that they are often met with without any such direct mechanical mode of production being dis-coverable. Bronchiectatic cavities are often seen in this form of phthisis where no connection with the chest wall by means of fibrous bands can be traced, and they may even be in the midst of crepitant lung tissue. In some cases these cavities arise by accumulation of the secretions and inflammatory products behind an occlusion of the tube. Such an occlusion may occur where the primary lesion has affected a tube of larger calibre than usual, and has left the bronchus free distal to the affected part. I am convinced that this is not infrequently the origin of cavities. I believe 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 complimentary to the shrinking which has taken place in some part of the lung. I shall have occasion farther on to refer to a case of congenital non-inflation of the lung, in which the bronchi had become converted into a series of sacs. Here the mere non-inflation, without any active disease, implying, as the chest enlarged, an excessive distensile 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.
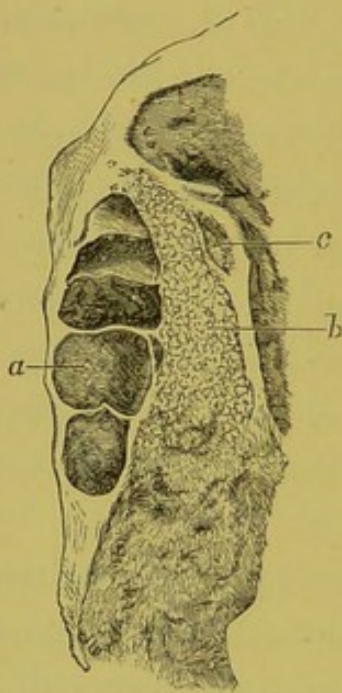
FIG. 10.—Fibroid phthisis. Effects of shrinking. *a.* Cysts in Pleura. *b.* Emphysema. *c.* Cyst in interlobular connective tissue. Natural size.

The EMPHYSEMA is undoubtedly a complimentary process, and occurs in parts of the lung which are otherwise normal. This condition is indicated in Figs. 8 and 10, *b*, and it is frequently very prominent, but without definite localisation, occurring wherever the disease has left a comparatively sound portion of lung in the midst of shrinking tissue.

Another result of this shrinking, and a somewhat unusual one, is illustrated in Figure 10, which is a drawing of a preparation in the Museum of the Western Infirmary. At the left side of the illustration, the greatly thickened pleura is seen to be the seat of a series of cyst-like cavities which were filled with serous fluid. The upper lobe of this lung was greatly contracted; and there were bronchi-

ectatic cavities as well as the emphysema and cyst-like formations shown in the illustration. Another small cyst-like space is shown at *c* in the illustration. It is in the midst of a greatly thickened interlobular septum, and it also contained serous fluid.

In this case the shrinking of the lung, dragging the adherent pleura with it, had caused spaces to form in the substance of the pleura and in the trabeculæ of connective tissue, which spaces were filled with serous fluid. The mode of production is essentially the same as that of emphysema, with which it co-existed.

There is one feature in the fibroid form of phthisis which seems to me somewhat difficult of explanation—namely, the great accumulation of black carbonaceous pigment in the affected tissue. This pigment is often present in the bronchial contents and wall, as well as in the surrounding connective tissue and tubercles, to such an extent that the scattered nodules of the earlier lesion have often a black appearance, and are manifest to the eye as black spots at intervals on the cut surface. Perhaps the explanation is that the inhaled carbonaceous matter is retained by the affected bronchi, and not swept outwards as it is normally by the cilia of the bronchial epithelium.

COMPARISON AND DISTINCTION OF THE TWO FORMS.

Before proceeding to consider any other forms of phthisis, it may be convenient to inquire what are the points of distinction between the two forms which have been under review, and what relation they bear to each other?

As an aid to this inquiry I will here introduce tables which bring out certain points in the history of such cases.

These tables have been constructed from the records already referred to in the Pathological Registers of the Western Infirmary, and as the figures have been with some labour compared with the records in the Ward Journals, they may be accepted as approximately accurate. One point which is exhibited in all these tables is, that the caseous form is much more frequent than the fibroid, being in the proportion of more than 5 to 1.

TABLE I.

AGES AT DEATH IN CASEOUS AND FIBROID FORMS OF PHTHISIS.

AGE AT DEATH.	CASEOUS.		FIBROID.	
	No. of Cases.	Per Cent.	No. of Cases.	Per Cent.
0 to 5 years,	1	0·6 ⎫	0	0 ⎫
6 ,, 10 ,,	2	1·3 ⎪	1	4 ⎪
11 ,, 15 ,,	3	2· ⎪	1	4 ⎪
16 ,, 20 ,,	22	14·75 ⎬ 58·35	2	8 ⎬ 48
21 ,, 25 ,,	23	15·5 ⎪	3	12 ⎪
26 ,, 30 ,,	36	24·2 ⎭	5	20 ⎭
31 ,, 35 ,,	16	10·75 ⎫	3	12 ⎫
36 ,, 40 ,,	22	14·75 ⎪	2	8 ⎪
41 ,, 45 ,,	11	7·4 ⎬ 41·65	2	8 ⎬ 52
46 years and upwards,	13	8·75 ⎭	6	24 ⎭
Totals,	149	100·00	25	100

In this table it appears that the ages at death are distinctly lower in the caseous form than in the fibroid. If we take 30 as a middle point we find that in the caseous

form 58 per cent died within that age, while in the fibroid form the number is 48. This is even more striking when we look at the higher ages. It appears that no less than 6 cases, constituting 24 per cent of the whole cases of the fibroid form, were over 45 years of age at the time of death, while only 9 per cent of the caseous form reached that age.

TABLE II.

DURATION OF THE DISEASE IN CASEOUS AND FIBROID PHTHISIS.

DURATION.	CASEOUS.		FIBROID. .	
	No. of Cases.	Per Cent.	No. of Cases.	Per Cent.
0 to 3 months, .	10	7·15 ⎫	1	4 ⎫
3 ,, 6 ,, . .	31	22·15 ⎬ 70·73	3	12 ⎬ 40
6 ,, 9 ,, . .	31	22·15	2	8
9 ,, 12 ,, . .	27	19·28 ⎭	4	16 ⎭
1 to 1½ years, .	10	7·15 ⎫	3	12 ⎫
1½ ,, 2 ,, . .	12	8·58	4	16
2 ,, 3 ,, . .	8	5·7 ⎬ 29·27	1	4 ⎬ 60
3 ,, 4 ,, . .	5	3·57	1	4
4 ,, 5 ,, . .	1	0·7	1	4
Over 5 ,, . .	5	3·57 ⎭	5	20 ⎭
Totals, . .	140	100·00	25	100

Still more striking is the difference in the duration of the disease in the two forms. The average duration (see Table III) in the caseous form was found to be in 140 cases 11·87 months, or less than a year, while, in the fibroid form, the average duration in 25 cases was 35 months, or nearly

three years. A more correct statement, however, is to be obtained by a comparison of the numbers dying at certain periods, as shown in Table II.*

It will be seen from this that in the caseous form over 70 per cent died within a year of the onset, whereas only 40 per cent of the fibroid cases did so. This result is even under the mark, because the caseous form, being usually acute, has a much more marked onset than the fibroid form, whose commencement is very often insidious and without obvious symptoms.

Another fact of interest which is shown also in Table III, is the much larger proportion of males in fibroid phthisis than in the caseous form. In the latter there were 106 males to 48 females, or in the proportion of about 69 per cent to 31, whereas, in the fibroid form there were 22 males to 4 females, or in the proportion of about 84 to 16.

TABLE III.

	No. of Cases.	Males.	Females.	Average Age.	Average Duration.
Caseous form,	155	106	48	29·08	11·87 months.
Fibroid ,,	27	22	4	30·25	35·08 ,,

These figures show a large excess of males in both forms; but this is in itself merely to be regarded as an indication of the fact that more male cases of phthisis are admitted to the Western Infirmary than female. Through the kindness of Dr. Russell, the Superintendent of the Infirmary, I have been supplied with a table showing the number of patients admitted with phthisis during a single year, with the ages and sexes.

* There may appear to be some discrepancy in the numbers given in this table and elsewhere. The explanation is that in the total list of cases there were a few instances in which one of the items, such as the age or duration of the disease could not be ascertained.

TABLE IV.

SHOWING CASES OF PHTHISIS PULMONALIS TREATED IN THE WESTERN INFIRMARY FROM 1st NOVEMBER, 1885 to 31st OCTOBER 1886.

Age.	Males.	Per Cent.	Females.	Per Cent.
Under 10 years of age, . .	3	1·5 ⎫	2	2·6 ⎫
Over 10 and under 15, . .	10	5·	3	4·
,, 15 ,, 20, . .	21	10·5 ⎬50	16	20·5 ⎬65·2
,, 20 ,, 25, . .	29	15·	16	20·5
,, 25 ,, 30, . .	35	18· ⎭	14	18· ⎭
,, 30 ,, 35, . .	20	10· ⎫	14	18· ⎫
,, 35 ,, 40, . .	26	13·5	6	7·7
,, 40 ,, 45, . .	28	14·5 ⎬50	4	5·2 ⎬34·9
,, 45 ,, 50, . .	11	6·	3	4·
,, 50 years and upwards, .	11	6· ⎭		⎭
Total, . .	194		78	

This table is interesting in various ways. In the first place it shows that the admissions to the Western Infirmary are nearly in the same proportion as regards the sexes as the *post-mortem* examinations. There were 194 males and 78 females admitted during the year; and there were 128 males and 52 females in the *post-mortem* records which are here used. Again, it is consistent with the facts already indicated in regard to the greater proclivity of the females to the caseous form, that the ages of the female patients are so much lower than those of the males. Taking, as in Table I, 30 as a middle point, we find that of the females 64·8 per cent were under that age, while of the males exactly 50 per cent were so.

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A point which we shall find of importance in our further inquiry as to these two forms of phthisis is, the frequency with which tubercular ulcers of the intestine are associated with them. This is exhibited in Table V. In this Table the numbers are given in which ulcers were actually noted as present; and in another column the numbers in which it is specifically noted that there were no ulcers; and again, a third column in which no mention is made of their presence or absence. As the intestine is regularly examined in cases of phthisis, it may be inferred that in nearly all the latter cases no ulcers were present, and they may be included together with the cases in which there is evidence that none were present.

TABLE V.
TUBERCULAR ULCERS IN THE TWO FORMS OF PHTHISIS.

	No. of Cases.	Ulcers Present.	None.	Not Noted.	Together.
Caseous,	155	78	46	31	77
Fibroid,	27	13	7	7	14

It will be seen from this Table that in both forms ulcers were actually present in about half the cases, so that in this regard the two forms stand on a very similar footing—a result which I confess I did not expect, but which is of considerable importance in view of what is to follow.

We may now renew our inquiry as to the essential distinction between these two forms. They have certain points in common—viz., that each begins with a bronchitis of the finer tubes, and that in each tubercles are characteristically present. In the one form, however, the bronchial inflammation extends to the proper parenchyma of the lung, constituting a lobular broncho - pneumonia, whereas in the other, the inflammation is more localised

in the connective tissue around the inflamed bronchi, constituting a bronchitis with peri-bronchitis. There is the further distinction that in the one form caseous necrosis is characteristic, and forms a regular stage in the process; whereas in the other, while probably present in most cases, it is limited in extent, and may be confined to the contents of the bronchi and the bronchial wall.

It is evident that both forms are tubercular, and that it is not a question here of tubercular and non-tubercular phthisis. This is singularly confirmed by the equal frequency of tuberculosis of the intestine in both forms. If we recognise tuberculosis as a special form of disease, then both of these must be regarded as merely different phases of a local tuberculosis of the lungs.

The conclusion I have come to is, that the difference is determined by the individual proclivities of the patients. It seems as if in the case of fibroid phthisis there were inherent in the person a much greater power of resistance to tubercular disease of the lungs—the greater age, the longer duration, the greater frequency in the male sex, all seem to point to greater powers of resistance. Another fact is that, as we shall see farther on, the process in fibroid phthisis in many ways resembles that which is concerned in the cure of phthisis, the most important difference being that in the former case there is the fatal complication with active tuberculosis. We may, indeed, regard the fibroid changes as probably to a large extent the expression of a prolonged process of resistance on the part of the living tissues. In the caseous form the tissues are directly killed by the progress of the disease, and sometimes with great rapidity. In the fibroid process there is a long struggle, and no palpable softening or destruction of the tissue by the direct action of the morbid agent.

It is proper to add that these two forms are by no means to be absolutely distinguished. They run into one another to a certain extent, and, especially in cases where partial recovery takes place, the caseous form may assume many of the characters of the fibroid, although it must be borne in mind that the points of distinction are usually manifest from beginning to end of the lesion.

CLINICAL ILLUSTRATIONS.

It may be useful at this stage to illustrate from actual cases in the wards some of the clinical differences of these two forms, and it happens that in Dr. Gairdner's wards, which are at present under my care, there are seven cases of phthisis to which I have to ask your attention, and which we may have the opportunity of examining after the lecture.

In transferring our attention from the pathological to the clinical aspects of these cases, I may briefly indicate the points of difference which they may be expected to present. In caseous phthisis we have a more rapid course, probably accompanied by fever, we have evidences of softening, the more rapid formation of cavities, more abundant expectoration, the greater tendency to emaciation, and the frequent occurrence of pleurisy and pneumo-thorax, whose relation to the pathology of this condition will be discussed afterwards.

In the fibroid form, on the other hand, we have the slow course probably without much fever, the probable absence of signs of softening, and possibly of any râles whatever except in connection with cavities, the slow formation of cavities, and the much less tendency to emaciation. Hæmoptysis occurs in both forms.

There are at present four cases of phthisis in the female ward, No. 6, and all of them present the characters of the caseous form.

I shall give a brief summary of each.

CASE I.—Mary M., aged 22, was ill for a month on admission, and as that is six weeks ago, she has been ill altogether 2½ months. Ever since admission, the temperatures have been highly febrile, mounting as high as 105·8°, and emaciation is extreme. For some time she has been almost moribund, and latterly hæmoptysis has occurred. Physical examination shows extensive dulness on the right side, both in front and behind, with abundant crackling râles and distinct evidences of cavity. Over the left side there is dulness at the apex, and râles are audible at the back.

This is a very acute case, and might be classed as pneumonic phthisis.

CASE II.—Susan F., aged 19, a millworker, and sometimes a farm servant, was ill for six months on admission, which was a week ago. Her illness began with cough, but she was not laid up till a few days before admission, when an attack of acute pleurisy caused her to cease work at the harvest field where she had been employed. Hæmoptysis occurred a fortnight before admission to the extent of a teacupful. There is a good deal of fever, the temperature going up to 103·8°. There are evidences of pleurisy on the right side—namely, percussion dull over the whole back and the lower part of the front, respiratory murmur deficient over the dull area, and vocal resonance diminished and slightly ægophonic. Abundant crackling râles are present at the right apex, but there are no distinct evidences

of cavity. There is some dulness and a few râles at the left apex.

Here we have a less acute case with a duration already of six months without, as yet, extreme emaciation.

CASE III.—Eliza C., a dressmaker, aged 16, dates her illness nine months back, when it began with a bad cold. She is much emaciated, and the temperatures are distinctly febrile, but not so high as in the two preceding cases. There is marked dulness at the right apex, with signs of cavity above and below the clavicle—namely, blowing and hollow respiratory murmur, tinkling and metallic râles, and whispered pectoriloquy. There are abundant râles throughout both sides.

This case is less acute, but there are distinct signs of softening, and cavities have already formed.

CASE IV.—Catherine C., factory girl, aged 18, was in the ward last year in June and July, affected with "pleurisy with effusion," and went out greatly improved, but with a suspicion of disease at the apices. The present illness began with cough nine months ago, and at that time the catamenia ceased. There was an aggravation of the symptoms three months ago, when hæmoptysis occurred. Fever has been slight. There is somewhat extensive dulness over the right side, especially behind, with abundant crackling râles, but no obvious signs of cavity. The hepatic dulness is enlarged; diarrhœa has been present for weeks, and there is excess of urine, but without albumen—specific gravity, 1010.

This is quite a chronic case, although probably still of the caseous form. It began apparently with acute pleurisy, and although there are distinct evidences of softening,

cavities have not formed after more than a year's duration. The diarrhœa, enlarged liver, and excess of urine, suggest amyloid disease.

In the male ward there are three cases.

CASE V.—David S., a draper, aged 19, has been ill for two or three months, his illness having a definite origin in "cold." Soon after the onset he was attacked by acute pain on the left side which a doctor diagnosed as pleurisy. At present there is considerable dulness at right apex with tubular breathing and crackling râles, but no definitely cavernous phenomena. Friction is audible over a large part of the left side. There is here distinct fever. Spitting of blood is reported to have occurred a year before present illness.

This is also a case of caseous phthisis—somewhat acute but not yet advanced to the formation of cavities.

CASE VI.—John R., a carter, aged 56, is a very massively built man. His strength has been failing for eighteen months, but cough and hæmoptysis began together twelve months ago. He has been subject to looseness of the bowels eighteen years, but this has been worse for the last eighteen months. There is not much emaciation, but the nails are curved, and the fingers clubbed. Percussion is extensively altered on both sides, and there is cavernous respiratory murmur and vocal resonance at left apex, both in front and behind, but without râles. There are few râles anywhere.

This is probably a case of fibroid phthisis. There are few signs of softening anywhere, even near the cavities. The next case is more typically fibroid.

CASE VII.—James S., a labourer, aged 35, traces his illness to exposure to extremes of heat and cold, and the inhalation of coal dust and sulphurous fumes, the symptoms beginning five years ago. Spitting of blood in small quantities was an early symptom, and he put up blood in large quantities two years ago when in the Edinburgh Royal Infirmary. After this he improved greatly. There is considerable dulness at the right apex, and here the respiratory murmur is tubular and suggestive of cavity, especially over the inner part of the spine of the scapula. There is almost no râle anywhere. The temperature has been almost normal, the only exception being a slight rise just after admission, perhaps traceable to a hæmoptysis which had just occurred.

LECTURE II.

CONDITIONS ALLIED OR ANALOGOUS TO PHTHISIS—CAUSATION OF PHTHISIS.

Allied or analogous conditions, *(1) Syphilitic disease of the lung— syphilitic phthisis. (2) Glanders and actinomycosis. (3) Conditions induced by foreign bodies in the larger bronchi. (4) Gangrene of the lungs. (5) Chronic pneumonia. (6) Diseases due to the inhalation of dust—Potter's phthisis, &c.*

Causation of phthisis. *Caseous necrosis the central fact; causes of progressive molecular necrosis in general; relations of phenomena in tubercular ulcer of intestine, and solitary tubercle of brain; the caseous necrosis not from non-vascularity, but from action of morbid irritant. Case of inoculation of lung from a caseating bronchial gland; probable inoculation from tubercular larynx.*

THE first subject which will occupy our attention to-day is one concerning which considerable differences of opinion have existed: we have to consider whether there are any other forms of disease besides those considered in last lecture which ought to be included under the term phthisis pulmonalis, and more particularly whether there is a non-tubercular form or forms distinguishable from those we have considered, and which we have found to be tubercular. It may be said at once that the great bulk of cases fall under one or other of the forms considered in last lecture, and as both of these forms were shown to be tubercular, there remains no doubt in my own mind that in the great majority of cases the disease is a local tuberculosis conforming to one or other of these types.

While this is the case there are certain conditions of the lungs which may be regarded as falling under our definition of phthisis, at least in some of their aspects, and it will be necessary to pass these under review, in order that they may be distinctly separated from the more typical forms already considered. It will probably be expedient to limit the term phthisis pulmonalis to true cases of tuberculosis, but in order to such a limitation we must understand the pathology of allied or analogous conditions. With this object in view we shall now briefly consider each of the following conditions:—(1) Syphilitic disease of the lung; (2) Glanders and actinomycosis; (3) Conditions induced by foreign bodies in the larger bronchi; (4) Gangrene of the lung; (5) Chronic pneumonia; (6) Diseases due to the inhalation of dust.

I.—SYPHILITIC DISEASE OF THE LUNG.

Syphilis and tuberculosis are now generally regarded by pathologists as belonging to the same class of diseases. They are ranked among the Infective Tumours. In syphilis as in tuberculosis we have two chief forms of lesion—namely, one having the characters simply of inflammation, and the other a more definite new formation in some respects comparable with the specific tubercle, although not difficult of distinction from it. The inflammatory lesions in syphilis consist chiefly in the formation of granulation tissue, which tends to develop into connective tissue, as does ordinary granulation tissue. The syphilitic gumma or tumour, on the other hand, is also composed at first of granulation tissue, but forms a more distinct mass, having many of the characters of a tumour. It does not to any considerable extent cicatrise, but rather tends to undergo caseous necrosis,

or some form of softening. As the inflammatory condition and the gumma are often present together, the latter is usually surrounded by cicatricial tissue. The resulting appearances are frequently seen characteristically in the liver, where deep cicatrices are met with, in the midst of which the definite gummatous tumours are found.

It is perhaps remarkable that while syphilitic lesions are so frequently present in the liver, they are rarely seen in the lungs. It is usual to describe, under the designation "white pneumonia," a syphilitic condition in which considerable tracts of lung tissue are condensed and infiltrated, the tissue having a dead white colour. This condition, however, is only met with, so far as I can learn, in newly born children, and therefore, it scarcely concerns us here as it is not liable to be mistaken for phthisis in the ordinary sense.

Syphilitic disease of the lung is so rare in the adult that I may be allowed here to quote somewhat in detail the record of two cases, in one of which at least no doubt could exist as to the nature of the case.

The first case is that of a man, aged 30, who had contracted syphilis five years before death. He complained of cough with slight blood-stained expectoration for four years, and, in some of its clinical features, the case resembled one of phthisis. The body after death presented, especially in the condition of the liver, quite definite evidence of syphilis. There were numerous cicatrices in the liver, in the midst of which definite yellow tumours, evidently caseating gummata, frequently occurred. The lungs were firmly adherent and they also, like the liver, presented cicatrices penetrating into their substance, but these were much less frequent than those in the liver. When these cicatrices were cut into, there were found in the midst of them small

yellow gummata similar to those in the liver. In this case the affection was comparatively slight, but it presented the typical lesions of syphilis—namely, fibroid new formation with caseating gummata. This patient died apparently from the results of amyloid disease which affected the liver, spleen, kidneys, &c.

In the other case there was no history of syphilis, but as the man was a seaman there may perhaps be rather a presumption in favour of its having been present. There were here large cavities in the upper lobe of one of the lungs, but the cavities were surrounded by cicatrices which had not to my eye the appearances of those in fibroid phthisis, being much more distinctly localised. These cicatrices were remarkable in respect that they were immediately surrounded by normal lung tissue, so that areas of fibrous tissue occurred, in the midst of which there was often a dilated bronchus in immediate relation to normal tissue. There was marked amyloid disease of the liver and kidneys, but not of the spleen.

It must be said that in this instance the syphilitic nature of the case was doubtful, but the following reasons written down at the time induced me to regard it as such:—(1) The remarkable tendency to an interstitial inflammation of a fibrous character. (2) The occurrence of this in isolated areas in both lungs, there being sound tissue between. (3) The absence of the commoner appearances of phthisis, as well as of the usual localisation. (4) The existence of extreme amyloid disease of the liver and kidneys.

It will be seen from these two cases that it is the fibroid form of phthisis which, if at all, syphilis imitates.

II.—ACTINOMYCOSIS, GLANDERS.

These belong to the same group of diseases as tuberculosis and syphilis. They also occasionally produce lesions having a resemblance to phthisis, and as they cause destruction of lung tissue of an advancing or spreading character, they fall strictly under our definition of phthisis. As I have not met with a case in the human subject in which either of these two conditions has been present in the lungs, I shall not refer to them here with any detail.

Actinomycosis is a disease due to the action of a special parasitic micro-organism, the actinomyces or ray-fungus. It occurs chiefly in cattle, where it generally gives rise to bulky tumours, usually situated in the neighbourhood of the mouth or jaws. These tumours consist of granulation tissue, in the midst of which there are usually suppurating centres, and they are sometimes permeated by fistulous canals. The disease has been identified in man in not a few cases, and according to Israel, who in 1885 had already collected 38 recorded cases, the primary seat may be either the mouth and pharynx, the lungs, or the intestine. When the lungs are the primary seat of the disease, it may somewhat closely resemble phthisis. The fungus reaching the lungs by the bronchial tubes produces its effects first in limited areas, of which the bronchi are the centres. These effects are inflammatory, consisting in new formation of granulation tissue around the bronchi, and exudation into the lung alveoli. There is indeed a broncho-pneumonia which in many respects resembles that of tuberculosis. There is not in actinomycosis a true caseous necrosis, but rather a more direct softening, with fatty degeneration and suppuration. In this way cavities may arise and attain considerable dimensions, although they are sometimes absent.

By the coalescence of various centres the disease may come to involve a large part of the lung, and as a result there may be a great new-formation of connective tissue, causing extensive fibrous condensation with shrinking of the lung tissue. While this is the case in the older parts, the disease may be advancing at the periphery. The disease has considerable tendency to extend beyond the lung to the pleura, the muscles of the chest and the skin, to the pericardium, diaphragm, and so on.

The micro-organism occurs in the form of minute masses, consisting of threads which have a radiating arrangement. To the naked eye they have a saffron-yellow colour, and are sometimes visible in the debris of cavities. They have been distinguished in the sputum of persons suffering from this form of disease.

Glanders may be communicated from horses to man, but it usually has its seat in the nose and its communicating cavities, or in the larynx and trachea, where it leads to acute inflammations, usually accompanied by suppuration. It may attack the lungs, but not in such a form as to lead to any confusion with phthisis pulmonalis.

III.—FOREIGN BODIES IN THE LARGER BRONCHI.

In its clinical aspects the condition brought about by the presence of foreign bodies in the larger bronchial tubes presents, in many cases, a rather confusing resemblance to phthisis pulmonalis. The lesions so produced come, strictly speaking, within the definition which I have given of phthisis, and yet there are such important differences that the whole subject appears to me to warrant the most careful attention.

When a foreign body of some size passes down the trachea and lodges in one of the larger bronchi at the

roots of the lungs, it becomes a centre to which lesions extending to the lung generally, may be traced. The foreign body irritates the bronchial wall, making for itself a cavity whose internal wall is ulcerated and discharges pus. The discharge so produced stagnates in the cavity and decomposes, and as it is in direct communication with the bronchial tree it may be insufflated into the lung, producing on account of its irritating characters inflammatory lesions in many situations. These secondary lesions have bronchial tubes for their centres and usually result in dilatation of these, so that cavities arise. These lesions are found chiefly in the one lung, but may be present to a limited extent in the opposite one. Cases of this kind present very varying degrees of acuteness according to circumstances. If the foreign body be such as to cause great irritation, and especially if it be in itself decomposible, then we may have a very acute course, while, if the body be physically inert and chemically innocuous, it may produce its effects very slowly. These different results are well illustrated by two cases which occurred to myself, and which I shall relate with some fulness.

The first case, that of J. F., aged 23, was seen by me about ten years ago. He was found to be suffering from pulmonary symptoms, the origin of which was not known for some time. It was, however, elicited that before the onset of his illness he had been eating some mutton broth when he choked upon a piece of bone which was stopped at the top of the wind-pipe. He was immediately in a state of great distress, his face became livid, and he appeared to be choking. With a great effort at coughing, and with the aid of the finger, the bone was carried downwards, and he afterwards described that he felt it go down and down in his chest till it stopped.

Pulmonary symptoms began to develop almost immediately, and the signs of disease were confined for almost the entire illness to the left lung, where in the course of a few months there was evidence of the existence of cavities in every part. Throughout the illness the sputum had a mawkish odour, and it was frequently expectorated in large quantities, a very fluid purulent matter coming out through nose and mouth at once. The course of the disease was marked by progressive emaciation. At first, when signs of softening and excavation developed so rapidly in the left lung, the case was looked upon as one of acute phthisis; but as the patient survived for seventeen months, and the right lung remained practically free, this diagnosis was latterly given up, and the presence of a foreign body was suspected from the account given as above, without, however, any confident assertion of such a diagnosis.

After death, the chest only was examined. The left lung was found firmly adherent in every part, especially behind and towards the apex. The adhesions were exceptionally dense, so that, although the lung tissue was itself very firm, and could be dragged upon with considerable force, yet the adhesions could not be separated without cutting. The lung tissue generally was exceedingly tough and leathery, and it was permeated in every region, but especially in the central parts, by cavities, consisting obviously of dilated bronchi. These were small for the most part, few of them being larger in size than a walnut. There were no localised condensations in this lung, the leathery condition being homogeneously distributed. The right lung was absolutely non-adherent, and extended slightly across the middle line; the heart also being somewhat displaced to the left. At two places, one

at the anterior margin, and another in the midst of this lung, there was a small cavity surrounded by condensed and pigmented tissue.

The source of the mischief was found in the left main bronchus. It consisted of a piece of mutton bone, comprising the lateral half of a vertebra, and measuring about $\frac{7}{8}$ of an inch along the spine, and about $\frac{1}{2}$ an inch in thickness. It lay comparatively loose in the bronchus, whose mucous membrane was ulcerated and calibre dilated.

The second case, which was also of a very instructive character, occurred in the Western Infirmary, and is fully recorded by Professor Gairdner in the *Glasgow Medical Journal* for January, 1886. As the case was admitted while I was in charge of the Ward, and as I made the *post-mortem* examination, it impressed me considerably in view of the present subject.

The patient was a boy, aged 11 years, who on admission presented great retraction of the left side, causing marked deformity. The case was regarded as one of gangrene of the lung, or fibroid phthisis, and there was distinct evidence of the existence of cavities, the principal ones being near the angle of the scapula. The other lung was suspected, but without definite evidence of active disease. There was latterly slight fever, and trivial hæmoptysis. He expectorated large quantities of thin pus, which had a distinctly fœtid odour. The history pointed to an inflammation of the lung 8 or 9 years before, which was recovered from. Another attack occurred 5 years before admission, since which time the principal symptoms had been continuous. The sputum was examined for tubercular bacilli and for lung tissue, but none were found.

I need not enter here into the further details of this case, as they concern chiefly the question of surgical interference

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in cases of pulmonary cavity, and they have been fully discussed in Prof. Gairdner's paper. An operation was performed in order to open the cavity, but the patient a few days afterwards succumbed.

A most unexpected result of the *post-mortem* was the discovery of a foreign body, which I now show you. It consists apparently of the barrel part of a metallic crow-quill pen, the writing portion having been broken off. It thus forms a split iron tube, $\frac{3}{4}$ of an inch in length, and $\frac{1}{8}$ of an inch in diameter. It is smooth on the surface, and shows little appearance of oxidation. This foreign body was present in either a cavity or a bronchus, but its exact locality could not be determined, as it was displaced by the knife in making the first incision, its
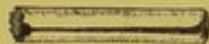
FIG. 11—Iron tube referred to in text.

existence not having been previously suspected. Otherwise the lung presented very much the appearances met with in fibroid phthisis, with the important exception that there were no isolated lesions. The lower lobe was converted into a congeries of bronchiectatic cavities separated by firm fibrous tissue, no trace of proper pulmonary tissue remaining. There was a large cavity in the upper lobe of this lung, which was not bronchiectatic in character, but probably originated by gangrene. There was also a small cavity in the upper lobe of the other lung of a similar character.

A very striking and unlooked for fact in this case was the entire absence of pleural adhesions over the greater part of the left lung. There was indeed no adhesion except at the apex, although the fibroid condensation and cavities

existed chiefly in the lower lobe. This fact was a very awkward one at the operation during life, and it was fully confirmed by the *post-mortem* examination.

This is a case in which the presence of a foreign body caused a condition closely allied, both in its clinical features and *post-mortem* appearances, to *chronic fibroid phthisis*. It may, perhaps, be inferred that the character of the foreign body had to do with the very chronic course. It was a piece of iron tube which would be chemically innocuous. In the other case, which had a much more acute course, the foreign body was a piece of bone with meat on it, and decomposition would readily lead to the evolution of irritating products.

When we come to study the causation of phthisis, these cases will be of some importance; meanwhile, it is interesting that acute phthisis on the one hand, and chronic phthisis on the other, may be somewhat closely imitated by conditions brought about by the presence of foreign bodies in the bronchi.

IV.—GANGRENE OF THE LUNGS.

. This is another condition which sometimes presents features resembling those of pulmonary phthisis. If a limited portion of lung dies the result is the decomposition and gradual softening of the lung tissue with the ultimate formation of a cavity. This in itself may lead to many of the symptoms of phthisis, but the resemblance is often made much closer by a further process resembling that which I have referred to in speaking of foreign bodies in the larger bronchi. The dead piece of tissue by decomposing becomes a centre of irritation. It induces inflammation around it, and the resulting discharge

passing into the cavity which is formed, undergoes decomposition. These irritating products are liable to be carried by the bronchial tubes, and they form in turn secondary foci of inflammation, which often result in the formation of cavities. The local inflammations which are thus produced have, as in the case of phthisis pulmonalis, bronchial tubes as their centres, the lung alveoli immediately around being filled with inflammatory products, and so condensed. These localised condensations, therefore, frequently resemble in shape those of phthisis pulmonalis, having a similar berry-like outline. Indeed in a case which occurred to me lately, it was at first a question whether it was not a case of phthisis with gangrene supervening. The analogy between the two conditions will again come up for consideration in speaking of the causation of phthisis.

V.—CHRONIC PNEUMONIA.

This name is applied—perhaps in some cases rather vaguely—to a condition in which, without any specific disease, the lung tissue is greatly altered by the existence of a prolonged chronic inflammation. It mostly occurs when an ordinary acute pneumonia, instead of resolving, passes into a chronic inflammation. It is said that pneumonia in drunkards, and in debilitated persons generally, is prone to become chronic, especially when it affects the apex of the lung.

The occurrence of this form of disease as a result of acute pneumonia has been questioned by no less an authority than Dr. Wilks. In support of it, however, I am able to cite a case in which the clinical evidences of acute pneumonia seem to have been unequivocal. This case may

be added to those given by Dr. Hilton Fagge in support of the same position.

The case was that of a man, aged 62, who was under the care of Professor Gairdner, in the Western Infirmary, with acute pneumonia in the upper lobe of the right lung. The disease gradually extended to the lower lobe, and persisted there while partially resolving in the upper lobe. Death occurred from semi-asphyxia.
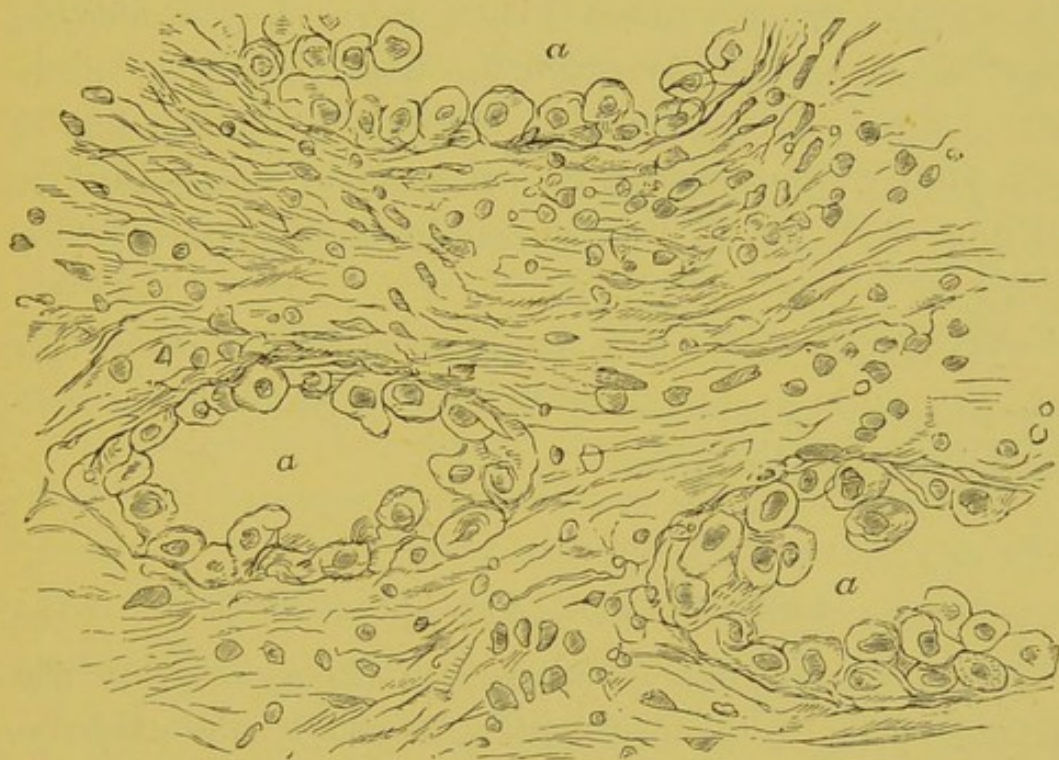
FIG. 12.—Chronic pneumonia. The connective tissue is greatly increased and the alveoli (*a a a*) are represented by contracted spaces lined with bulky epithelium. × 350.

At the *post-mortem* the body was found much emaciated; the right lung was condensed in every part except at the anterior portion of the upper lobe. The pleura was adherent, but not greatly thickened. The lung, as a whole, was bulky and solid, presenting externally much the appearance of a hepatised lung. On section it was found to be much tougher than a hepatised lung, and the colour was iron-grey, while the cut surface was smooth and rather glistening. There was no proper formation of cavities, but the bronchial

tubes presented a cylindrical dilatation. The lung was very heavy.

In this bulky lung there were almost no alveoli containing air. The accompanying illustration (Figure 12) shows the appearances as seen under the microscope. It will be seen that there is enormous increase of the connective tissue of the lung, the alveoli being in consequence crushed together and reduced to gland-like spaces with a lining of bulky epithelium. When it is considered that the lung, as a whole, was unduly bulky, and that there was virtually no air in the alveoli, some estimate may be had of the amount of new formed connective tissue. In fact the appearances are such as to make the tissue scarcely recognisable as lung tissue.

This disease, in the later stages of its clinical course may resemble phthisis pulmonalis, but pathologically it has sufficient points of distinction.

VI.—DISEASES DUE TO INHALATION OF DUST.

It has been matter of observation for many years that persons living in a dust-laden atmosphere are subject to disease of the lungs, and the names applied in certain cases to the diseases so produced sufficiently indicate their resemblance to if not identity with that which is the subject of discourse here. We are familiar with the expressions coal-miner's lung, potter's phthisis, knife-grinder's consumption, &c.

I need scarcely remind you that in the adult lung we always find more or less black pigment, so that it is customary to speak of this as a normal constituent of the lung tissue. It is not strictly normal, however, as evidenced by the fact that it is absent from the lungs of young

children, and from those of animals which live an out-door life. This pigment is in fact the dust which floats in the air, inhaled into the lungs and carried into their tissue. The lung alveoli seem to be exceedingly absorbent, so that gases, liquids, or finely divided solids, if they penetrate through the bronchi to their utmost terminations and pass into the alveoli, are taken up and carried into the tissue of the lung. All kinds of solid particles are in this way carried into the lung, and, passing from the alveoli, they find their way into the lymphatic channels. The black pigment of the lung is intimately connected with the lymphatic system, and as this system is contained in the connective tissue of the lung, the black pigment is also there. Persons living in dusty atmospheres of course inhale more foreign matter than others do, and it is scarcely remarkable that their lungs suffer in consequence.

It can hardly be regarded now as matter of controversy that the carbonaceous pigment is really inhaled and not formed in the body. As recently as 1871, however, I find that Ross [*] in discussing the black pigment which is present in the potter's lung, indicates its possible origin in carbon formed in the body and imperfectly burned. This view prevailed in regard to the normal pigment in the adult lungs up to the beginning of the present century. In 1813 Pearson, in a paper contributed to the Royal Society of Edinburgh, held that the black pigment is introduced with the air. In 1831 Gregory, of Edinburgh,[†] and in 1834 Hamilton, of Falkirk,[‡] published cases and held that the dust came from without. Chemical analysis of the lungs by Christison in Edinburgh and Graham in Glasgow confirmed this view. These observers,

[*] Ross, *Dublin Quarterly*, vol. li, 1871, p. 93.
[†] *Edin. Med. Jour.*, vol. xxxvi, p. 389.
[‡] *Edin. Med. Jour.*, vol. xlii, p. 297.

by the chemical manipulation of the black matter found in coal miners' lungs, concluded that this black matter was virtually charcoal. Christison identified it with coal, and produced a gas "which had the odour of coal gas and on the approach of a light took fire and burned with a dense white flame." Graham* on the other hand, from his analyses which were made on a number of different lungs, some of them sent by Dr. Hamilton, and some by other physicians, concluded that the carbonaceous matter was rather lamp black, and arose from the ill-trimmed oil lamps the smoke from which loads the atmosphere in ill-ventilated mines.

The observation by Zenker † of red dust in the lung tissue in persons working with the red oxide of iron, definitely proved that particles of pigment find their way into the lungs and pass beyond the bronchi and alveoli into the connective tissue. This whole subject has been very fully worked out by Dr. Greenhow in a series of communications to the *Pathological Transactions*, extending from 1866 to 1869. In the last of these papers he enters somewhat fully into the subject, and concludes that the resulting disease of the lungs is somewhat similar, whatever the kind of dust inhaled, except that the heavier and more penetrating kinds, such as angular particles, more readily excite serious disease than the lighter kinds.

I am able to show you an example of a potter's lung, in which the disease has been set up by the inhalation of the fine dust which lies about so abundantly, especially in the pressing rooms, in potteries. (See Figure 13.)

The changes in this case, which I am able to illustrate by

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* Graham was at this time Professor of Chemistry in Anderson's University, and member of the Faculty of Physicians and Surgeons, Glasgow. He afterwards became Master of the Mint.

† *Deutsches Archiv für Klin. Med.*, vol. ii, p. 116.

specimens under the microscope, quite coincide with those
described by Greenhow and others, and may be taken as
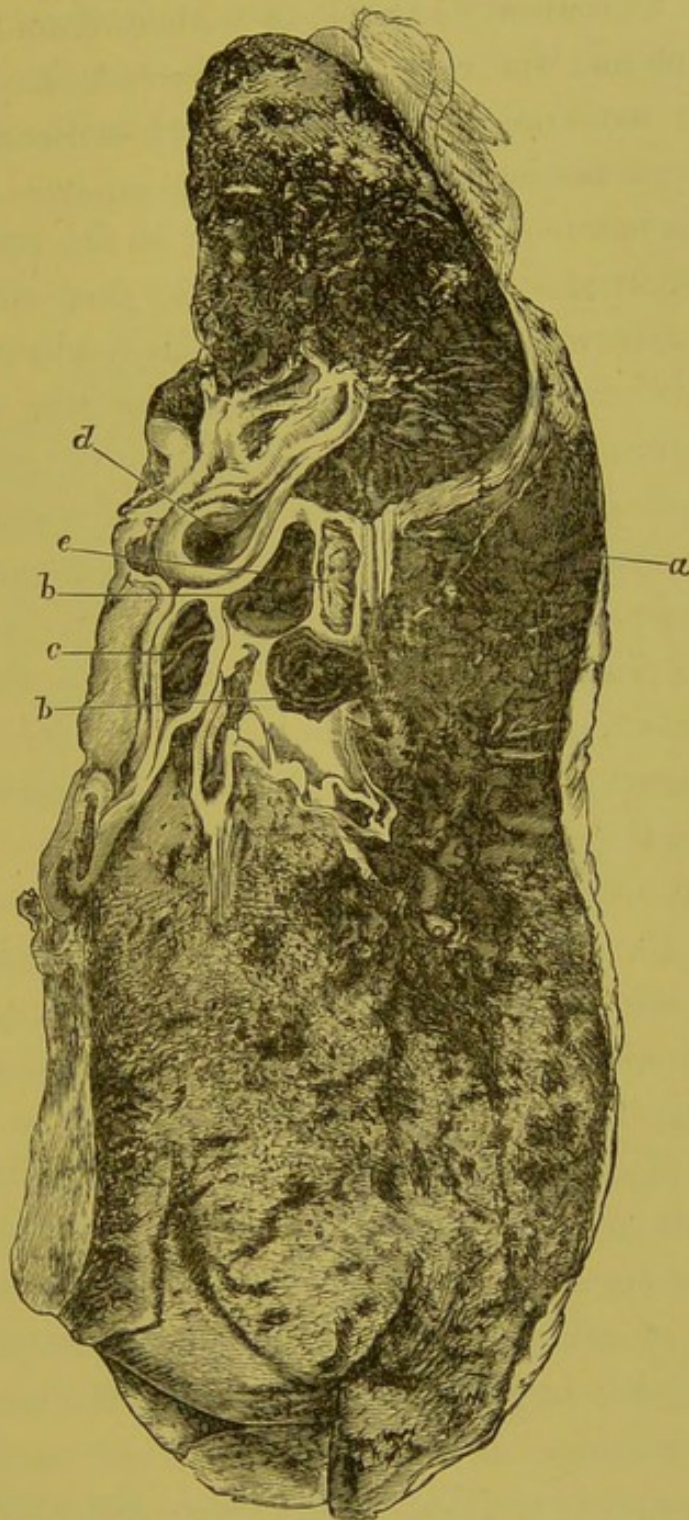
FIG. 13.—Potter's lung—naked eye appearances. *a*. Condensed and pigmented tissue. *bb*.
Enlarged and pigmented lymphatic glands. *c*. Pulmonary artery. *d*. Main bronchus.
e. Pulmonary vein.

identical with those produced by the inhalation of various

kinds of dust. It may be added that Hirt * divides the different forms of disease due to dust into four—namely, those due to coal dust, metal dust, stone dust, and tobacco dust ; to which Merkel adds a fifth †—namely, from cotton fibre. My case is thus described by me in the catalogue of the Western Infirmary museum :—

"The surface of both lungs, as seen in the preparation, is exceedingly irregular, being contorted by deep cicatrices and occasional intervening emphysema. The emphysema is very well marked in the left lung, there being in some places distinct bullæ. In both lungs there is a very marked condensation involving the lower half of upper lobe and the upper fourth of lower lobe. The condensed part is dense and heavy, and of a deep slaty colour. Outside the part which is continuously condensed, and in the midst of the otherwise sound tissue, there are patches of a similar slaty appearance. The bronchial glands also present a deep or almost black colour."

The appearances presented in this lung are exhibited in Figure 13, and the study of microscopic sections shows that here we have to do with a condition very dissimilar to that in either of the two forms of phthisis described in the first lecture. The lesion consists of a great new formation of connective tissue somewhat similar to that which occurs in chronic pneumonia, but much more localised ; indeed, it presents in its localisation evidence that, like the two forms of phthisis just referred to, the irritant reaches the lung by the bronchial tubes, and not by the blood, as in chronic pneumonia.

The annexed figure (Figure 14) shows the conditions presented by one of the more outlying centres beyond the

* Hirt, *Die Staubinhalationskrankheit*, 1871.
† *Ziemssen's Handbuch*, vol. i, p. 500.

continuously affected portion of the lung, and it indicates
the nature of the process, although, even in this com-
paratively recent portion there is evidence that the disease
is a very chronic one.   A bronchus, which has originally
been of considerable size, is the centre of the lesion.
Its calibre, already much contracted, is shown at *a*, while
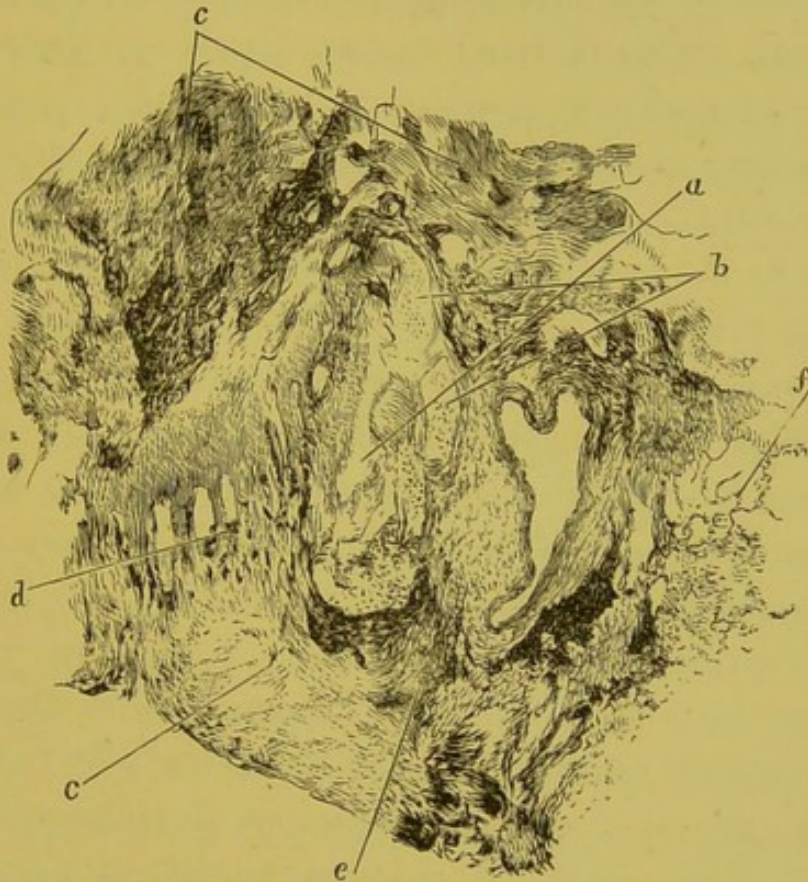
Fig 14.—Potter's lung.   *a*. Bronchus compressed and narrowed.   *b*. Two of its cartilages—
there are other two lower down.   *cc*. Condensed and pigmented tissue (indurated
lung).   *d*. Lung alveoli stretched and enlarged, some with pieces of pigment in them.
e. Some of same, less affected.   *f*. More normal alveoli.   × 8.

the cartilages (*b*) indicate that the tube was a considerable
one.   Around the tube there is a great new formation
of connective tissue (*cc*), which is in many places deeply
pigmented.   This new formed and indurated connective
tissue has involved many lung alveoli, which are obliterated,
but in some places, as at *d* and *e*, there are some
alveoli remaining, but greatly contorted.   Those at *d* are

drawn out into narrow spaces, in which portions of black pigmented material are frequently present. Outside the affected portion we find more ordinary lung alveoli, as at f, but these are dilated and emphysematous.

A very prominent appearance in this case, and apparently in all forms of disease due to dust-inhalation, is the presence of carbonaceous pigment in great abundance in the affected lung tissue. This is seen in the figure to occupy not only the lung alveoli, in which it is sometimes present in such quantity as to give the appearance of coal-black casts of the alveoli (as at d in Figure 14), but also in the connective tissue as at c. It extends to the bronchial glands which, as shown in Figure 13, are as dark in colour as the most deeply affected portion of the lung. It will be remembered that in fibroid phthisis a similar accumulation of black pigment occurs, and it must be inferred that in both cases the affection interferes in some way with the expulsion of the ordinary carbonaceous dust which continually floats in the air. The affection of the bronchial wall, which is perhaps the primary lesion in both cases, probably interferes with the action of the ciliated epithelium lining the tubes, and besides, as shown in Figure 7, this epithelium is soon detached from the wall of the tube, and shed into its calibre, at least in fibroid phthisis.

The presence of the black pigment greatly obscures all other elements as it is extremely opaque, but it is not very difficult by careful examination to convince oneself that in the lung in our case of potter's phthisis there is in addition a very large amount of siliceous material. It is to be found in the form of angular transparent particles in the connective tissue as well as in the walls of the bronchial tubes, and it is sometimes present in considerable aggregations. This is shown in Figure 15, which represents a part where the

siliceous particles were very abundant. It is seen that larger and smaller nodules are grouped irregularly together. Under the microscope these have a characteristic refracting appearance which is very insufficiently rendered in the woodcut. While such a massive accumulation as this is somewhat exceptional, it is possible by careful examination in almost every part of the condensed connective tissue to detect similar particles, frequently in elongated collections. Even in the lung alveoli, where the carbonaceous pigment is contained in large catarrhal cells, careful focussing will
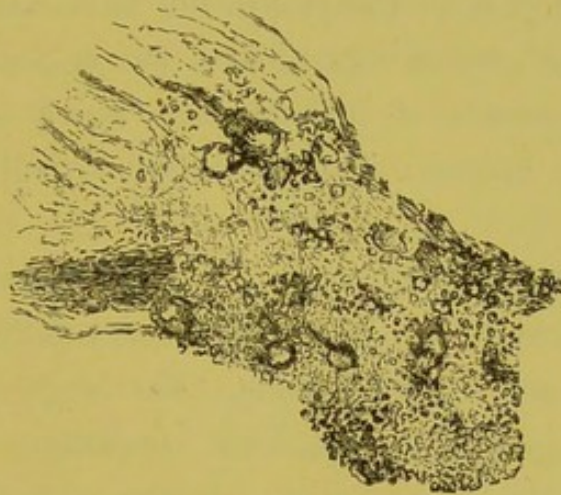
FIG. 15.—Siliceous particles in the same lung. × 340.

often bring out the fact that clear siliceous particles are present as well.

I have taken some pains, by the examination of the dust in a pottery in Glasgow, and by the comparison of it with the various constituents which enter into the paste of which the pottery is made, to determine whether any special constituent predominates in the lung. The following note was made of the microscopic examination of the various substances whose mixture forms the potter's clay :—

Ball clay consists chiefly of a fine amorphous *débris,* the granules being very fine and opaque. Even with very high powers, such as the $\frac{1}{18}$ of an inch, the granules are

still very minute. Sometimes there is a larger piece of the same opaque material, and in addition there are a few fragments having a transparent crystalline aspect, but usually with rounded edges.

China clay consists altogether of much larger pieces than ball clay, and they are nearly all clear or crystalline in aspect. They present very great varieties in size, the larger being scarcely less numerous than the smaller. Some of the larger fragments have a laminated structure, sometimes resembling bundles of bristles, and sometimes having a striated appearance. The fragments have not sharp angles.

Cornish stone, which appears to be a decayed granite, consists of fragments of various sizes, nearly all crystalline, and with sharp angles. The pieces are usually larger than in china clay.

Flint consists of fragments of various sizes, which are mostly opaque, and have little of the crystalline appearance of China clay and Cornish stone. The larger ones show on the surface a spotted or speckled condition.

Dust was collected from the shelves and corners of the pressing room where it lay thickly. Under the microscope it was found to consist of small fragments. The smallest of them were opaque and amorphous, but there were very abundant crystalline fragments, of small size, indeed, but with distinct sharp edges. As the dust was taken from places not lower than the workmen's heads, it afforded a fair sample of what they inhale.

Comparing these appearances with the granules found in the lungs, it can scarcely be said that any one constituent of the clay is specially absorbed into the lung tissue. There may be, and probably is, a great deal of the fine amorphous matter of the ball clay, which cannot be identified in the midst of the tissues; but there is also,

as shown in Figure 15, a considerable aggregation of large masses, which possess a distinctly transparent appearance, suggestive of a crystalline structure.

In regard to the pathology of the entire class of lung diseases due to the inhalation of dust, we may, I think, conclude that the lesions in the lungs are due essentially to the mechanical irritation of the particles of dust. These particles act as foreign bodies, and set up a chronic inflammation, resulting in the new formation of connective tissue. Something will depend on the mechanical characters of the dust inhaled. If the particles be very fine and with rounded edges, then the effects will not be so considerable
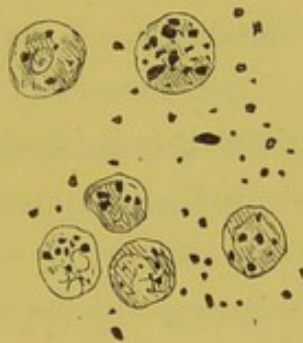
FIG. 16.—From coal miner's lung. Black particles free and in catarrhal cells. ×350.

as when they are larger, heavier, and with sharp edges. In this respect, the dust of coal mines seems to be less irritating than that to which potters are exposed. The carbonaceous particles found so abundantly in the coal-miner's lung, which give such a deep colour to the lung as a whole, are of low specific gravity, and usually possess rounded edges (see Figure 16). It accords with this that the lung may be laden with this black dust, without presenting much induration or condensation. Indeed, Merkel makes the rather remarkable statement, that it is generally acknowledged that phthisis is uncommon in coal miners, and that the coal dust may, perhaps, be

regarded as a kind of preventive of phthisis. Arlidge, on the other hand, notes that bronchitis and phthisis are distinctly more common among potters than in the general community.*

It will be observed that in the potter's lung, which has been more particularly described, there is nothing in the least resembling a tubercle either recent or old, and that there is not a trace of caseous necrosis.† There is, indeed, very little destruction of lung tissue in the sense of necrosis. To the naked eye no cavities are visible, but in the midst of the most affected parts, the microscope reveals some irregular ragged apertures, which may be the beginning of cavities. These remarks apply also to the other forms of disease due to the inhalation of dust.

It is clear that we must draw a strong line of distinction between true phthisis pulmonalis and the conditions due to the inhalation of dust, although we may acknowledge it as possible that the inhalation of certain kinds of dust may, by damaging the lungs, render them more liable to tuberculosis.

The *Clinical features* of this class of diseases differ scarcely less than the anatomical, from those of ordinary phthisis. For example, the following is a *resumé* of the clinical history of our case of potter's phthisis, written by Prof. Gairdner for insertion in the Museum Catalogue.

" Patient was a potter, aged 40, who had suffered for at

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* Arlidge, " On the Diseases Prevalent Among Potters," Social Science Congress, 1871.

† Prof. Hamilton describes in the lungs of stone masons a lesion resembling tuberculosis, which he ascribes to the mechanical irritation of the particles of stone, these forming local centres around which small rounded fibroid new-formations are produced. I have been unable to confirm this observation of Hamilton's, and have not found this form of lesion except in cases of tuberculosis. (See Hamilton, *On the Pathology of Bronchitis*, 1883, p. 202.)

least a year from symptoms attributed in the first instance to exposure while under the influence of drink, but also in all probability to dust inhaled in his occupation. The expectoration was appreciably carbonaceous (though he had never worked underground); there had been no hæmoptysis. The temperatures while under observation were mostly normal or subnormal (once only 101·5°). A notable feature in the case was the extreme feebleness of the radial pulses, and the permanent acceleration of their rate, out of proportion to the other symptoms. The patient was cyanotic, and albuminuria with dropsy was present throughout the period of observation; but though breathlessness was a leading symptom, orthopnœa was by no means constantly present, nor were the physical signs very appreciably different from those of very advanced bronchitis and emphysema, with dilatation of the right side of the heart, but with dull percussion in the inter-scapular regions."

It is a fact particularly emphasised by Greenhow that persons affected with diseases due to the inhalation of dust of various kinds go on working, without apparently much inconvenience, till the lung is very extensively involved. In my visit to the pottery already referred to, I found this amply confirmed. I saw men who had been subject to cough for years and who were very much emaciated, but they were still doing their regular work. I was told that it was usual for them to continue at work till the dyspnœa was very extreme, and that they did not generally live long after they gave up work. Such persons are subject to cough, expectoration, and dyspnœa, but do not regard themselves as ill till a more definite catarrhal attack sets up more acute symptoms. In our own case, for instance, the patient referred his disease to an exposure to cold a

year before death, but he had ceased his occupation as a potter long before death, and we may, I think, infer that by that time most of the fibrous condensation had occurred. In this respect it will be observed that the contrast is very striking between this class of diseases and either of the forms of phthisis proper. In these the constitutional symptoms are such as usually to cause the patient to lay himself up, or at least to count himself seriously ill, at a comparatively early period of the disease.

We have now considered a number of affections, each of which presents a certain resemblance to or analogy with phthisis pulmonalis, but each of which also has a definite and distinct pathology essentially different from that of the two forms of phthisis which we considered at the outset. We may, therefore, conclude that the term phthisis should be reserved for these two forms. This implies, further, that phthisis pulmonalis is essentially a tubercular disease, and that the term is virtually synonymous with pulmonary tuberculosis or local tuberculosis of the lungs. It will be observed that we have not considered in this section acute miliary tuberculosis in its pulmonary manifestations. I shall have something to say regarding it further on, but in the meantime would remark that, while in it the lungs are, as a rule, much involved, yet, as the disease is a general one in which many organs are simultaneously affected, it is in no proper sense a local disease of the lungs, although, as we shall see, it may develop in connection with a true pulmonary phthisis.

THE CAUSATION OF PHTHISIS.

I do not propose in this lecture to go systematically into all the questions related to the etiology of phthisis. I intend rather to deal with the problems which have been presented to my own mind, and to give the answers which have commended themselves in the course of study and observation.   In speaking of the causation of phthisis it is to be understood that we have to do with the initiatory lesions, the real primary conditions, such as we have seen them to be in studying the two forms at their outset.

We have seen that in the great majority of cases the disease begins with an inflammation of the finer bronchial tubes, resulting for the most part in plugging of these and inflammatory thickening of their walls.   From the bronchial tubes the disease extends according to the type followed, either to the alveoli, or to the interstitial tissue. In both cases the lesions are associated with the formation of tubercles which partake of the characters of the other lesions, assuming a caseous or fibroid tendency according to circumstances.   It will be remembered that even in the fibroid form caseous necrosis is probably present in all cases to some extent, and especially in the bronchial tubes, which are the structures primarily affected.

THE SIGNIFICANCE OF CASEOUS NECROSIS.

It will be seen that caseous necrosis is a kind of central fact in the pathology of phthisis, and it will be well at the outset to consider what may be the more direct causation of this condition. With a view to this, we may glance, in the first instance, at the conditions under which

necrosis occurs in the body in general, so as to come to some conclusion as to the cause of the necrosis in phthisis. We may put out of account, to begin with, cases in which the tissues are killed by such obvious and coarse interference with their nutrition as the application of excessive heat or cold, the occlusion of arteries, or the action of violent chemical agents, such as strong acids and alkalies.

MOLECULAR NECROSIS IN GENERAL.—In seeking for an analogy for the necrosis in phthisis, we turn to diseases in which we find a progressive destruction of tissue. This we meet with, for example, in syphilis, in lupus, in hospital gangrene, in typhoid fever, in diphtheria, in leprosy, in glanders. An ulcer of the skin which is progressively enlarging, is doing so by a continuous necrosis in its walls, and such an ulcer may be due to syphilis, to hospital gangrene, to lupus, to leprosy, or even to a continuous local irritation from pressure on the part. In all these cases there is some agent acting continuously on the affected part, although the agent may be aided in its action, or find the occasion for it in some other local condition. The same kind of conditions will be found in progressive necrosis elsewhere. In all syphilitic lesions you are liable to have necrosis, which frequently assumes the caseous form, and which in the case of mucous membranes takes the characters of a progressive ulcer. In typhoid fever there is the necrosis of the inflamed Peyer's patches and solitary follicles, which is to be associated with the action of the special agent concerned in typhoid fever. In lupus the ulceration of the skin has to do with the action of a special irritant, and in glanders the ulceration of the mucous membrane is again related to a specific morbid agent. In pyæmia also, we have, in the lungs especially, numerous localised necroses,

evidently due to the carriage of the products or agents of decomposition to the lungs. In each of these cases it will be observed that the progressive necrosis is associated with inflammatory phenomena, and it need hardly be added that in each of them we have to do with a specific morbid poison which, in many of the cases adduced, has been shown to be related to the growth of micro-organisms.

In considering the cause of necrosis in phthisis I may refer once again to what I have several times seen in cases of gangrene of the lung. In this condition you may have decomposing juices discharged into the bronchi, and these juices may be, as it were, insufflated into the finer bronchial tubes by the processes of respiration. In this way I have seen, in the midst of the otherwise sound lung tissue, small isolated lesions having a marked resemblance in their shape and distribution to those in acute caseous phthisis; so that, just as the condition during life may somewhat resemble that of acute phthisis, the appearances after death may do so also. Each of these little centres generally goes on to the formation of a minute abscess or cavity, there being here in a very acute form necrosis and inflammation such as we have in a more chronic form in phthisis.

In a case which recently occurred, it was interesting to observe that in the centre of each of these little lesions there was a bronchial tube containing a collection of micro-cocci which in some instances also had penetrated into the lung alveoli. In microscopic sections stained with gentian-violet according to Gram's method, the deep blue aggregations of micro-organisms represented the course of small bronchi, sometimes bifurcating. These appearances were very striking, and they indicate that, after being transported to the finer bronchi, the micro-organisms have undergone a rapid multiplication. Without entering more fully

into this condition it may be observed that, although in the putrid fluid there were many forms of micro-organisms, yet in these localised centres only one form was visible, the living tissue apparently having the power of inhibiting the growth of all except this one.   It is one of the forms of the micrococcus, and the effect of its local action is, as I have said, to produce necrosis and inflammation, with the ultimate result of the formation of an abscess, just as the same or a similar micro-organism conveyed to the lung by the blood in pyæmia induces multiple metastatic abscesses, which also imply acute necrosis and inflammation.

Returning to the case of phthisis pulmonalis, I think we must associate the necrosis and inflammation which are so characteristic of the caseous form with similar determining conditions.   There must, as I think, be some agent acting on the tissues of a sufficiently injurious character to induce molecular necrosis and inflammation.   We may infer also that this agent produces the special structures which we designate tubercles.

MOLECULAR NECROSIS IN TUBERCULOSIS.—It may be profitable here, I think, if we look away for a moment from the lungs and consider what are the changes occurring as a result of tuberculosis in other parts.   In doing so we shall find that we have virtually the same conjunction of lesions as we have seen in the case of the lung— namely, inflammation associated with the formation of tubercles and necrosis.

Let us consider first the *tubercular ulcer of the intestine.* The first change here is enlargement of the closed follicles, whether solitary ones or those agminated in the Peyer's patches, this enlargement being apparently inflammatory and due to accumulation of round cells, in the midst of which tubercles may be seen.   The next stage is the

caseous necrosis of the central parts of the enlarged
follicle, which become more strikingly visible to the eye
by their yellow cores. By the softening of the caseous
centre a crater-shaped ulcer forms, the necrosed matter
being carried away. Here, as in the case of the lung,
the necrosis does not confine itself to the tubercles, but
involves also the inflammatory products, and as much of
the normal tissue as may be included in these. When the
ulcer is once formed it enlarges by the advance of the
necrosis, the dead matter being removed from the surface
as soon as it dies; at the same time the inflammatory
infiltration and tubercular formation advance.

As another example take the so-called *solitary tubercle
of the brain.* To the naked eye this presents itself as a
solid yellow mass, occasionally softened in its central parts.
This is nothing but a piece of necrosed tissue which has
accumulated by slow degrees. If we examine the peripheral
parts we shall find that round cells are present in immense
numbers along with frequent giant cells, but it is only
occasionally that distinctly defined tubercles are visible.
What we commonly have is a general infiltration of the
nervous tissue, the cells crowding so as to destroy and take
the place of the proper nervous elements. It is by the
successive necrosis of these new-formed structures and the
simultaneous advance peripherally of the new-formation
that the caseous mass enlarges, until it may acquire the
size of a small apple or larger. As it is buried in the
brain substance and protected from external influences
the dead mass is preserved as a dense yellow tumour,
and it is exceptional to find even central softening.

These are typical examples of local tuberculosis, and
it will be seen that in them, as in phthisis, you have the
inflammatory lesions and the typically tubercular, and these

alike subject to necrosis. It often happens in all situations that what is histologically inflammatory obscures the tubercles, and the only elements distinctive of the latter are the giant cells which are nearly always to be found. But as the necrosis occurs whether there be distinct tubercles or not, we may take the caseous change as in itself a distinct part of the tubercular process.

CAUSATION OF CASEOUS NECROSIS.—Various explanations have been suggested to account for this process of caseous necrosis. One of the most simple and direct is, that as the tubercle is devoid of blood-vessels its elements readily perish. There are, however, serious objections to this view. In the first place it is to be remembered that a tubercle is an exceedingly small body, about the $\frac{1}{50}$ of an inch in diameter or less, and we know that pieces of living matter, although of much larger dimensions than this, may remain alive in the midst of the living tissues when separated from all connection with the blood-vessels. We have, for instance, loose bodies in joints, composed of cartilage and bone, or of adipose or fibrous tissue, attaining to a diameter, it may be of an inch, and yet not suffering necrosis. They may apparently not only survive, but even grow, although separated from all vascular connection with the body, being nourished by the fluid of the joint permeating their substance. It is to be remembered, moreover, that in tuberculosis it is not merely the non-vascular tubercles, but also the inflammatory structures, some of which are vascular, that suffer necrosis. It is a process which does not depend on any indirect action such as the deprivation of blood.

Another view which has been urged is, that in the process of tuberculosis cells are accumulated to such an extent that by their mere pressure they empty the vessels, and so cause

necrosis. This has always seemed to me an exceedingly peculiar explanation. We have to do with cells, the material for whose formation must be obtained from the blood, and yet these cells, according to this view, continue to be formed and to crowd together till the blood is completely expelled, and there is not enough even to keep them alive. One would think that the new formation of cells would stop short at a point where the nutriment had begun to run short, but had not failed so entirely as to cause them to perish. We know of other conditions in which cells accumulate in tissues. In the stage of grey hepatisation in acute pneumonia, for instance, the lung alveoli and the lymphatics of the lung are packed with cells, and the lung tissue is pale from the emptiness of the blood-vessels, but during life sufficient blood gets into the blood-vessels to keep the tissue alive, and it is only exceptionally that necrosis or gangrene occurs. When it does so, we never think of referring it to the accumulation of cells, but rather to some special condition, local or general.

We must, I think, draw closer the analogy between the necrosis of tuberculosis and that met with in other forms of disease. We find that in various examples of necrosis, such as in diphtheria, dysentery, syphilis, glanders, typhoid fever, leprosy, pyæmia, we have special virulent agents applied to the tissues; and the whole local phenomena of these diseases, the inflammation, the specific new-formation, and the necrosis, are all to be related to the action of the virulent agent. Even so, in the case of phthisis, we are to look for the influence of a direct morbid poison which produces all the typical phenomena, although we may expect these phenomena to be to some extent modified by the local and general peculiarities of the tissue and person. It will next be our concern to examine as to the more

direct evidences of the existence of such a morbid poison, especially in the case of phthisis pulmonalis.

PHTHISIS FROM AUTO-INOCULATION OF THE LUNGS.

I do not propose to dwell here upon the evidence obtained by the inoculation of tubercular products in animals, although experiments of this kind have conclusively proved that tuberculosis is in the highest degree inocuable into certain animals. I prefer to take examples from my own experience in the human subject, which seem to me to prove that phthisis pulmonalis is due to the direct application of a morbid poison to the lungs.

I will take in the first place, a case which occurred here on the 19th of June last, and I am able to present to you the preparation of the lung illustrating the points to which I am about to refer. The preparation consists of a child's lung divided so as to show the lung tissue from apex to base. There is here a remarkably localised condensation in the form of a band from an inch to an inch and a half in breadth, occupying the lower part of the upper lobe. This part is almost continuously condensed, and it presented in the fresh state a mottled appearance, due to the occurrence of caseous areas in the midst of a generally grey or red basis. Besides this, there were a few isolated areas near this large one, and there was one patch of a similar character in the other lung. Other parts of the lung were normal, and there was nothing in the history of the case pointing to disease of the lungs. When I made the incision into this lung, and saw what I took to be, and what subsequent microscopic examination proved to be, a recent tuberculosis of a definite piece of lung, it occurred to me that there must have been, by some means or other, a simultaneous

infection of the whole of that piece. The fact that in this broad piece of tissue there was abundant caseous necrosis, yet that there was no softening, served to indicate that the infection must have been recent, and as the lesion was virtually in the same stage of advancement in every part, it was natural to look for a cause which would produce a sudden invasion.

The preparation before you gives a sufficient explanation of these facts. On opening up the main bronchus of this lung I found a small aperture in its wall, communicating directly with a large caseous gland which was firmly adherent to the bronchus. In the preparation this aperture is indicated by a piece of whalebone introduced. We have here, in fact, an example of the somewhat rare accident of a caseating gland perforating the wall of a bronchus, and discharging its contents into the latter. The caseous matter thus discharged has been insufflated, and you will notice that the perforation is just opposite the bronchial branch leading to the condensed piece of lung, and the matter must have been discharged almost directly into this bronchus, although some of it has got into other bronchi, and even into those of the other lung, but apparently in much smaller quantity. It is obvious that this matter must have been exceedingly virulent in its character, as when insufflated even in comparatively small quantities it has produced very intense results.

If the question be raised whether this enlarged caseous gland may not have been affected secondarily to the lung, the answer is not difficult. In the first place the disease in the lung is very recent, whereas that in the gland is obviously of some duration, as evidenced in the case of the gland by its size, by the complete caseation which it has undergone, and by the softening in its central parts. In

the second place, the aperture in the bronchus has all the appearance of a perforation from without. The mucous membrane is entire up to the edges of the aperture, but it is undermined, and a bristle can be passed beneath the edge, and for about a quarter of an inch into the softened gland substance.

In view of what is to follow, it may be interesting to consider for a moment the probable manner in which the bronchial gland became itself infected. The case was in its general aspects one of tubercular peritonitis, and the patient died apparently in consequence of an acute peritonitis, perhaps due to perforation of the intestine in connection with a tubercular ulcer, there being many of these present. The primary tuberculosis being in the peritoneum or in the intestine, we have to consider what path it may have taken to reach the bronchial gland. There was no tuber-culosis of the abdominal lymphatic glands, and the disease has not in this case travelled from the abdominal lymphatics up into those of the chest, a course which, however, is not infrequently followed, at least in the reverse direction. The path of infection was a more direct one—namely, through the diaphragm to the pleura and thence to the gland at the root of the lung. It is noted in the report that "the liver is firmly adherent to the diaphragm, and there is a layer of tubercles contained in the adhesions. The tuberculosis involves the substance of the diaphragm and extends through it to the pleura, so that on the pleural surface of the diaphragm several white nodules are visible."

I presume that the sub-pleural lymphatic vessels pass to the glands at the root of the lung, and we may thus account for the occurrence of a single large tubercular gland at the root of the right lung, there being no enlarge-ment of any other bronchial glands.

This case, then, illustrating the sudden invasion of a definite piece of lung by caseous matter, with the effect of producing a distinct local tuberculosis of that piece, seems to me to be an interesting experiment performed for us by a pathological process.

Another case which also occurred here this summer, is of a somewhat different kind, but illustrates the same points, as well as some others.

It was, to appearance, a case of laryngeal phthisis. The patient, a man aged 31 years, had been attacked with a throat affection four years before death and seems never to have recovered completely. Seven months before death he is reported to have had "congestion of the lungs" lasting 8 weeks, and since then the throat had been worse. During his residence in the Infirmary the symptoms referrible to the larynx were predominant.

The lungs after death presented appearances which to me were very striking and interesting. There were evidences of phthisis in two such widely separated stages, that the case might be regarded as presenting virtually two different pathological entities. At both apices there were conditions which would be correctly designated as those presented by a healed phthisis, conditions which I have not infrequently the opportunity of observing. At the apex of the left lung there were old pigmented cicatrices, in the midst of which were some firmly encapsuled chalky masses. At the apex of the right there was also pigmented cicatricial tissue, and in the midst of it, two small clean cavities with pigmented walls and no trace of caseous matter. While this was the condition of the apex, the middle and lower lobes of this right lung presented a very extensive recent tuberculosis. The greater part of these lobes was in a state of almost homogeneous caseous condensation, only the extreme base

being free, and there was none of this recent affection in the upper lobe. In the left lung there was, beneath the level of the old pigmented cicatrices, a homogeneous recent caseous condensation, occupying a considerable portion of the upper lobe.

Looking to these extensive condensations, obviously of recent date, we are, as in the case already described, induced to seek for a recent source of infection, for a somewhat sudden insufflation of tubercular matter. As both lungs were involved, we had to look for the source higher up than in the previous case. The source, I believe, was found in the larynx. There was in the larynx a very marked ulceration, producing partial detachment of portions of both cords, so as to give rise to prominent warty-looking projections. There was also somewhat deep ulceration, especially at the ventricles, and the mucous membrane of the ary-epiglottic folds was considerably swollen and œdematous.

From what I have seen clinically I have been led to infer that the insufflation of the discharges in cases of tuberculosis of the larynx, perhaps of accidental occurrence, may lead to a rapid tubercular infiltration of the lungs. I know from observation that the expectoration in laryngeal phthisis often teems with tubercular bacilli to an extraordinary extent. In one case which I observed clinically for many months, I was greatly struck by the fact that, while the lungs remained apparently free from evidences of disease for a considerable period, there was at a particular time a sudden invasion of them, and an exceedingly rapid extension of the disease. This observation has been confirmed by others made subsequently.

It is undoubtedly true that we find the larynx affected secondarily to the lungs much more frequently than the

converse, but even when that is so, we may believe that
the insufflation from the affected larynx may aggravate the
existing lung affection. It is not improbable that in the
case which has suggested these remarks, the old healed
phthisis may have been the starting point of the laryngeal
affection. Circumstances may have led to the healing of
the lung while the laryngeal condition continued. If that
be so, then the tuberculosis had lingered in the larynx
for years, only attaining to an aggravated ulcerative con-
dition lately.

LECTURE III.

THE TUBERCULAR BACILLUS. EXTENSION OF THE TUBERCULOSIS FROM ITS ORIGINAL SEAT.

The tubercular bacillus; *mode of detection and appearance; artificial culture; its presence in the lungs, uncertainty in distribution; the lungs a favourable seat, case in illustration; mode of invasion of the bacilli; their action on the tissues, not due to the bacilli but to their products, concentrated or dilute; the results specific or simply inflammatory.* Contagiousness *of phthisis; only in exceptional circumstances.*

Extension of the tuberculosis; *tuberculosis a superficial process; illustrated in the urino-genital tract, in the intestine, the peritoneum, the lymphatic glands; contrast with other infective processes.* In phthisis pulmonalis; *begins in finer bronchi, and extends by air passages, to further parts of lung, to bronchi, trachea, larynx, alimentary canal; and by lymphatics to general connective tissue of lung and to bronchial glands.*

FROM the considerations already put forward we may now I think, with some confidence speak of tubercular infection, and may proceed to enquire as to the nature of the infective matter, the mode in which it reaches the lung, and its course of procedure there.

This brings us at once face to face with the consideration of the tubercular bacillus. Whatever we may think of the actual part taken by this micro-organism in the tubercular process, there can be no doubt of its presence and no difficulty in identifying it. It has a quite definite appearance and mode of growth. You will find under the

microscopes many examples of it, in the lung tissue and in the sputum from cases of phthisis.

The method by which these preparations have been made is that which has been perfected by Ehrlich and is generally known by his name. It is not necessary to give more than an outline of this method, the details of which are now accessible in many books devoted to Bacteriology as well as in those on Practical Pathology. We prepare a solution of fuchsine. This is done by first shaking together 4 parts of aniline in 100 parts of water for about fifteen minutes. This solution of aniline in water is filtered so as to remove excess of aniline oil (the filter should first be moistened). To the 100 parts of aniline water we now add 5 parts of a strong alcoholic solution of fuchsine and our staining fluid is completed. In proceeding to use it we filter as much as we need into a watch glass. The sputum prepared on a cover glass, or a section of the tissue to be examined, is now placed in the solution. The sputum should be left for about half an hour, and the sections for several hours, but the process may be much accelerated by heating the fluid. This modification we generally adopt, holding the watch glass over the flame of a spirit lamp for a few seconds till the solution is slightly warm, and repeating the warming at intervals. The material thus treated assumes a deep red colour, and the next process is to destroy the colour in everything except the bacilli. This is done by placing the material in nitric acid. In the case of sputum we use a strong solution of 1 part of strong nitric acid to 2 of water, which decolorises in a few seconds. In the case of sections of lung tissue, the solution of nitric acid is weaker, and instead of a watery solution we may use one in which 3 parts of nitric acid are added to 100 parts of alcohol. In any case we allow the

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nitric acid to act till the structure loses its red colour and presents merely a slight pinkish tint. While undergoing treatment with nitric acid the specimen is occasionally removed and washed in water, which stops the action of the nitric acid. If the colour is not sufficiently removed it is returned to the nitric acid, and the process repeated till a sufficient degree of decolorisation is attained.* The preparation may now without further treatment be mounted in the usual way in Canada balsam, and examined under the microscope with the aid of Abbé's illuminating apparatus, when the bacilli will stand out as brilliantly coloured threads. It is generally desirable, how-
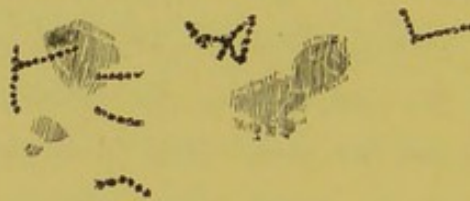
Fig. 17.—Tubercular bacilli in sputum.   ×1,200.

ever, to use a contrast stain, and the preparations which you will find under the microscopes to-day have been treated with a watery solution of methyl-blue before being mounted in Canada balsam. When thus prepared the brilliant red colour of the bacilli contrasts with the blue of the surrounding structures.

The individual bacillus is a fine rod or thread beaded by reason of the presence of what appear to be spores (see

* I have lately adopted Ziehl's method of staining, which presents the advantage that the fuchsine solution is much more permanent, and does not require to be prepared every time it is needed. Ziehl's solution consists of water 100, carbolic acid 5, fuchsine 5. The preparation is first stained with this in the same manner as with Ehrlich's solution. It is afterwards transferred to dilute sulphuric acid (20 per cent), instead of nitric acid, and then it is further treated as described above.

Figure 17). When present in large numbers in the lung, they form a fine matted net work.

It is well known that Koch has taught us *to cultivate the bacilli* outside the body, and as his researches are now open to all, by the publication of a translation of his paper on the Etiology of Tuberculosis in the volume of *Essays on Bacteria in Relation to Disease*, recently issued by the New Sydenham Society, it will not be necessary for me to go into this part of the subject with any degree of fulness. Pure cultivations are to be obtained on solidified blood-serum kept at a temperature between 98° and 99° F. Let me here give in his own words Koch's description of the appearances presented:—

" For the first few days no alteration is to be observed in the cultures in the incubator. If, however, there is a change, and drops or spots of white or other colour form on the surface of the serum, increase more or less rapidly in size, render the fluid at the bottom of the glass turbid or cause the serum to liquefy, it is a sign that the culture is not pure, and that foreign bacteria have entered, and choked the growth of the tubercle bacilli. If these drops or spots are examined, they are found to consist of bacilli, or micrococci, which, by Ehrlich's method of staining, assume a different colour from the tubercle bacilli, and are distinct from them also in size and shape.

"In the glasses free from these impurities, the first signs of the growing colonies of tubercle bacilli are not visible to the naked eye for ten to fifteen days. They then appear as whitish points and small spots lying on the surface of the serum ; they have no lustre, and consequently stand out clearly from their moist surroundings. They are best compared to dry scales adhering loosely to the surface of the serum. The number of the scales, and the extent of surface covered by them, vary with the richness of the

infecting material in bacilli, and with the extent of the surface over which it was rubbed or spread out."

Further on (pages 144 and 145 of the translation), Koch in his more minute description of the mode of growth of the bacilli emphasises the fact that they increase not in thickness but in superficial extent. They do not penetrate into the serum, but always remain on the surface, loosely attached to it.* When examined by a comparatively low magnifying power, the colonies of bacilli present a very peculiar form, which in itself is distinctive. On the surface
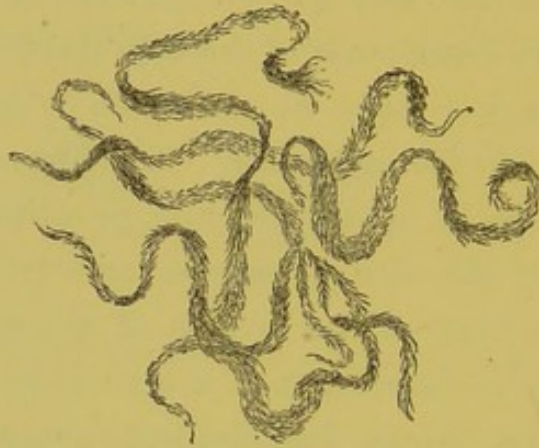
FIG. 18.—Colonies of tubercular bacilli (after Koch). × 700.

of the serum they assume the form of fine strongly curved and crooked lines like the letter S or like serpentine figures (see Figure 18). By pressing a cover glass down on the growing colonies, and then lifting it, it is possible by staining in the usual way to get the figured appearance shown in our illustration.

* I observe from the *British Medical Journal*, 12th February, 1887, that M. Rocard has succeeded in simplifying considerably the method of cultivating the tubercle bacillus. He found that ordinary broth gelatinised with gelatine or with gelose was one of the best media for the cultivation of the bacillus, if from 6 to 10 per cent of glycerine were added to the broth. The bacillus was found to grow more quickly and freely in this than in gelatinised blood or serum, eight days being sufficient to produce a thick layer.

Having said so much regarding the tubercular bacillus as the agent of infection, we have now to consider its position in relation to phthisis pulmonalis. In order to avoid mistake, I may here state that it is intended to speak later on of other influences concerned in the etiology of phthisis, and we shall see that these influences form a very important element in the causation. In the meantime we have to concern ourselves with the bacillus, and to endeavour to indicate how it comports itself in the living body. This organism is present in the lung tissue in enormous numbers in many cases of phthisis. If you take for instance some curdy matter from the inside of a cavity in a case of caseous phthisis, and, after spreading it on a cover glass, treat it in the way I have described, you will always find immense numbers of bacilli. You will also as a rule find them abundantly if you take a small piece of caseous matter from the cut surface of one of the condensations. It must be said however, that the search for tubercular bacilli in the lung tissue, after hardening in alcohol, is often very laborious, and sometimes rather disappointing. If you examine in the fresh state in the way just mentioned, you will be nearly always successful, but if in the same case you take a piece of the lung, harden it in alcohol, and then make sections and prepare them in the usual way you will often be disappointed. The bacilli have somehow undergone alterations which interfere with efficient staining. In a considerable proportion of cases, however, by patience in making sections you will succeed in finding them, although they are present in very different proportions even in the same case. A very interesting and elaborate investigation has been made by Dr. Percy Kidd into the

distribution of the bacilli in the lesions of phthisis.* In
the paper describing this research, frequent reference is
made to the apparent uncertainty in the distribution of
the bacilli. I am induced to believe that this uncertainty
is largely to be referred to imperfections in our methods
of detection, an inference which is confirmed not only by
what has just been said in regard to the difference in the
result when preparations are made in the fresh state as
compared with those after hardening, but also by the
observation of Koch that caseous matter in which no
bacilli can be detected is often highly infective when used
for inoculation in animals. This may be because only
spores are present, but spores if in considerable numbers
ought to be visible.

In seeking for the tubercular bacillus in phthisis pul-
monalis, we shall find it most abundantly and most readily
in those cases in which the disease is advancing rapidly.
Its detection is easiest in structures which have already
become caseous, or in the walls of cavities. In caseous
structures the bacilli stand out more distinctly than in
those which have not undergone this change. The caseous
change renders everything homogeneous, and the altered
structures do not show any differentiation of structure, so
that the bacilli are plainly seen on a homogeneous back-
ground. In other parts where there are abundant cells,
which attract the eye, the bacilli are less plainly seen, but
their presence in much more recent lesions can often be
determined on careful examination. They are to be found
in the bronchial tubes obstructed by inflammatory products,
being present inside of and between the cells which fill the
bronchus. They are often visible in the alveoli in caseous

* *Medico-Chirurgical Transactions,* vol. lxviii, 1884-85.

phthisis, sometimes enclosed in the large catarrhal cells which plug the alveoli. In the very chronic fibroid form the bacilli are scarcely to be detected in the lung tissue, although usually present in the sputum and in the cavities.

It is evident that the bacilli if once they obtain a lodgment, find the lung a very favourable place for their growth. In this connection I would refer here to a case which I have already published, and which, in other aspects, I shall have occasion to bring up again. I have placed under the microscopes sections illustrative of most that I have to say here. The case was one of acute general tuberculosis, and there was evidence not only as to the method by which the bacilli reached the blood—namely, by tuberculosis of the wall of a pulmonary vein, but also of the presence in the blood of large numbers of bacilli. They were found in the blood clot present in the heart after death, and they were seen in the pulmonary vessels. From these vessels they had passed into the alveoli, and their abundance in the alveoli is very extraordinary; evidently they had multiplied there with great energy. The sections under the microscopes have been stained with fuchsine, according to Ehrlich's method, and some estimate may be formed of the abundance of the bacilli by using a low power. In some places it looks as if the alveoli were injected with a red material, so numerous are the bacilli. It is interesting, also, to find that in some places there is little beyond bacilli in the lung alveoli, as if their growth had taken place before there was time for any considerable inflammation. In most instances, however, there are the usual cells in the alveoli, the results of catarrhal inflammation. In this case it may be inferred that the bacilli had been carried into the alveoli, probably by the regular currents, which pass from the capillary blood-vessels into

the alveoli, and back into the lymphatics or veins, and that, finding themselves in a favourable position, they have multiplied greatly. It was found that they were present in every part of the lungs in almost equal abundance, not presenting that localisation which is so characteristic in phthisis, and which is related, as we have seen, to the extension of the disease by the air passages. It may be added that Koch [*] has described an escape of bacilli from the vessels of the Malpighian tufts of the kidney into the uriniferous tubules; and that, in acute general tuberculosis the local manifestations are presumably brought about by bacilli, which have passed from the blood-vessels into the lymphatics or serous spaces. This is not to be regarded as a passage through membrane, as it is known that in the walls of the blood-vessels there are fine apertures (stigmata) by means of which they form communications with the serous spaces and lymphatic vessels. It has been demonstrated that finely divided granular matter, such as vermilion, may pass through the walls of the blood-vessels by these apertures.

In what has gone before we have considered, for the most part, cases which present lesions of advanced phthisis, and in which the more recent manifestations are directly referable to the older ones. When we consider the first onset of the disease, the conclusions we come to must be matters of inference; but we may with considerable probability suppose that these lesions are not very different from those seen in the recent parts of an advanced case. We may presume with Koch that, except under very unusual circumstances, a few bacilli first find access to the lung, and that in certain cases they multiply and produce their

* Koch, *Mittheil. aus dem K. Gesundheitsamte.* 1884.

effects. Our view of the case, however, cannot be regarded as by any means complete without the consideration of a considerable number of further questions. We have, in fact, to deal with such questions as these:—What are the exact relations of the bacilli to the actual lesions in the lung? and, Are there any conditions of the body whose prior existence is necessary to the growth and multiplication of the bacilli?

In regard to the first of these questions, it cannot be inferred that the bacilli, in themselves, are in any serious degree harmful to the tissues. They are minute vegetable bodies, which, as we shall afterwards see, multiply along surfaces or channels, and do not usually penetrate into the living tissues. Such minute bodies cannot, by their merely physical properties, produce any serious damage. It must be rather that, in their growth, they evolve certain chemical principles which are injurious to the tissues. We may infer, also, that although the bacilli themselves are usually confined to surfaces and canals, yet their products need not be. These are presumably soluble, and being dissolved, they may extend beyond their seat of origin, being, however, weakened by dilution the further they are removed from their source. These statements are hardly to be taken as mere speculations when we consider certain facts of every-day observation in tubercular processes in general. I have already pointed out that in all forms of tuberculosis you have, for the most part, besides the specific tubercular lesions, the more ordinary phenomena of inflammation. In tuberculosis of the peritoneum, for instance, you have, as the more specific effects of the tubercular infection, the flat yellow tubercles in the highest degree caseous; but these are buried and covered in by new-formed connective tissue, which is fully organised

and contains blood-vessels, resembling in every respect that which is produced in any ordinary chronic inflammation of the peritoneum. It cannot be believed that the bacilli are present in that new-formed connective tissue; they are confined to the caseous masses, and it is the diluted products which induce the more simple inflammatory lesions.

In the case of the lungs, we have in the condition of the pleura a very striking example of an ordinary chronic inflammation produced no doubt by the products of the tuberculosis, but without any of the specific characters which imply the actual presence of the bacilli. In phthisis pulmonalis chronic inflammation of the pleura is a constant concomitant. Leaving out of account in the meantime acute pleurisy, which has a special mode of origin to be considered afterwards, we have here a simple chronic inflammation characterised by the new formation of vascular connective tissue resulting usually in adhesion by coalescence of the opposed pleural surfaces. This is often described as a conservative process opposing itself to the destructive lesion in the lung, and as a matter of fact it often prevents serious mischief which might otherwise ensue from perforation of the pleura, as we shall see farther on. In the meantime I would emphasise the fact, that while in the lung we have a progressive infection due to the multiplication of the bacilli, in the pleura we have merely an inflammatory process due to the more or less diluted products, and possessing no infective characters; the inflammation indeed remains confined to the neighbourhood of the portions of lung affected, and there is no independent affection of the pleura. In order to produce the inflammation in the pleura the irritating products must pass beyond the lymphatics, for as we have seen, the lymphatics of the lung do not communicate

with those of the pleura. They must in fact soak through the pleura. We may infer indeed that a good deal of the inflammatory lesion in the connective tissue of the lung itself is of a similar nature, and that it is only where we have tubercles or caseation that we have bacilli actually present, these conditions only occurring where the products act in a concentrated form. Whenever the bacilli appear they soon induce the new formation of cells which group themselves around them, and partly take them into their substance.

CONTAGIOUSNESS. — In connection with the tubercular bacillus the question of contagion naturally suggests itself, and there are some who find in this a ready explanation of many cases in which several members of the same family are attacked. The question, however, is not so easily decided. It is quite certain that many persons exposed to the tubercular bacillus remain free from phthisis, and this applies even to persons who may be presumed to be specially exposed.

A very important paper bearing on this subject was published by Dr. C. Theodore Williams in the *British Medical Journal*, September, 1882. His paper is based on data furnished by the hospital for consumptives at Brompton, since its opening in 1846. It was opened with 90 beds, which were increased in 1856 to 200, and in 1873 to 240. A new building was opened in 1882, but the statistics apply to the old one in which the ventilation was much less perfect. "The deficiency in the ventilation must have led to a large accumulation in the wards of the products of respiration, and also of our friends the bacilli." The out-patient department was even worse. "The attendance of two hundred to three hundred out-patients daily, of whom a large proportion was consump-

tive, must, on the theory of infection, have proved a considerable source of danger to the assistant-physicians, to the clerk who enters the names, and to the porters who marshal them and keep order."

An analysis embracing the whole attendants, including medical officers, clinical assistants, matrons, nurses and servants, porters, dispensers, &c., shows that although some died of consumption, yet an equal amount of phthisis "might be found in any large institution not specially devoted to consumption, nay, more, in any mass of town population."

While these statistics prove that phthisis is not contagious like typhus fever, measles, or small-pox, there are still a good many facts which indicate that in cases where persons are in very close relation with consumptive patients, they are liable to contract the disease. The results of the enquiry instituted by the Collective Investigation Committee showed that cases of this kind do occur. The conclusion come to by Prof. Humphrey and Dr. Mahomed, from an examination of these results, was that "if phthisis is a communicable disease, it is only under circumstances and conditions of extremely close personal intimacy, such as persons sharing the same bed or the same room, or shut up together in numbers in close, ill-ventilated apartments." Dr. Reginald Thomson found among 15,000 consumptives, 15 cases in which he was of opinion that the wives were infected from the husbands.

We may, I think, fairly conclude that phthisis is rarely contagious, and that when it is so the circumstances are such as to make us infer that the person has been exposed to the contagion in an exceptionally concentrated form. This may be by his living in very close contact with the patient, or else from the disease being in such a form and

stage as that an unusually large amount of infective matter is expectorated by the patient.

MODE OF EXTENSION OF TUBERCULOSIS.

TUBERCULOSIS A SUPERFICIAL PROCESS :—In considering the mode of extension of the tubercular process in the lungs we shall, I think, be considerably assisted by taking into account its habits in other situations. Studying tuberculosis in its various localities, we shall find, I think, that it is not usually a penetrating lesion, but that it rather extends on the surface of membranes, and along canals or other channels. It is nearly always stopped and confined by a membrane, if there are no channels through it, and if it does penetrate, it is after it has formed by necrosis a passage through the membrane. Let me illustrate this by one or two examples :—

In *tuberculosis of the urinary tract* the disease often begins in one kidney, and for the most part its primary seat seems to be in the pelvis, where it forms in the way I have already indicated a tubercular ulcer. From the pelvis the ulceration and the tubercular process as a whole, gradually extend into the kidney tissue. There are two channels by which it may travel from the pelvis to the kidney tissue—namely, the uriniferous tubules and the lymphatics. The process, extending slowly, penetrates deeply into the kidney, forming irregular cavities, which may, by slow degrees, extend even to the periphery of the organ. In its whole course the process in the kidney may be said to be a superficial one, the lesion is always in the form of an ulcer, and the edge of the advancing tuberculosis is never far removed from the surface of the ulcer. Although, after the lapse of a considerable time,

it may extend to the external surface, it does not penetrate the capsule. The capsule, in fact, seems to oppose a definite limit to its extension. Besides this extension into the kidney tissue the tuberculosis commonly travels along the urino-genital tract to considerable distances. The ureter is nearly always affected, and it often happens that throughout its extent from the pelvis to the bladder it is continuously involved in the tuberculosis. I place before you, for instance, a specimen from the museum, in which the ureter is greatly thickened and lined continuously with a consistent yellow opaque layer of caseous matter, and yet the outside is smooth, and there is no appearance of tubercles on the surface. The tubercular process does not extend through the wall of the ureter to the tissue around. From the ureter it extends readily to the bladder, which nearly always presents numerous tubercular ulcers of a superficial character. From the bladder it may extend to the vas deferens and to the vesiculæ seminales, and even in some cases to the testicles; at least it is common to find the vas deferens and testicle affected continuously with the bladder, ureter, and kidney. According to Weigert,* the invasion of the kidney occurs the reverse way—namely, from the testicle along the vas deferens to the vesiculæ seminales and prostate, then the bladder, ureter, and kidney. In support of this view he cites the fact that in the great majority of cases the affection occurs in males. Whether this be correct or not, the affection extends as I have said, along surfaces, and there is no penetration deeply except by a process of ulceration.

Take, again, the very interesting cases of *tuberculosis of the intestine* on the one hand, and *of the peritoneum* on the other. Tubercular ulcers are very common in the

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* *Virchow's Archiv*, vol. ciii, p. 539.

intestine, and while the tuberculosis advances locally as in
the kidney, by extension of the ulceration, there is nearly
always evidence also of extension by the lymphatics. On
examining the peritoneal surface of the intestine opposite
an ulcer, little white tubercles are almost always to be seen.
It is usually possible before opening the intestine to deter-
mine the existence of ulcers by the presence of groups of
tubercles at intervals beneath the peritoneum. These
tubercles are, however, under the peritoneum, and not in
it. There is scarcely any tendency to extension of the
tuberculosis to the peritoneum generally. It is surely a
very striking fact that, while cases of tubercular ulceration
of the intestine are of every-day occurrence, yet it is very
unusual to find the peritoneum itself engaged, although
each ulcer has a group of tubercles visibly present imme-
diately beneath the peritoneum. An interesting corollary
to this is the converse condition in which the peritoneum
is the seat of tuberculosis, while the intestine is not affected.
Tubercular peritonitis is a superficial tuberculosis of the
peritoneum as a whole, but the tubercles apparently con-
fine themselves to that membrane, and do not readily
penetrate into the wall of the intestine. You may have
tubercles thickly sown over the entire surface of the
peritoneum, but none in the intestinal wall beneath. This
is the more interesting when we notice the directions in
which the tuberculosis may travel from the peritoneum,
following as it does the open paths from that cavity.
In the female, for instance, tubercular peritonitis is very
frequently associated with tuberculosis of the Fallopian
tubes, extending sometimes to the cavity of the uterus.
Then, again, it often extends through the diaphragm to
the pleura, as in a case to which I referred in the last
lecture. The tubercular infection, it will be seen, extends

along the lymphatic channels, or by any open pathway, but usually respects the barrier of a membrane. The extension by the lymphatics is everywhere one of the commonest modes, and hence in connection with most tubercular processes the lymphatic glands are involved.

Tuberculosis of the lymphatic glands themselves affords another example of the limitation of the tubercular process. The familiar scrofulous enlargement of the glands is a tuberculosis. This is proved by inoculation experiments in animals, and also by the presence of the tubercular bacillus. We have seen also, as confirmatory of this view, that in the rare case of the bursting of a scrofulous gland into a bronchus, the result is a tuberculosis of the lungs of the usual caseating form. Now, it is a familiar fact that scrofulous glands do not readily infect the neighbouring structures, unless, as a result of softening and suppuration, the capsule of the gland has been destroyed by inflammation. The glands enlarge and become caseous; they may even become completely converted into obsolete masses of caseous matter without the disease extending beyond the capsule, so that the mass may be shelled out as from a sac. The therapeutic device of cleaning out the gland and scraping the inside of the capsule with Volkmann's spoons is a practical application of the fact that the lesion does not penetrate into the capsule.

From these considerations it would appear that tuberculosis is a process in which surfaces and canals are concerned, and scarcely at all the substance of organs, except in so far as these are penetrated by channels. It may perhaps be said that in all kinds of infection, extension more readily takes place along existing channels, but it will be found that this is true to a much smaller extent in regard to other processes than in regard to tuberculosis. Cancer undoubtedly

has special affinities with the lymphatic system, and has a peculiar proclivity to extension by the lymphatic channels, but it by no means respects such limits. We often find cancer of the stomach, for instance, extending through the wall of this organ, and involving the peritoneum. This is the regular mode of extension in colloid cancer, and it sometimes occurs in other forms. Sarcomas again, in their growth, show in most cases little respect for bounding membranes. These are, of course, cases which are scarcely comparable with that of the extension by means of a micro-organism, but even in this class of cases I know of none which has the same superficial habit. We have many diseases presumably dependent on the action of micro-organisms, which primarily attack the surfaces of membranes or canals, out in nearly all of them the penetration of the infection is soon evidenced by manifest changes due to its presence in the blood. Take for instance the case of diphtheria. Whether we believe that the specific micro-organism in this case has been discovered or not, we may assume that the disease is due to a morbid poison which has its local seat in the affected mucous membranes. It has its local point of attack, but rapidly extends to the blood. The same applies to typhoid fever, syphilis, hydrophobia, &c. It is unnecessary to illustrate this with any detail, but these considerations seem to me to emphasize the fact that tuberculosis is a disease of surfaces, and that when it extends to the blood it is for the most part by causing necrosis of the walls of blood-vessels, and not by a penetration of the living wall. It is a point of very great moment from many points of view, that tuberculosis is in this sense a superficial disease. I would call to mind, for instance, the usually satisfactory results which surgeons obtain in cases of excision of scrofulous joints, as contrasted

with the removal of cancerous tumours or of syphilitic ulcers.

IN PHTHISIS, EXTENSION BY AIR PASSAGES AND LYMPHATICS. —Turning now to the case of phthisis pulmonalis, we shall find that here also the same principles hold; it is here as elsewhere a disease of surfaces, of channels, of tubes. If we take the primary lesions, such as have been described in considering the two forms of phthisis, it is apparent that in both forms the disease begins in the finer bronchial tubes. The focus of the disease in each little centre is an inflamed and tubercular bronchiole. If we believe that these lesions which, in a case of phthisis, represent the advanced guard of the disease, are the same as those at the proper origin, then the disease itself begins in an invasion of the finer bronchi, the infective matter coming from without. The bronchioles being the primary seat, we shall find that there are two open channels by which the infection may extend—namely, the air-passages, and the lymphatics. By these channels the disease may extend in the lung tissue itself, infecting neighbouring parts. It may also extend by them beyond the lung, to the lymphatic glands on the one hand, and to the larger air-passages and so onwards on the other. It will be necessary to look at each of these somewhat closely, and also to consider the more unusual extension into the blood-vessels, in consequence of which the infection may be carried to distant parts of the body.

In all cases of phthisis it is probable that extension occurs both *by the air passages* and *by the lymphatics ;* but it may be said that in the caseous form it occurs principally by the air passages, while in the fibroid form the lymphatic extension is very important, and has largely to do with

the special character of that form. The fact that in both
forms the early lesions of the extending disease centre in
the finer bronchial tubes, indicates that it is mainly by this
path that the disease advances in the lung as a whole.
We may infer that matter from older lesions is carried
by the bronchial tubes, and drawn into further parts of the
lungs by the processes of respiration. This will especially
be the case when caseating cavities are present.

In many cases a more obvious extension by the air-
passages is visible. It often happens, for instance, that the
mucous membrane of larger bronchial tubes, more particularly .
in the caseous form, becomes the seat of tubercular ulcers.
This is more especially seen in bronchi connected with
cavities; it is always well to open up the bronchi leading to
cavities, so as to discover if such ulcers be present. They
will be found in a large proportion of cases in the form of
more or less rounded superficial erosions of the mucous
membrane, sometimes with distinct white tubercles at their
edges. In like manner there may be extension to the main
bronchi, to the trachea, and to the larynx. In these two
latter situations, there is often considerable destruction of
the mucous membrane, not infrequently leading to exposure
and necrosis of the cartilages.

The extension to the alimentary canal may be regarded
simply as a prolongation of that to the air-passages. All
observers seem agreed that, in at least half of the cases
which die of phthisis pulmonalis, tubercular ulceration of
the intestine is present. The lesion is due to tubercular
material being carried into the stomach and intestine instead
of being discharged by the mouth. These grosser extensions
seem to imply a greater abundance of bacilli than the more
ordinary extension to the finer bronchial tubes. It may be
presumed that the bacilli have less opportunity of lodging

in the larger bronchi, where the currents of air are voluminous and the cilia active, than in the finer ones, where a trivial inflammation is liable to lead to obstruction, so that any material present will be longer detained.

In considering the *extension by the lymphatics* we have to bear in mind the general facts concerning the distribution of the lymphatics in the lung, and more especially their relation to the bronchial tubes. As the lesion begins in the finer bronchi we may expect that the extension to the lymphatics will be, first, to those immediately connected with the affected bronchi, and then to those farther out. The lymphatic vessels in the lungs, as elsewhere, run in the connective tissue, and are present wherever that tissue exists. They are found in the walls of the bronchi, in the tissue around them and the larger vessels, in the interlobular and in the sub-pleural connective tissue.

The lymphatic system of the lungs is usually described as consisting of three sets of vessels. One set has its seat in the walls of the bronchi and in the connective tissue around, forming the peri-bronchial lymphatics; a second set forms a plexus beneath the pleura, the sub-pleural lymphatics; and a third occupies the interlobular connective tissue. These three sets of vessels form one system, however, and evidently anastomose freely, so that the whole system may be regarded as one. The circulation in these vessels is carried on chiefly by means of the respiratory movements, and can scarcely be regarded as occurring regularly in one direction. It has often been pointed out that the carbonaceous pigment of the lungs, to which we have already referred in speaking of the diseases due to the inhalation of dust, is deposited in the lymphatic vessels, and is carried into all parts of that system, being found wherever lymphatics exist. It is found in the three situations just

mentioned; but it is a fact worthy of notice that it does not extend to the pleura proper, and is not to be found in its sac, a sufficient indication that the pulmonary lymphatics form a separate system which does not directly communicate with that of the pleura. The lymphatics of the lung pass to the lymphatic glands at the root of the lungs.
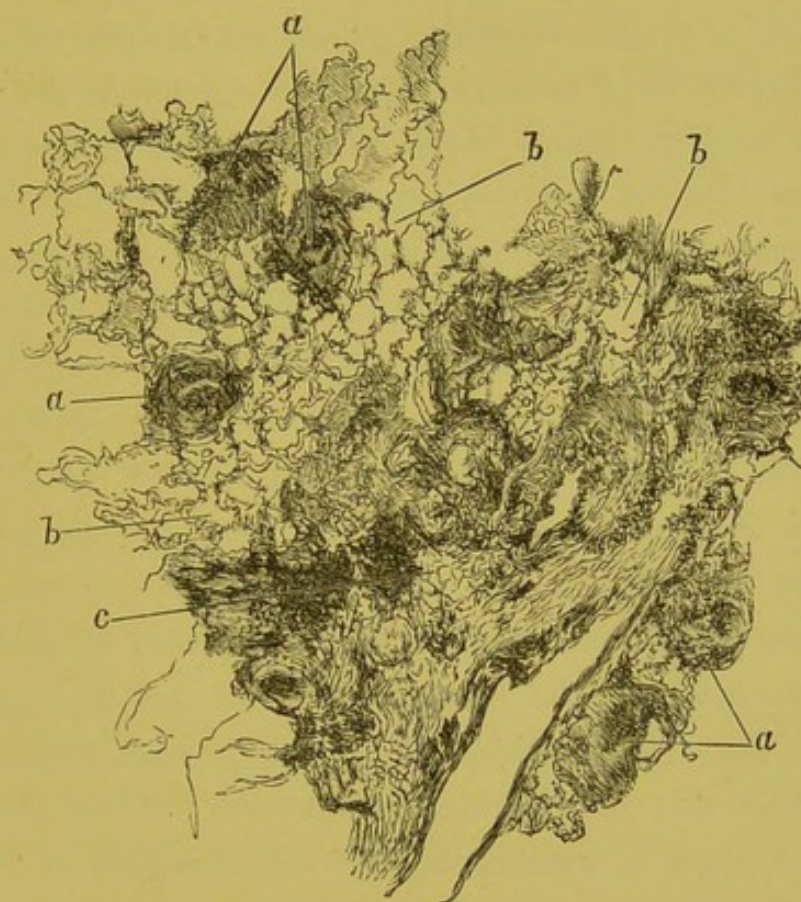
FIG. 19.—Fibroid phthisis. *a a.* Tubercles in the connective tissue around a bronchus; the darker centres indicate caseation. *b b.* Emphysematous lung alveoli. ×20.

In phthisis pulmonalis, particularly in the fibroid form, we frequently find evidences of the extension along the lymphatic system. This can often be inferred from the manner in which the tubercles are grouped in relation to the bronchi. It is shown in Figure 19, in which tubercles are represented as in the wall of the bronchus and at various distances outside it, but so arranged around the

bronchus as to indicate that it has formed the centre or focus. In the more fully developed cases of fibroid phthisis the lesion consists essentially of a tuberculosis and thickening of the connective tissue, chiefly the interlobular and sub-pleural portions of it. There can be no reasonable doubt that the infection is carried to these situations by the lymphatics, just as the carbonaceous particles are so conveyed.

In both forms the bronchial glands are always tubercular, and it is worthy of note that here, as elsewhere, the disease does not readily extend beyond the capsules of the glands.

The relation of the process to the pleura will be more fully considered farther on; but it may be here remarked that although pleurisy, both acute and chronic, is probably the commonest complication of phthisis, yet, a true tuberculosis of the pleura is rare, unless there be actual perforation. You may have a tubercular pleurisy by extension from the peritoneum, or from the pericardium, or by the penetration of a tubercular abscess in connection with bone, but a true tuberculosis is not usual in phthisis.

The extension to the blood-vessels will not be considered here, as the whole subject of acute general tuberculosis, with which this is intimately connected, will be taken up in another place in considering the complications of phthisis pulmonalis.

LECTURE IV.

PREDISPOSING CAUSES OF PHTHISIS. THE PROCESS OF HEALING. SECONDARY PHENOMENA IN THE LUNG.

Predisposing Causes. *Inheritance, evidences of its influence in phthisis: general principles of inheritance, illustrated by inheritance of structural peculiarities, &c.; inheritance in infectious and infective diseases, influence of race and family in yellow fever, small-pox, &c.; application to phthisis. Other predisposing causes.*

Possibility of dealing with bacillus in treatment. *Antiseptics; isolation.*

Healing of Phthisis. *No recovery of necrosed caseous tissue. Two methods of disposal illustrated by case of lymphatic glands; discharge and cicatrisation, or obsolescence and calcification. Compensatory hypertrophy as a result of healing.*

Secondary Phenomena in the Lung. *Acute pleurisy and pneumo-thorax, both imply necrosis of pleura without adhesion. Pulmonary hæmorrhage; early hæmoptysis associated with the initial lesions; late hæmorrhage, mostly from aneurism in cavities.*

WE have seen reason to infer that phthisis pulmonalis begins with the inhalation of a few bacilli which lodge in the finer bronchi or in the lung alveoli, but in saying this we do not by any means explain the whole pathology of this disease. It may even be said that in the meantime at least, so far as practical purposes are concerned, we explain the lesser half of it. There is no doubt that in certain cases, such as in two which I related in a former lecture, the tubercular infection is so strong, the virus is admitted in such quantity that the development and further

extension of the disease are matters of certainty. But when it is the case of a few bacilli straying into the lungs with the inspired air, it is quite another matter; every one is liable to such accidents, and no doubt all dwellers in towns at least, frequently inhale bacilli. While all are exposed to the infection in such minute doses, it is only a certain small percentage in whom the disease develops. There must, therefore, be something besides the mere entrance of a few bacilli to account for the facts. This leads us to further enquiry as to the causation, and more particularly as to the predisposing conditions.

INHERITANCE.

Almost the first matter that occurs to us in this connection is the question of the inheritance of phthisis. We have to bring the undoubted facts of heredity into relation with the equally undoubted facts of infection. It is necessary at the outset to distinguish carefully between direct inheritance of a disease, and the inheritance of a proclivity to it. There are several infectious diseases which are capable of direct transmission from parent to offspring. The occurrence of hereditary syphilis, for instance, in this sense is by no means uncommon. Small-pox, intermittent fever, measles, and relapsing fever may be communicated to the child in utero. Children have been born with small-pox or the cicatrices of it, and there are also cases in which at birth the large spleen and cachectic appearance of ague were present.

In regard to tuberculosis there is no undoubted evidence, so far as I can learn, of its direct transmission in any form from parent to offspring. Even if the possibility of such a transmission be not denied, we can still say that the direct

inheritance of all tubercular processes, and of phthisis pulmonalis among them, may for practical purposes be put out of consideration.

Opinions undoubtedly differ as to the exact extent to which inheritance plays a part in the causation of phthisis, but it seems generally agreed that to some extent it has a distinct influence in predisposing to the disease. One of the most convincing evidences on this subject is furnished by a very interesting investigation made by Dr. Reginald E. Thomson, and published by him in his work on *Family Phthisis* (1884). Using the material afforded by the Hospital for Consumption at Brompton, he endeavoured to obtain a full record of the family histories of cases of consumption, and he succeeded in obtaining 80 family histories which were apparently complete. The total number of children for these 80 families was 385, giving an average of nearly 5 for each family. Of these 203 were males, and 182 females. Subtracting those who died in childhood, numbering 21 males and 16 females, who may or may not have been affected with tubercular disease, we have 348 remaining, of whom 194 became phthisical—namely, 98 males and 96 females, leaving only 154 who were exempt, of whom 84 were males and 70 females. The details given by Dr. Thomson show that inheritance was from the father in 24 families, from the mother in 30, from both in 14, and from grand-parents (atavism) in 12. While these results are of great importance, they are not to be regarded as establishing any fixed proportions, although undoubtedly proving the influence of inheritance in determining the disease.

A somewhat similar line of enquiry was prosecuted by Dr. Theodore Williams, and the results recorded in the *Medico - Chirurgical Transactions* for 1871. His patients were seen in private practice, and the results may therefore

be regarded as perhaps as little exposed to fallacy as possible. He found that on enquiry amongst persons affected with phthisis, the disease could be traced in relatives in 48·4 per cent.

Again, Hérard and Cornil in their work, *De la Phthisie Pulmonaire* (1867), give statistics in regard to 100 patients suffering from phthisis whom they interrogated with great care as to the occurrence of this disease in their parents. Of these patients some were in the city, and some in the hospital. They found that of these 100 cases no less than 38 had sprung from parents who had been tubercular, and if we add those whose grand-parents had been so, we should probably have about half of all cases showing hereditary predisposition.

Lastly, I may quote the opinion of Laennec as expressing the general conclusions of a man who looked broadly at the subject. In his treatise on *Diseases of the Chest* (English translation, 1834) he says—" If the question of contagion (in phthisis), is very doubtful, the case is very different with the hereditary predisposition. The universal and habitual experience of practitioners proves that the children of phthisical parents are more subject to this disease than others are." Then he goes on to comment on the frequency of exceptions, sometimes only one or two members of a family are consumptive in each generation, or we may find large families destroyed whose parents are healthy. He instances a case of his own in which the father and mother died upwards of 80 years of age of acute diseases, after seeing 14 children die of consumption between the ages of 15 and 35.

In order to appreciate the proper influence of hereditary predisposition I may be allowed here to refer at some length to the question of *inheritance in general*, and more particu-

larly to inheritance as bearing on susceptibility to particular diseases. We have to ask first, What it is in our structure and vital conditions that is determined by inheritance.

If we take racial and family peculiarities as examples of inheritance, we shall see that these consist in variations of structure and function, sometimes descending to very minute details. In the case of family peculiarities the transmitted detail may be a particular shape of the nose, a special colour of the iris, even a wrinkle on the brow which is a reproduction from the parent. These facts are the more extraordinary when we consider that all these special characters are inherent in the impregnated ovum when it begins its career of development, and that the potentiality of some of them may be retained during many years of life, only to be developed at a period far removed from birth.

When we turn to the inheritance of diseased conditions, we find that in nearly all cases of undoubted transmission it is minute peculiarities of structure which are so transmitted. Supernumerary fingers are in a remarkable degree hereditary, as are also webbed fingers and toes. Ichthyosis, which is a faulty condition of the epidermis, hæmophilia, which seems to be due to some abnormality of the structure of the blood-vessels, colour blindness, which is probably referable to some variation in the structure of the retina, are hereditary in a very striking fashion. All of these are conditions in which peculiarities in the structure or function of particular organs or tissues have been transmitted from parents to offspring.

Turning to the class of diseases to which tuberculosis belongs — namely, those depending on parasitic micro-organisms, we find that inheritance plays a very striking and remarkable part. Many facts indicate, for instance,

that race has a great influence on the susceptibility to
this class of diseases. Take first the very remarkable
observations recorded by Koch in his work on *Traumatic
Infective Diseases* (New Sydenham Society's Translation,
1880). In this book he relates that, by the injection of
putrid blood into the subcutaneous tissue in mice, he
succeeded in producing a disease which he calls septicæmia
of mice. The ordinary mouse is highly susceptible to this
disease, so that inoculation of the blood of a mouse affected
with it into another mouse inevitably produces the disease.
The influence of race is seen in the fact that field mice,
although so closely resembling ordinary mice that at a first
glance they may be mistaken for one another, are apparently
quite insusceptible. Again, Chauveau,* while investigating
the subject of splenic fever, found that although he could
produce this disease in ordinary sheep by simple inoculation
with the lancet, he could not do so in the Algerine race of
sheep. In order to produce it in them, he had to use
considerably larger quantities of the virus. Again, Pasteur,
in his experiments on chicken cholera, another disease due
to a micro-organism, found that while ordinary fowls were
highly susceptible, those of the cochin-china breed were
hardly at all so.

A similar fact comes out in regard to the specific fevers
in man. The most remarkable instance is that furnished
by the immunity possessed by certain of the dark races from
yellow fever. It is a fact frequently attested that negroes
in the West Indies are nearly exempt from that disease. I
learn also from a resident in the West Indies, who has
seen several epidemics of yellow fever, that the Hindoos
imported as coolies are also almost exempt. A similar
immunity is possessed by the Chinese.

* *Comptes Rendues*, 1880, vol. i, pp. 15-25.

A certain immunity is also possessed by the negroes in relation to malarial fevers, and the other dark races also possess it to a less degree. Here is a table, quoted by Hirsch, * in which the number of persons who died of malarial fevers in Ceylon, per 1,000 of population, is given :—

Negroes, ...	1·1
Natives of India, ...	4·5
Malays, ...	6·7
Natives of Ceylon, ...	7·
Europeans (English), ...	24·6

It is clear from these facts that the negroes enjoy a certain immunity from such fevers, even greater than that of natives of malarial districts.

As a contrast we find that small-pox is much more fatal among negroes than among whites. My friend in the West Indies tells me that during an epidemic of yellow fever when the negroes exult over their immunity, they are told, "Wait till small-pox comes, and then it will be your turn." This difference is apart from vaccination, as we find according to Hirsch (vol. i, p. 15,) that "In Boston there died in the epidemics of 1749 to 1792, or at a time when there was no question of protection by vaccination, 10·8 per cent of white patients and 23·7 per cent of black." As further examples of the influence of race may be mentioned the almost absolute immunity of the negro race from syphilis, and the great susceptibility of the same race to cholera and leprosy. (See Hirsch's Handbook.)

I may add to this the interesting fact pointed out by Major Tulloch in his papers before the Statistical Society, that negroes are remarkably susceptible to phthisis when removed from warm climates. He states that while the

* *Handbook of Geographical and Historical Pathology*, vol. i, p. 245. New Sydenham Society's Translation, 1883.

mortality among negro troops in the west coast of Africa from phthisis was 6·3 per 1,000, it rose to 33·5 in Gibraltar.

In regard to family peculiarities, which are also matters of inheritance, I suppose no one has been a few years in practice without becoming aware of facts which indicate that infectious diseases have particular affinities for certain families. There is the fact that diphtheria again and again visits with terrible effect the same family. Dr. H. C. Cameron of this city has called attention to this fact as a result of his experience in performing tracheotomy.* A similar statement may be made in regard to scarlatina, which may recur with fatal frequency in the same family. We must suppose that in all these cases there is a structural or physiological difference which renders persons more or less susceptible to the inroads of specific pathogenic micro-organisms, and that these differences are parts of the matter of inheritance.

Applying these principles to *phthisis pulmonalis,* we must believe that there are certain peculiarities of structure and function which render certain persons more susceptible to the tubercular bacillus, so that when it finds a lodgment in the lungs of such persons it is liable to multiply and increase, whereas in others its growth is inhibited by the living tissue of the lungs and it dies. The precise nature of this peculiarity cannot be stated, but there seems a general concensus of opinion that it is not confined to the lungs. As there are racial and family peculiarities, affecting almost all the structures of the body, which are correlated to certain conditions rendering the persons more or less susceptible to the inroads of the specific poisons of the various fevers, so there are conditions of the body as a whole

* *British Medical Journal,* 22nd February, 1879.

correlated to the liability to phthisis pulmonalis. It may be difficult to agree with Jenner in his descriptions of the tubercular and scrofulous diatheses, or with any other general picture of the hereditary phthisical constitution, but we must believe in the existence of such peculiarities.

OTHER PREDISPOSING CAUSES.

In regard to the further predisposing causes of phthisis, it may be perhaps possible to divide them into first, those which, by directly affecting the lungs, render them more susceptible; and secondly, those in which the susceptibility is part of a general condition of the body. It will not always be possible, however, strictly to differentiate between these two, although in some instances it will not be difficult.

The pre-existence of certain affections of the lung apparently produces a distinct predisposition. Observation seems to show, for instance, that the pneumonia which sometimes accompanies whooping-cough and measles is not infrequently followed by a true tubercular phthisis. It is not improbable that in these cases the products of inflammation contained in the air-passages may form a peculiarly fitting nidus for the propagation of the bacillus. It is believed also by some that catarrh of the respiratory mucous membrane may induce the settlement of the bacillus, and it may do so, according to Dr. Weber,* by producing abrasions of the mucous membrane, by weakening the ciliary action, by rendering the respirations more shallow, and by weakening the nutrition and energy of the whole system. Considering, however, in what an enormous number of cases catarrhs produce no such effect, it seems possible to ascribe only a

* *Croonian Lectures*, 1885.

subordinate influence to such "neglected colds." There must indeed be other predisposing factors present, and if there be, then it is not unlikely that the existence of catarrhal products in the air-passages, and the injury done by the inflammation to the bronchial mucous membrane may render it easier for the bacilli to obtain a lodgment.

Probably of similar import is the asserted influence of the inhalation of dust in its various forms in producing phthisis. From what was said in a previous lecture, it will be seen that the inhalation of dust leads to conditions which, although resembling phthisis, are quite distinct from the true tubercular forms. I am not convinced that in the asserted prevalence of phthisis among workmen specially exposed to the inhalation of dust, this fact is sufficiently taken into account, and I do not know of any collection of facts which bear on the proportion of tubercular cases among such occupations as compared with the general community.

There can be no doubt, on the other hand, that in-door occupations, especially where ventilation is deficient, strongly predispose to phthisis. There is the universally recognised fact, that phthisis is vastly more common in urban than in agricultural districts. This is very strongly brought out in Greenhow's contributions to the Reports of the Medical Officer of the Privy Council for 1860-61. In these he says that "the high death-rate from lung disease belonged, according to occupation, to men or to women of the district; that it sometimes was nearly twice as high for the employed sex as for the unemployed sex, and that it only extended to both sexes when both were engaged in the occupation."

The extraordinary frequency of phthisis in prisons is a fact probably of similar import.* Its prevalence, also, in

* See Hirsch's Work, vol. iii, page 222.

the army has been ascribed especially to the residence of soldiers in badly ventilated barracks. Welch,* for instance, in reference to the British army, says that "nearly half of army consumption is connected with vitiated barrack atmosphere."

In regard to all these cases, it is difficult to say whether the predisposition is due mainly to direct injury to the lungs from the vitiated or dust laden atmosphere, or to the interference with the general health which such insanitary conditions are liable to produce.

Climate is usually regarded as another element in the causation of phthisis. There seems no doubt that phthisis is more prevalent in low-lying and damp localities. This result has been worked out with considerable detail by Bowditch in the case of certain of the New England States, by Milroy in the case of eight of the chief towns of Scotland, and by Buchanan in a number of English towns. It is certainly an interesting fact that while in Alexandria, Damietta, and Port Said, with a moist climate and great range of temperature, the inhabitants are subject to the disease in a high degree, it is very uncommon in the inland districts of Egypt, which are characterised by uniformity of temperature, and comparative dryness of the air. †

The relative immunity afforded by residence at high altitudes is a fact which is now-a-days taken advantage of for therapeutic purposes, and admits of no doubt. The climate of such elevated regions may be regarded as virtually the converse of that in low-lying and damp localities. In the meantime it can only be matter of speculation how far these climatic influences affect the lungs directly or the body in general.

* Quoted by Hirsch, vol. iii, page 221.
† See Hirsch, vol. iii, page 199.

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Among the influences which predispose to phthisis, by interfering with the general health, may be mentioned diabetes. In this disease the general condition of the body induced by the disease renders the persons affected peculiarly liable to a form of phthisis pulmonalis, which is probably nothing more than an ordinary acute tubercular phthisis. The frequency of phthisis, after recovery from typhoid fever, may also be mentioned.

In concluding this part of the subject I may be allowed to add that, in regard to *Treatment*, we are as yet without sufficient evidence as to the existence of any means by which the tubercular bacillus may be directly attacked within the body. The method recently devised by Dr. Bergeon of introducing sulphuretted hydrogen into the large intestine, with a view to its absorption and subsequent exhalation by the lungs,* does not seem to have stood the test of experience. Another attempt in the same direction has still to be put to the test. In the recently issued *Bulletin du laboratoire de recherches expérimentales et cliniques, sur la traitement aseptique de la phthisie pulmonaire*, we have an elaborate exposition of a method of treatment by the subcutaneous injection of carbolic acid in such quantity as to saturate the tissues of the body. In the absence of sufficient indications in this direction our endeavours, as practical physicians, must be directed towards those conditions of the body which render it susceptible to the inroads of the bacillus. In the case of family proclivity the person should be placed in such surroundings as will foster his resisting power. Where the occupation and ways of life are such as to predispose, then we must endeavour to alter these circumstances for the better. It may be

* See Dr. Bennett's paper in *British Medical Journal*, December, 1886.

said, indeed, that even after the actual occurrence of phthisis much may be done with a view to cure. No one who has seen many *post-mortem* examinations can doubt that such treatment must often be successful; the appearances indicating the healing of phthisis are very common.

It may possibly in the future come to be a practical sanitary question whether consumptive patients should not in some way be isolated from the general community. There can be no doubt that their presence in the general community, by continually breeding and giving out large quantities of tubercular bacilli, supplies one of the necessary elements in the propagation of all tubercular disease. It may indeed be a question whether, in the interests of the patients themselves as well as of the community at large, it will not be advisable to undertake their treatment in isolated special hospitals. If consumptives were treated in sanatoriums placed in dry, airy situations, and with efficient ventilation indoors, as well as the opportunity of sufficient out-door exercise, then surely there would be a much larger proportion of cures than can be looked for in the general community under present circumstances.

Modern pathology indicates that leprosy depends upon a micro-organism very similar in appearance to the tubercular bacillus, and although there is not much evidence that this disease is contagious,* yet our ancestors had probably good reason for the isolation of persons subject to it. We may,

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* A very important case in which leprosy was communicated by vaccination has been recorded by Prof. Gairdner in the *British Medical Journal*, June, 1887. A medical man, residing in an island in the South Seas, vaccinated his own son from a child who afterwards showed evidences of leprosy. From his son he vaccinated the son of a captain. This boy was seen years afterwards by Professors Gairdner and M'Call Anderson, who regarded it as an undoubted case of leprosy. He died in Helensburgh with the most typical symptoms of that disease.

I think, with considerable probability refer the disappearance of the disease from most European countries to this isolation of the persons affected, which would undoubtedly render the bacillus of leprosy much less prevalent among the general community.

THE HEALING OF PHTHISIS PULMONALIS.

It is quite common in the *post-mortem* room to meet with cases in which the apices of the lungs are the seat of old pigmented cicatrices, sometimes with cavities in their midst, sometimes with particles of chalky matter. These conditions indicate the former existence of a tubercular phthisis, and it now concerns us to consider how active infective processes such as those we have been considering may take pause, and pass into the state of healing.

We can hardly speak of the healing of phthisis in the sense of a recovery of the portion of lung involved. We have seen that in most cases there is an actual necrosis of the tissue involved, and that the caseous matter which results contains the active bacilli by means of whose growth the infective process extends. We have to consider how this dead matter is disposed of, and how the lung as a whole comports itself.

It may assist us in the understanding of this subject if we glance first at the *process of healing* in tubercular lesions *elsewhere than in the lungs*. Take as an example, the familiar case of a tubercular or scrofulous gland in the neck. A slowly advancing tuberculosis takes place in the lymphatic gland, caseous necrosis accompanying the process in regular course, till it may be the whole gland is converted into little more than a caseous mass.

The gland is greatly enlarged, but, as a general rule, the caseous mass is enclosed by the capsule through which the tubercular process does not extend to the parts around, although it may proceed by the lymphatics to neighbouring glands. When the gland concerned is completely transformed and necrosed, or even before that, one of two things may happen. If it be a superficial gland the tissues over it may inflame, and by and bye give way, and the caseous matter will then be discharged. By this process the infective matter is let out, and it is liable to produce a tuberculosis of the structures with which it comes in contact. Thus there may be produced a tubercular sinus in the skin and subcutaneous tissue which may continue for years, constituting the common scrofulous sores of the neck. On the other hand, the discharge of the caseous matter may be complete, or the tissues may resist the infection, and in that case healing occurs by the formation of healthy granulation tissue and proper cicatrisation, just as in an ordinary simple wound. It is well known that healing is usually much accelerated when, instead of allowing the caseous matter to come in contact with the healthy tissue, the gland is laid open by the surgeon, and all the contents of the capsule thoroughly scraped out with Volkmann's spoons. This then is one mode of healing in tuberculosis—the caseous matter is completely evacuated either by a natural process or with the aid of the surgeon, and healing is brought about by the ordinary processes of chronic inflammation.

Another course is followed when the glands are not in a position to evacuate their contents. This frequently happens in the case of the mesenteric and abdominal glands. When tuberculosis attacks them it proceeds just as in the other case till, it may be, the whole enlarged

gland has become caseous. As the glands here are in a protected situation, it is seldom that inflammation around them leads to penetration of the capsule, and the tuberculosis, therefore, remains limited to the glands. Having exhausted the whole material of the gland the process very often pauses, and each affected gland is left in the form of a caseous mass inside its capsule. This dead material, in which I presume tubercular bacilli are at first still present, although inactive, may be long retained in the body, just as dead matter may lie in the tissues anywhere. It is a well known fact in pathology that dead animal matter lying in the living tissues is usually by degrees impregnated with lime-salts, and this process occurs in the case we are considering. The caseous gland may thus be converted into a putty-like, or finally into a stony mass, definitely confined by the capsule. It is by no means uncommon to find in the mesentery of adults hard stony masses of considerable size, which on being cut into look like pieces of chalk. These represent old tubercular glands which date back probably to the days of childhood, and have been lying in an obsolete condition, it may be for many years. I presume that in the course of these processes, the bacilli gradually perish.

In the case of the lung we may have healing brought about by processes somewhat analogous to those two. Of course it cannot be said that in the lung the disease ever comes to a natural pause by the exhaustion of its material, although there are cases in which, the whole of one lung having become involved, it has, by the discharge of the necrosed products, been converted into a single large cavity in which no active tuberculosis is visible. This is the case in a preparation which I show you, and which, during life, presented characters resembling those of pneumo-

thorax. (See *Museum Catalogue*, series iii, No. 28). In most instances, however, the patient will die before the whole lung has been thus used up, and even if he lives, the process will almost certainly go on in the other lung. While this method in its details may be left out of account, we often find that the process becomes in some way checked, probably as a result of a change in the hygienic conditions of the person. As there are persons whose tissues resist the inroads of the bacilli, so, one who is already infected may acquire sufficient vigour to check further progress. In that case we have, in the lung, portions of tissue in a state of caseous necrosis, along with inflammatory lesions. The dead caseous matter may be dealt with by methods somewhat similar to those already specified. The caseous matter may be expelled, and if, in passing, it does not in the usual way produce an extension of the disease, then this clearing out of the caseous matter is really, as in the case of the scrofulous gland, a step towards healing. By it we get rid of a quantity of infective material, and a process of healing may now set in. We have thus a cavity which becomes lined with granulation tissue. The granulation tissue, here as elsewhere, develops into the connective tissue of the cicatrix, and, as usual, this tissue is of a peculiarly dense and contracting character.

The ultimate result will depend somewhat on the local conditions. If the lung be sufficiently free from adhesion, or if the cavity be so situated that the lung can shift to some extent, then the cavity may contract and actually close, the cicatricial tissue coalescing till nothing of a cavity is left, but only a cicatrix, around which the lung tissue may become emphysematous. On the other hand, the circumstances may allow of only partial contraction; the pleura may be so firmly adherent to the chest wall as to prevent

the dragging of the lung towards the cicatrix, and in that
case the cavity will not close. It becomes, however, perfectly
quiescent, being lined with dense connective tissue, and
it may even be shut off from all connection with the air
passages. In both of these processes there will be considerable
shrinking of the contents of the thorax, and this will imply,
on the one hand, a displacement of the thoracic contents
towards the seat of the process, usually the apex of the
lung; and on the other hand, the dragging in of the chest
wall, with a resulting flattening or contraction, mostly seen
beneath the clavicle.

It is a fact of some interest that the connective tissue
formed in these processes is always pigmented, so that
the cicatrix or the walls of the cavity present a deep
slaty colour. The coloration here is due to the inhaled
carbonaceous dust, and it will be remembered that in
fibroid phthisis and in the conditions due to the inhalation
of dust, a similar dark slaty colour is produced. The
process of healing may, in this way, possibly be related to
that which occurs in the fibroid form of phthisis, and it
may perhaps be said that in that form there is a persistent
effort towards healing alongside of the advancing process.

It sometimes happens that the method of healing differs
from that we have been considering, and rather resembles
that which I have described in the case of the tubercular
mesenteric glands. The caseous matter in the lung may
fail to undergo the process of softening, and in that case
no cavity is formed. The dead mass remains embedded in
the living tissue, but as its infective characters have been
checked, it is now treated as a piece of ordinary dead
animal matter. A layer of granulation tissue forms around
it, and if the mass be small enough, the granulations may
actually eat into it and cause its absorption. For the most

part, however, the dead matter becomes, at least in part, surrounded by a capsule of connective tissue, which is formed by the development of the granulation tissue, and the dead mass, like the scrofulous gland, becomes by degrees infiltrated with lime salts, and converted into putty-like or chalky matter. It is not uncommon to meet with pieces of chalk in the midst of cicatrices at the apices of the lungs. It is well known also that such chalky particles are sometimes expectorated. This latter occurrence would imply a renewal of the disease in an active form, the destruction of the lung setting free the encapsuled masses.

COMPENSATORY HYPERTROPHY AS A RESULT OF HEALING.

An important question in relation to the healing of phthisis is, whether hypertrophy of the remaining lung occurs to compensate for the loss of tissue which has been sustained. We have seen that phthisis almost always implies necrosis of tissue, and when healing occurs there is a certain amount of permanent loss of lung tissue.

In a paper which I read before the Medical Society of London in the year 1884, entitled "On Compensatory Hypertrophy of various organs," I entered somewhat fully on this subject amongst others, and I shall here depend chiefly upon that paper for what I have now to say.

We have first to deal with the question of hypertrophy of the lung in general. Does such a condition exist? The answer to that question is given in the affirmative, by the mere relation of a case which occurred to me and which I have recorded in the paper referred to, as well as briefly in my *Manual of Pathology*. It was a case in which one lung had apparently failed to expand, except very imperfectly, at birth. There was a complete absence of the

ordinary carbonaceous pigment from the upper lobe, and
pigment was present only in a small portion of the lower
lobe. The unpigmented and unexpanded lung was replaced
by a congeries of sacs, which were dilated bronchial tubes.
The lung of course occupied much less than the normal
space. The other lung had undergone a very striking
enlargement. Its anterior part projected beyond the medias-
tinum so as to a great extent to fill up, along with the
hypertrophied heart, the space left by the atrophied lung.
At the time of the *post-mortem* it was noted that the anterior
margin of this (which was the right) lung reached two
inches to the left of the left nipple. The tissue of this
lung was of normal consistence and deeply pigmented, and
there were none of the ordinary appearances of emphysema,
although the air vesicles throughout the lung were larger
than normal. A point of some interest was, that the principal
bronchi of the atrophied lung were of equal size with those
of the hypertrophied one, indicating that in their development
the bronchi are independent of the pulmonary parenchyma.
The blood-vessels, on the other hand, were much smaller,
this being consistent with the general fact that the blood-
vessels follow the tissues in their growth and are dominated
by them. In the case of the enlarged lung we had a true
hypertrophy, but without an actual increase in the number
of the air vesicles. The lung was much heavier than a
normal lung, and there was a great excess of new formed
tissue. The air vesicles were enlarged and their walls ex-
panded, and the capillaries had been multiplied, but there
was no actual development of new air vesicles. This I
may say is consistent with what we find in other forms of
compensatory hypertrophy, such as that of the kidney, in
which there is no increase in the number of glomeruli and
uriniferous tubules, but only in the size and length of these.

The enlarged air vesicles could not compensate completely for the defect of lung tissue, as a large space has less extent of surface proportionately than a small space, and we may conclude that during life this man had less breathing surface than normal. The very striking hypertrophy of the right ventricle which existed, was an indication that the heart was excessively exercised in carrying on the pulmonary circulation. But yet the patient continued to live on to the age of 46.

We may regard this case, then, as determining the existence of hypertrophy of the lung as a pathological condition. I may add that since my case was published there have been two other similar ones recorded by Schuchardt and Reckling-hausen (*Virchow's Archiv*, vol. ci, 1885, p. 71).

This leads to the question whether a local disease of one lung may lead to a true hypertrophy of the remaining sound parts, whether in the same lung or in the other. Læennec mentions that Morgagni observed hypertrophy in connection with compression of the lung from empyema, and asserts for his own part that it is much more general than this author imagined. I am not aware, however, of any exact observations bearing on this point. In the case of phthisis, however, the results of observation on its treatment at high altitudes give us some interesting data.

It may be stated to begin with that residence at consider-ble heights above the sea level causes enlargement of the chest in healthy persons. Williams read a very interesting paper on this subject before the International Medical Congress in London in 1881 (see the *Transactions*, vol. ii, p. 164). In this paper he says—"The large size of the chest of dwellers in high regions has been noted in various parts of the world." "Armieux found a considerable increase of chest circumference in eighty soldiers after a residence

of forty-three days at Barèges [in the Pyrenees, 4,200 feet above the sea level], and a still further increase at the close of four months' stay. The average increase was twenty-five millimetres (or an inch). In the Himalayas Kellet found the European soldiers at Landour (7,300 feet) increase in chest circumference at least an inch during their stay of six months." A similar effect has been noted by Williams himself in the elevated regions of South Africa in one instance.

It may perhaps be questioned by some whether this increase in the size of the chest indicates a real hypertrophy of the lung, and not rather a mere dilatation of the air vesicles, a kind of emphysema. I think the condition is not properly designated by the term emphysema, which implies an over-distension of the air vesicles to such an extent as to interfere with their function. There is really increased function and a permanent enlargement to meet the continued increase of function. This is just what we find in other forms of hypertrophy. The organs generally are capable of meeting temporarily a great increase in their function, but when such increase is continued there is a hypertrophy to meet it. It is consistent with this view that the actual enlargement of the chest is of gradual development. In Kellet's cases the increase was noted after six months, and in Armieux's the increase which was visible after forty-three days was more marked after four months. We may say, I think, that the air vesicles permanently expand without undergoing atrophy and so expose an increased surface to the inspired air.

In the case of phthisis pulmonalis a similar increase in the size of the chest has been observed, but it may be presumed that in order to the occurrence of a true hypertrophy, the affected person must to a considerable extent recover from

the disease in its active form, because, for two reasons, hypertrophy is not likely to occur while the disease is advancing. In the first place, for such new formation a somewhat vigorous state of health is requisite, such as a phthisical patient hardly possesses, and, in the second place, such persons being invalids, do not exercise their respiratory organs so much as healthy people and there is not so much call for hypertrophy. In the paper by Dr. Williams, already quoted from, the expansion of the chest shown by phthisical patients who have been treated at high altitudes is fully considered. At Dr. Williams' request Dr. Ruedi, of Davos, made careful measurements in 105 cases, and found enlargement in ninety-five (90 per cent). From measurements made by himself in patients treated at Davos, Dr. Williams tabulates his conclusions, of which for our purpose the following are the more important:—

" 1. That as a rule the portion of chest wall overlying the healthy lung more frequently undergoes dilatation than that overlying the diseased lung."

" 5. That the length of residence required to produce this thoracic expansion varies in different cases, but that, as a rule, some months are necessary; . . . ."

" 6. That the duration of this expansion after a return to lower levels varies, but, in the majority of patients, is permanent. . . ."

It will appear from what has gone before that the hypertrophy here is related, on the one hand, to the recovery or partial recovery of the patient, implying increased general vigour, and, on the other, to the influence of the high altitude. We should expect that a certain amount of hypertrophy would occur even at low altitudes where recovery takes place, and we believe that this is the actual experience of those who have observed cases before and after a residence

in the Riviera and other southern health resorts near the
sea-level.

That we have to deal with a true hypertrophy here there
can hardly be any doubt. It is certainly different from
emphysema, as the lung tissue is more active than normal,
and it is really an enlargement of the organs from increased
healthy exercise.

SECONDARY PHENOMENA IN THE LUNG; ACUTE PLEURISY AND PNEUMO-THORAX.

It has already been more than once indicated that pleurisy
is an almost constant accompaniment of phthisis. This
applies to chronic pleurisy resulting in adhesion of the
opposed pleural surfaces, and we have already related this
to the extension to the pleura of the diluted products evolved
by the bacilli. This chronic pleurisy, by producing a vascular
connection between the parietal and visceral layers of the
pleura, frequently prevents the extension of the necrosis
in the lung to the pleura. In this sense it is a conservative
process, while the conditions which we have now more
particularly to consider—namely, acute pleurisy and pneumo-
thorax, are intimately related to necrosis, and only occur
when, by reason of the absence of adhesion, the necrosis
has the opportunity of extending to the pleura.

ACUTE PLEURISY.—It is a prevalent belief that acute
pleurisy is often the precursor of phthisis, and that in
persons of a certain age and with the general signs of
ill-health the occurrence of a pleurisy is rather ominous.

Assuming in the meantime that a simple non-tubercular
pleurisy often precedes, and has some part in determining
the occurrence of phthisis, some explanation of this conse-

quence has to be looked for, and it must be confessed that a fitting explanation is difficult to find. Trousseau suggests that the existence of a simple pleurisy, having no intra-pulmonary origin, may so affect the lung as to render it more susceptible of phthisis. In this view of it, the existence of a pleurisy, whatever its origin, has the effect on the lung of a predisposing cause, rendering it a more fitting nidus for the settlement of the tubercular bacillus. I am not in a position to offer any opinion as to the frequency or probable cause of such preliminary pleurisies. I rather suspect that in most cases a tuberculosis of the lung already exists although undiscoverable by physical signs, the more so because pleurisy, as we shall see immediately, is more liable to occur in the early stages before the conservative adhesions have taken place. At any rate, we may say that acute pleurisy, usually of limited extent, is very frequent in phthisis pulmonalis. There are often evidences of its presence on *post-mortem* examination, and at the same time there are frequently indications of its mode of origin.

It is very common in phthisis, especially in acute cases, to observe on *post-mortem* examination a limited necrosis of the pleura affecting in some cases a number of small areas. There is usually associated with this acute pleurisy, which may be limited in area, but may extend to the whole pleural surface in so far as it is not limited by adhesions. This acute pleurisy may or may not be associated with pneumo-thorax.

As the necrosis of the pleura is the cause of the acute pleurisy, it will be proper to consider how the former is produced. While the lymphatic arrangements of the pleura are apparently independent of those of the lungs, and there is therefore no extension by the lymphatics of the proper

tubercular process to the pleura, the same cannot be said of the blood-vessels. The pleura is supplied by branches of the bronchial artery, which also supplies the general connective tissue of the lung and the sub-pleural tissue. This being so, the caseous necrosis of a piece of lung immediately beneath the pleura, involving as it does death of the smaller arteries and capillaries, will cause necrosis of the pleura lying over it, provided no other source of blood-supply exists. We have already seen that, in most cases, the adhesion of the two surfaces of the pleura provides for this additional supply of blood. Nothing is to my mind more striking than the manner in which blood-vessels adapt themselves, by new formation when necessary, to the requirements of the tissues. More particularly is it observable that in inflammatory new-formations vessels are abundantly formed, in communication with the existing ones. The practical result of a pleural adhesion is, that the pulmonary and parietal pleuræ coalesce, and their blood-vessels are brought into immediate communication. When this is the case, then obviously a necrosis of the lung beneath the pleura will not induce a necrosis of the pleura, because the latter will continue to be nourished by the vessels from the parietal layer. Even if a limited necrosis should occur beneath the adhesions, through imperfect communication of the blood-vessels, we shall not have acute pleurisy as a result, because the necrosed surface will be shut off from the general cavity of the pleura by the adhesions.

From these considerations it is clear that we are to look for necrosis of the pleura only where the disease in the lung has extended beyond the adhesions, and where some part of the lesion has its seat immediately beneath the pleura. At the very outset of a caseous phthisis, one of

the affected areas may be immediately beneath the pleura, and we may have a necrosis before there has been time for the formation of adhesions. In this way we may perhaps explain many of the cases in which pleurisy has occurred before there was any evidence of disease in the lungs. In all acute cases of caseous phthisis we are liable to have necrosis of the pleura from a similar cause, and it is not uncommon to find quite a number of little dead white areas visible on the surface, indicating necrosis of the pleura, perhaps concealed in part by the fibrine deposited as a result of the acute inflammation. The comparative frequency of acute pleurisy may be judged of by the fact that it was a prominent feature in 19 cases out of the 186 *post-mortem* examinations on which my tabulated results are based.

It is not quite easy to say how the existence of a necrosed surface should induce an acute inflammation of the pleura, but it seems as a matter of fact to do so, not only when the necrosis has its origin in phthisis, but when it is due to other causes. As an instance of this, I may mention the hæmorrhagic infarction of the lung. In this condition embolism of a branch of the pulmonary artery has induced hæmorrhage into the lung alveoli, usually in a wedge-shaped piece of tissue, the base of the wedge being at the surface of the pleura. The pleural surface, if not actually necrosed, is greatly reduced in its vitality, and, apparently as a consequence of this, an acute or sub-acute inflammation of the pleura results, manifesting itself by the deposition of fibrine and abundant effusion of serum. In the case of the infarction, there can scarcely be a question of micro-organisms extending to the pleura, and this is still further put out of account by the fact that infarction of the spleen induces a some-

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what similar acute peritonitis. In the case of necrosis in the caseous form of phthisis the dead tissue may possibly give more ready passage to juices containing irritating products in solution, and perhaps even to micro-organisms.

If the patient survive, as will mostly be the case, the acute pleurisy will probably subside by degrees, and give place to a chronic inflammation. The results of this, as in ordinary simple pleurisies, is adhesion of the pleura, and in this way we may have obliteration of the pleural sac by general adhesions such as we often meet with in phthisis.

PNEUMO-THORAX also implies necrosis of the pleura, but it implies more than this. There must be in addition a partial separation of the dead piece, and the establishment of a communication between the pleural sac and the air passages. This infers the existence of a cavity beneath the necrosed piece of pleura. If beneath the necrosed pleura there is only a caseous piece of lung, then, although by softening of the caseous matter a cavity may ultimately form, yet before that has happened, adhesion may have occurred, and the danger of perforation may have been averted. But if, on the other hand, a cavity forms rapidly, then along with the softening caseous matter the necrosed pleura is also liable to soften, and may separate at its edge, just as does an ordinary slough. As the cavity communicates with the air passages, an open communication is thus formed, by which air passes into the pleura. It is by the separation of such sloughs of the pleura that, in my experience, pneumo-thorax is brought about.

I have here two preparations from the museum, which illustrate this mode of occurrence. I shall read to you the description of these cases as detailed in the Museum Catalogue :—

"Series iii. No. 27. PNEUMO-THORAX: CAVITIES IN LUNG: SLOUGH OF PLEURA. This preparation illustrates the mode of occurrence of pneumo-thorax. The lung tissue is seen to be largely occupied by cavities, and at one part, the pleura over an area as large as a shilling is dead. In the fresh state it presented a dead white colour, which contrasted
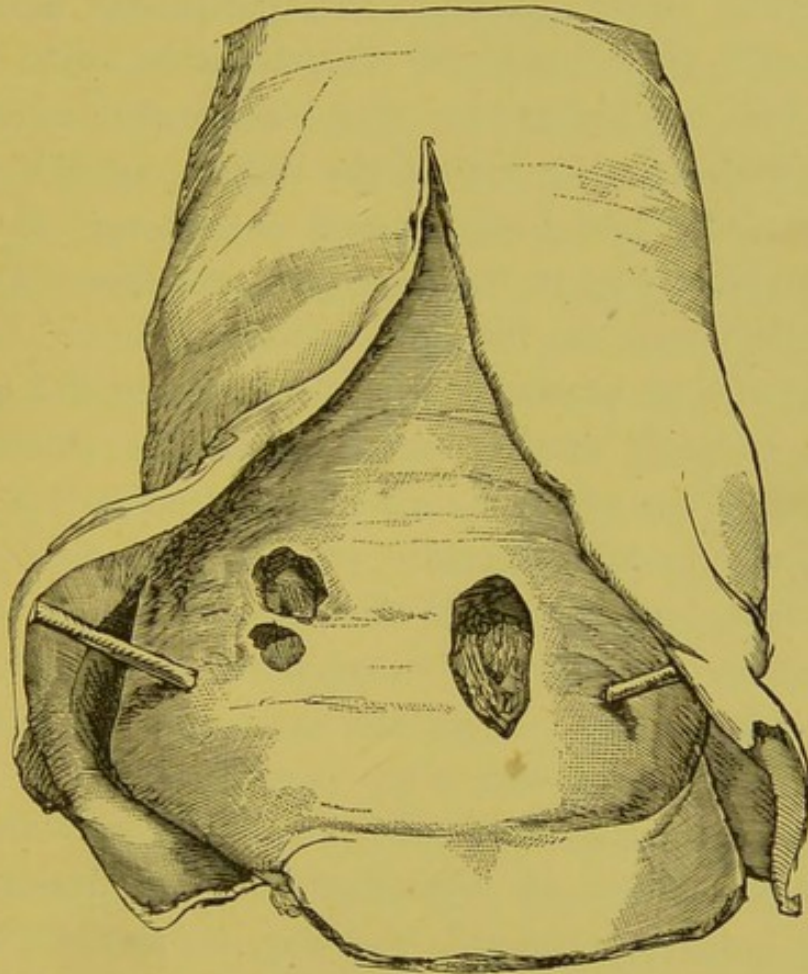
FIG. 20.—Lower part of lung and pleura, from case of pneumo-thorax. There are three apertures caused by necrosis of pulmonary pleura.

with the surrounding tint. At one edge the slough has begun to separate, and a communication is shown between the pleural cavity and the lung. The pleura was covered by a fibrinous exudation, and the sac contained gas and pus.

"Thirteen days before death there was a sudden occurrence of pain in the side corresponding to the lesion, with

great breathlessness, &c. Simultaneously there occurred
amphoric respiration, metallic tinkling, &c., with a sub-
sequent development of splashing sounds on succussion.
The patient was a girl 23 years old, and her phthisical
history went back a year."

"No. 26. PNEUMO-THORAX IN PHTHISIS PULMONALIS. The
preparation shows parietal and pulmonary pleura, the
latter with three apertures communicating with cavities
in the lung (see Figure 20). The borders of these apertures
are rounded and they present the appearance as if portions
of the pleura had been punched out (slough of pleura).
The pleural cavity, in its lower part, was found filled with
air, while, above, the lung was adherent."

It remains to be stated that the occurrence of pneumo-
thorax naturally implies an acute pleurisy as well. The
exposure of the necrosed surface may itself induce pleurisy
before the actual occurrence of the perforation, but the
perforation, by allowing of the discharge into the sac of
the contents of a pulmonary cavity, will lead to a still more
acute inflammation. It will be seen, therefore, that acute
pleurisy and pneumo-thorax are closely related both in their
causation and actual occurrence. It will be observed also that
both of them occur in connection with the advanced guard
of the pulmonary process, resulting, it may be, from an
isolated lesion considerably removed from the main body of
the disease. Hence it often happens that acute pleurisy or
pneumo-thorax occurs in connection with the less affected
lung or portion of lung, and by causing serious damage to
the lung on which the patient mainly depends for respira-
tion, may induce much more serious dyspnœa than if it
had attacked the lung in which the disease was already
far advanced.

Pneumo-thorax was present on no less than 21 occasions

in the 186 *post-mortem* examinations on which my tabulated results are based, and in one of the cases it existed on both sides.

PULMONARY HÆMORRHAGE.

1. EARLY HÆMORRHAGE.—Hæmoptysis is one of the most frequent signs of phthisis in its earlier stages, and it is even in many cases the first circumstance that attracts the attention of the patient. The idea that hæmorrhage may be the starting point of phthisis has established itself to a considerable extent in the public mind as seen in the expression that so-and-so has "burst a blood-vessel," which one often hears in regard to a person who has shown the first symptoms of phthisis. A similar idea has found adherents among clinical observers, and has given rise to the designation of a presumed class of cases under the name of *phthisis ab hæmoptoë*. The name of Niemeyer has been in recent times associated with this view.

In examining the recent lesions in phthisis, both in the fibroid and caseous forms, it is by no means uncommon to meet with alveoli filled with blood, presenting an appearance similar to that in Figure 21. The blood may be so abundant, occupying so many alveoli, as to give to the piece of lung concerned quite the character, in some cases, of the hæmorrhagic infarction. It is difficult to state positively what is the exact mechanism of this hæmorrhage, but it is apparent, at least, that the blood comes from the pulmonary capillaries, escaping evidently by diapedesis, that is to say, without rupture, through the walls of these capillaries. I am led to this conclusion by the fact that the lung alveoli are often homogeneously occupied by blood, being in fact fully distended as in Figure 21. I cannot believe that such

a homogeneous packing of the alveoli could occur, to
the entire exclusion of air, by a process of insufflation such
as has been supposed to occur. A regular leakage of blood
from the capillaries of an alveolus would gradually expel
the air as the alveolus got filled. An insufflation would
give an irregular accumulation in which the contained air
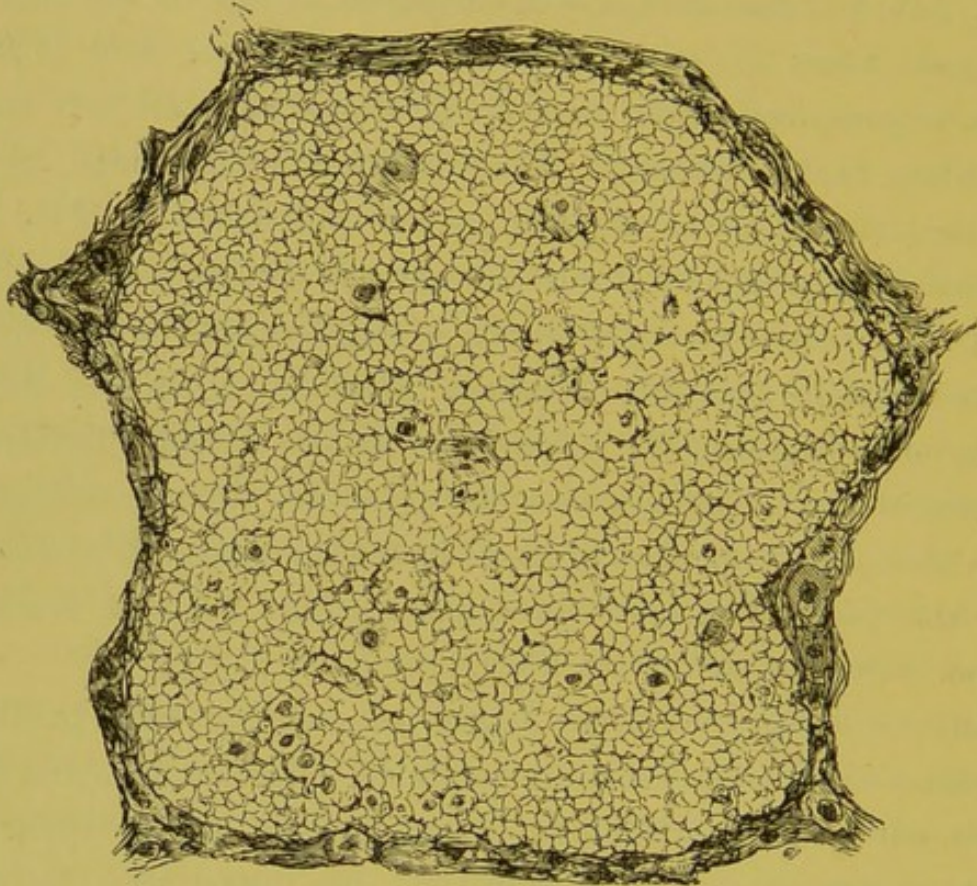
FIG. 21.—Lung alveolus filled with blood. In the midst of the blood corpuscles a few
catarrhal cells are visible, a row of them at lower margin apparently raised from
the wall of the alveolus. × 350.

had broken up the mass of blood. We sometimes find both
of these in the large hæmorrhages of the infarction.
Another fact of importance is, that this hæmorrhage may
be associated with comparatively little evidence of previous
disease in the alveoli. There are usually, as shown in the
figure, a few catarrhal cells mixed with the blood in the
alveoli, but these are not more numerous than in an
ordinary hæmorrhagic infarction. It seems to me, therefore,

that the hæmorrhage here does not arise from any local lesion in the alveoli themselves, more particularly as it occurs simultaneously in a considerable group of alveoli. This would lead to the inference that the cause is to be looked for in a derangement of the circulation affecting small branches of the pulmonary artery. Looking to the cause of the hæmorrhage in the ordinary hæmorrhagic infarction, we may perhaps ascribe it in the present case to obstruction of small branches of the pulmonary artery.

In the ordinary hæmorrhagic infarction the primary condition is embolic obstruction of a branch of the pulmonary artery, this obstruction resulting in an overfilling of the veins and capillaries, and leakage of blood from the latter into the lung alveoli. In the case of early phthisis it is to be remembered that the lesions, as we have so often observed, begin in the smaller bronchial tubes and their walls, and as these are in the immediate neighbourhood of the branches of the pulmonary artery and vein, the swelling of the bronchial wall, and of its surrounding connective tissue may readily lead to interference with the circulation in these vessels. It is a point of some importance that even the terminal branches of the pulmonary artery do not anastomose, but that each supplies a distinct and separate area of lung.

I may here add that a similar kind of hæmorrhage is often to be found in acute general tuberculosis, where the tubercles form in the walls of, and sometimes around, the branches of the pulmonary artery. I have been astonished in some cases to observe the extent to which the alveoli have been filled with blood; I notice that Dr. Hilton Fagge states that, in cases of this kind, "hæmorrhage is no uncommon symptom of that affection, and may even be immediately fatal at a time when there is neither ulceration nor obvious consolidation

of the lung substance, and when the only lesions found *post-mortem* are recent miliary tubercles which had apparently produced no other symptoms whatever."

Another fact which I would place in this connection is, that fat embolism, in which fluid fat absorbed by the veins, say after fracture of a bone, obstructs the finer branches of the pulmonary arteries and the capillaries, is also frequently associated with the presence of blood in the alveoli.

From these considerations, it will appear that the hæmorrhage at the outset of phthisis pulmonalis, and also that which accompanies the process during its advance, is to be related to the primary lesion occurring in and around the finer bronchial tubes. This hæmorrhage usually manifests itself during life in a streaking of the sputum with blood, but it may amount to a considerably larger quantity, in which latter case we may presume that there is some accidental disturbance of the circulation increasing the amount of actual bleeding.

As this hæmorrhage is associated with the earliest lesions, it may occur at a time when no symptoms of disease of the lungs are present, and may be the apparent starting point of the disease, although not really so. It is not improbable, however, that the occurrence of hæmorrhage may accelerate the progress of the disease, as the tubercular bacilli may possibly find in the blood in the alveoli a more suitable nidus, and exhibit a more rapid growth.

2. LATE HÆMORRHAGE.—The hæmorrhage which occurs in the later stages of phthisis, after the formation of cavities, is altogether different in its causation. We have here to do with much more considerable losses of blood, and the hæmorrhage is not infrequently fatal. In these cases the bleeding arises from rupture of considerable branches of the pulmonary artery, which have been partially exposed in the walls of

cavities. As a general rule, the branches of the pulmonary artery in the walls of pulmonary cavities are obliterated, and this is more especially the case in the caseous form. But where obliteration has not completely occurred, the wall of the artery, being unsupported and probably also softened by inflammatory infiltration, may give way. There may, in this way, be a direct rupture of an artery, but more commonly, as I believe, there is in the first place a bulging of the wall so as to form an aneurism. I am confirmed in this opinion from the fact that in seven of my cases in which
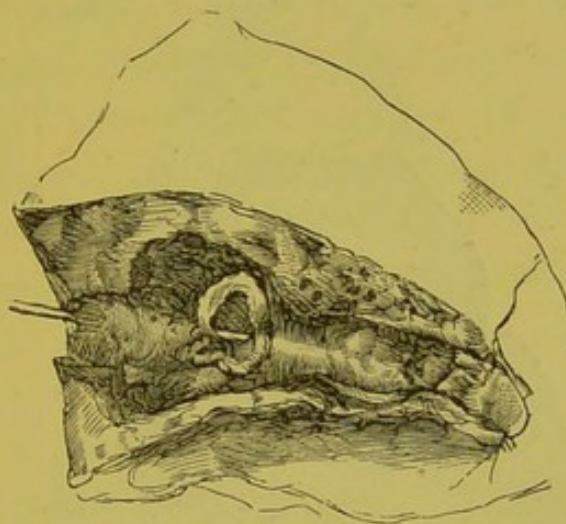
FIG. 22.—Aneurism in a bronchiectatic cavity. A probe has been introduced into the artery, and is visible through the gaping aperture in the aneurism. It is also indicated in the wall of the bronchus beyond the cavity.

death occurred as the result of hæmorrhage, aneurisms were found in the pulmonary cavities in five. I have here these five specimens of aneurisms in pulmonary cavities from the Museum of the Infirmary, and the accompanying illustrations represent two of them. (See Figures 22 and 23.)

These aneurisms may form in the walls of cavities, either in the caseous or in the fibroid form, but their mode of formation is somewhat different in the two cases. In the caseous form, the internal wall of the cavity is the seat of tuberculosis, and probably of progressive ulceration. By this

process the wall of the artery is softened, and the aneurism
is composed of brittle tissue. Two of the examples in the
Museum are of this kind, and in one of them, two aneurisms
were attached to a bridge which spanned a cavity, and they
almost filled the latter. In this case there were for four
months before death, frequent attacks of hæmoptysis, some-
times amounting to as much as 20 or 30 ounces, and
death occurred after a violent discharge of blood which
amounted to 44 ounces.
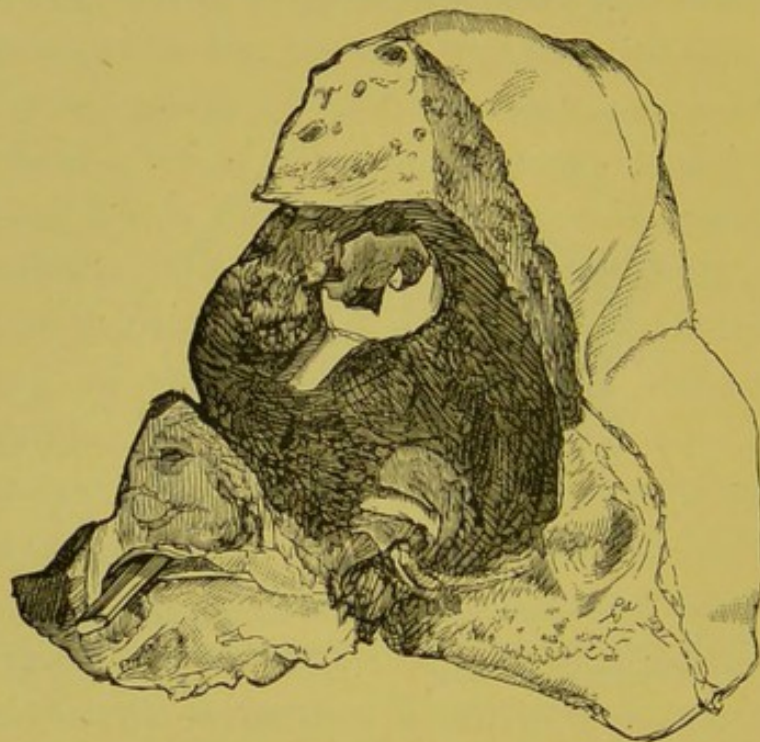
FIG. 23.—Aneurism in a pulmonary cavity. A probe is passed through the artery.

In the fibroid form the cavities, as we have seen, result
from dilatation of bronchial tubes, and the aneurisms have
a somewhat different mode of origin to that just described.
This is indicated in Figure 22. In consequence of the
dilatation of the bronchus, the branch of the pulmonary
artery which, according to the regular arrangement, accom-
panies the bronchus, has to some extent projected into the
cavity, forming a longitudinal ridge in it. The portion of

the artery which lies in the cavity will to some extent lose its natural support and this will apply especially to the aspect of it next to the cavity. This was well seen in the specimen of which the figure is a representation. In it a probe introduced into the artery and passed beyond the aneurism, was so little removed from the interior of the cavity that its colour was visible through the wall of the cavity, and it produced a longitudinal ridge in the course of the latter. Under such circumstances, the unsupported wall of the artery may readily yield and dilatation occur. It will be observed that in this form the wall of the aneurism is not likely to be softened in the same way as in the caseous form. The wall will be thinned, as in all aneurisms, and correspondingly weakened, but it will be composed of sound tissue. In the case referred to the aneurism which resulted is not much less in size than the cavity itself, and it has been torn open so as to present a wide gaping aperture. There was severe hæmorrhage lasting for some days, but ceasing after a time, to be again renewed with fatal results. Looking at the gap in the wall of the aneurism it seems difficult to understand how a hæmorrhage from it could have been stilled before the death of the patient, but examination gave a sufficient explanation. The pulmonary cavity, having resulted from the dilatation of a comparatively small bronchus, had a narrow neck. The blood had coagulated in the confined space of the cavity and had formed a kind of plug or cap over the aneurism. This cap formed of condensed blood, obviously of some duration, was found in the cavity at the *post-mortem* examination.

LECTURE V.

SECONDARY PHENOMENA OUTSIDE THE LUNGS.

Tuberculosis of larynx and trachea; *its frequency; due to infection from sputum. Primary tuberculosis of larynx, cases.* Tuberculosis of intestine, *secondary to that of lungs; localisation and characters of lesions.* Tuberculosis of stomach, *very uncommon; case given in detail.*

Extension by the blood. Acute miliary tuberculosis. *A minor extension by blood in many cases of phthisis; in general tuberculosis extension to pulmonary vein; case detailed.*

Amyloid disease in phthisis. *Its occurrence according to form, sex, &c. The organs affected, spleen (sago spleen distinguished from lardaceous form), liver, kidneys, intestine.*

The kidneys in phthisis. *Local tuberculosis or renal phthisis. Association of amyloid disease and Bright's disease.* Fatty liver. Other complications.

The fever of phthisis. *Elevation of temperature means extraneous matter in the blood. Absorption of dissolved products of bacilli,—also from cavities, and of blood after hæmorrhage.* Emaciation.

TUBERCULOSIS OF THE LARYNX AND TRACHEA.

THESE have been already considered incidentally in relation to the extension of the tuberculosis along the respiratory tract. There is no doubt that in a large proportion of cases the larynx and trachea are affected secondarily to the lung. I am not able from my own observations to say in what proportion of cases tuberculosis of the larynx results from phthisis pulmonalis, but we have two trustworthy sets of observations on this point. Sir Morell

Mackenzie examined with the laryngoscope 100 cases of pulmonary consumption in the London Hospital, and found that the larynx was tubercular in 33 of them.[*] Again, Heinze examined the *post-mortem* register of the Pathological Institute at Leipzig, and found that in 1,226 cases of pulmonary phthisis the larynx was tubercular in 376, or in 30·6 per cent of the cases.[†]   We may infer, therefore, that in about a third of the cases of phthisis pulmonalis the larynx becomes affected before death.

It can hardly be doubted that the sputum is the vehicle by which the infection is conveyed from the lungs to the larynx, as the phthisical sputum contains the tubercular bacillus, often in very large numbers.   In the passage of the sputum from the lungs outwards there are two places where it is liable to make a rather prolonged stay—namely, at the inferior and at the superior extremities of the trachea.   The sputum gathers at the main bronchi or lower part of the trachea, till, by a special effort of coughing, it is carried upwards to the larynx, where it is caught and probably lodged for a time.   It is usually by another effort of coughing that it is brought into the mouth. This applies especially to the erect posture, but, as many patients, for some time before death, are recumbent, the sputum will lie in any part of the trachea, and we often find the latter extensively involved.   It is the case, however, that the two situations mentioned, but especially the larynx, are the special seats of tuberculosis.

The tubercular process, whether in the larynx or trachea, is, to begin with, superficial.   The mucous membrane beneath the epithelium is infiltrated with round cells and tubercles, and there is, not uncommonly, in addition, œdematous

* Morell Mackenzie, *Diseases of the Throat and Nose*, vol. i, p. 366.
† Heinze, *Die Kehlkopfschwindsucht*, Leipzig, 1879.

swelling of the mucous membrane, especially in the larynx.
Ulceration ensues here as in other tubercular lesions of
mucous membranes, and this too is at first superficial.
As the process advances, the infiltration and ulceration
extend more deeply, and in course of time there may be
considerable necrosis, involving sometimes considerable por-
tions of the epiglottis, vocal cords, and other structures.
The ulceration is associated with irregular thickening of
the remaining mucous membrane, so that there may be
considerable projections from the surface. In the trachea
it is not uncommon for the ulceration to cause exposure
of one or more cartilaginous rings, which may be visible
in the floor of the ulcer, as in some of the preparations
which I show you from our museum.

PRIMARY TUBERCULOSIS OF THE LARYNX.—While in the
great majority of cases tuberculosis of the larynx is second-
ary to that of the lungs, the possibility of an infection in
the reverse direction must not be overlooked. The exist-
ence of a primary tuberculosis of the larynx has been
doubted by so high an authority as Heinze, while Morell
Mackenzie, who formerly published some fatal cases, seems
inclined to question his own observations.

From two cases about to be related I have been
induced to believe that it does occur, although it is un-
doubtedly infrequent.

Some years ago I met with a case which impressed
me strongly with the view that a primary tuberculosis
of the larynx may induce a tuberculosis of the lungs.
A man, apparently in fair health, consulted me for a
throat affection. On examination with the laryngoscope,
I found that there was marked thickening of the mucous
membrane, which was to a considerable extent œdematous.

As the man was apparently in moderately good health, and nothing could be found in the lungs, I regarded the case as one of simple chronic inflammation of the larynx. The case went on for months without signs of healing, and before long ulceration ensued and the appearances were obviously those of laryngeal phthisis. At this stage tubercular bacilli were present in the sputum in enormous numbers, more abundantly, I think, than in any sputum that I have examined. All this time no signs of disease in the lungs were discovered. At last the chest began to show signs of condensation at the apices of the lungs, and from the time when this was first detected there was a very rapid extension, with the formation of cavities. Seeing the enormous numbers of bacilli in the sputum, I was not surprised at the rapidity with which the disease progressed in the lungs when once it began there.

If any ambiguity be suspected in this case, it can scarcely exist in regard to a case in which I made a *post-mortem* examination in the year 1878. I shall read to you an extract from the record of the case as entered in the Pathological Register at the time.

D. H., aged 42. There is great emaciation. The larynx is the seat of very extensive ulceration. The entire mucous membrane is superficially ulcerated, and the ulceration extends over the whole of the epiglottis. As a rule the ulcers are superficial, and the mucous membrane is highly infiltrated. On the right side, however, and just about the level of the vocal cords (which are not visible as cords, but involved in the general infiltration and ulceration), there is a deeper ulcer, in which a necrosed piece of cartilage, which is quite loose, presents itself. In the trachea the mucous membrane generally is much infiltrated, and there are three or four ulcers, two of them as large as sixpenny pieces.

These ulcers penetrate through the mucous membrane, and one of them exposes a ring of the trachea.

Both lungs contain large numbers of small pale nodules, which have the characters of condensations resulting from filling of the pulmonary alveoli. On the bronchial mucous membrane miliary tubercles are visible, but no ulceration. There is marked amyloid disease affecting chiefly the liver and spleen.

The man had complained of weakness and loss of appetite for nearly a year. He had cough and spit for 18 months, and hoarseness for 8 months. He was unable to swallow food for some time before death, and latterly even liquids were rejected through the nose. There was considerable dyspnœa, which on several occasions was very severe.

The history of this case and the existence of amyloid disease indicate a somewhat prolonged history, and the condition of the larynx quite corresponds with such a history. On the other hand there was no old lesion in the lungs, merely the earliest stage of caseous phthisis, such as I described in the first lecture. There was no extensive condensation or excavation. There was tuberculosis of the bronchial mucous membrane, such as we find, not in the early stage of phthisis, but in the later stages, and in connection with cavities. It seems clear that the tuberculosis of the bronchi, and the recent tubercular lesions in the lungs, took origin from the tuberculosis of the larynx. There was here a source of infection of similar potency to a tubercular cavity, and the movements of respiration would readily cause the dissemination of the infection.

I may be allowed to suggest that this course of events may be more frequent than is usually supposed. I have myself recently seen a case in which, from the history as given by a very competent observer, I believe that the

tuberculosis began in the larynx. If such cases can be made out at an early enough period before the lung has become involved, surely we have the indications for a thorough clearing out of all the affected structures if not for excision of the larynx.

TUBERCULOSIS OF THE INTESTINE. .

This stands in a somewhat similar category with tuberculosis of the larynx and trachea, and is even more frequent as an accompaniment of phthisis pulmonalis. I have already in a table constructed from my own cases shown that, in at least one-half of these, tubercular ulcers were present in the intestine, and that this applies both to the fibroid and to the caseous form.

TABLE VI.

INTESTINAL ULCERS IN THE TWO FORMS OF PHTHISIS.

	No. of Cases.	Ulcers Present.	None.	Not Noted.	Together.
Caseous,	155	78	46	31	77
Fibroid,	27	13	7	7	14

From one-half to two-thirds may be regarded as the usual proportion of cases in which the intestine is affected secondarily to the lungs.

The structures primarily involved are the closed follicles of the intestine, both the solitary ones and those aggregated in the Peyer's patches. These follicles are small lymphatic structures like lymphatic glands, but without proper connective tissue capsules such as the glands possess. These small lymphatic follicles are at their summits very close to the internal surface of the intestine, being covered only by a layer of epithelium. We may perhaps associate this circumstance with the fact that they form such ready

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receptacles for the tubercular bacilli. It may be remembered also that the same structures in the small intestine, are the primary seats of the bacilli in typhoid fever, the intestinal lesions in this disease almost limiting themselves to the solitary follicles and Peyer's patches.

It is to be understood that as the solitary follicles and those of Peyer's patches are the same in structure, the changes which they present in tuberculosis are precisely similar. There is no homogeneous involvement of the whole of the follicles such as we find, for instance, in scarlet fever, where the morbid poison is in the blood, and reaches the follicles by that medium. The conditions rather suggest the presence in the intestinal contents of a particulate virus, which may or may not be taken into any particular follicle as circumstances determine. Isolated follicles are sometimes picked out in rather a remarkable manner, and with no sort of regularity. We may find, for instance, at the outset of the affection, one or two of the follicles in a Peyer's patch enlarged and caseous while the rest of the patch is normal, or the lesion may be in different stages in the same patch, such as crater-shaped ulcers and enlarged follicles side by side.

While the location of the lesion is to a large extent determined by accidental circumstances, we are able to form some estimate of certain of the conditions which, in a broad way, determine the localisation of the tuberculosis. For one thing, it is noteworthy that the lesion very commonly, though by no means uniformly, assumes its most developed proportions in the neighbourhood of the ileo-cæcal valve, that is to say, in the lower part of the ileum and in the caput cæcum coli. There are several circumstances which appear to have a causal relation to this localisation. I have already, in speaking of tuberculosis of the larynx, referred

to the fact that the sputum, in being expectorated, lingers about the larynx before being finally coughed up. In like manner there seems to be no doubt that the fæces pass much more quickly through the upper part of the small intestine than through the lower part, and that they lie especially in the caput cæcum. In some animals the caput cæcum is a kind of second stomach, and even in man the intestinal contents lie long in this part of the canal, where absorption of fluid, leading to the concentration of the fæces, proceeds. The more prolonged contact of the fæces will favour the infection in these special localities.

Another circumstance which probably has some bearing on this localisation is that these parts are far removed from the stomach. We shall see immediately that tuberculosis of the stomach is very rare, and we shall connect this chiefly with the characters of the gastric juice. The influence of the gastric juice extends beyond the stomach, and so we scarcely ever have tuberculosis in the duodenum or in the extreme upper part of the jejunum.

The changes which present themselves in tuberculosis of the intestine are not difficult of apprehension. The lymphoid follicles first enlarge, the enlargement being due to a great accumulation of round cells. The change is virtually an inflammatory one, but the special characters of the tubercular condition soon show themselves. I do not know how early actual tubercles with giant cells are visible, but very soon caseous necrosis shows itself in the enlarged follicles. It is very common to see in the intestine occasional rounded prominences, some of them with obvious yellow kernels. These are the enlarged follicles with caseous centres. The caseous matter breaks down and an excavation occurs in the centre, the result being a crater-shaped ulcer with overhanging edges. The ulcer enlarges, but always retains

the characters just indicated; that is to say, the infiltration always extends further out than the necrosis, and so the edges of the ulcer are prominent and overhang its base (see Figure 24). The infiltration of the walls of the ulcer consists of round cells and tubercles. These are indicated in the accompanying figure (Figure 25), and it is seen that the coats of the intestine are destroyed by the advance of the infiltration. The infective character of the lesion is often shown by the appearance of white nodules visible to
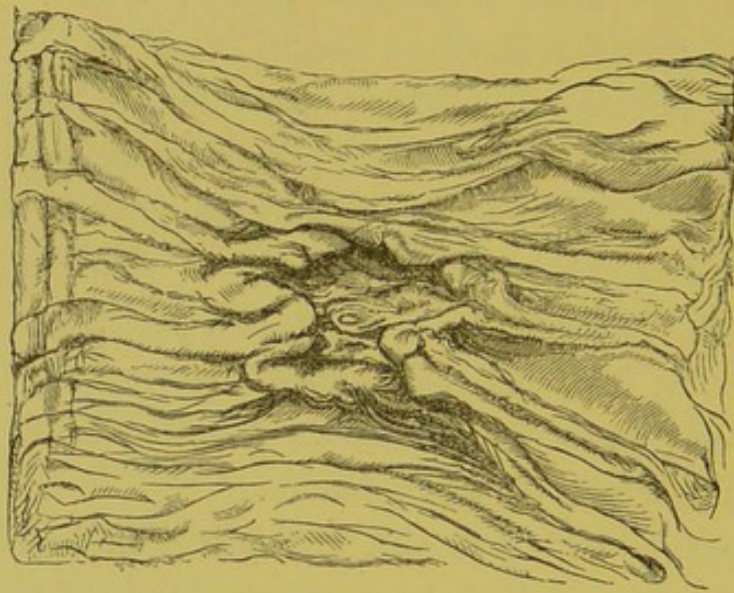
FIG. 24.—Tubercular ulcer of intestine, naked eye appearances. The raised overhanging edges are indicated.

the naked eye beneath the peritoneal coat, it may be at some distance from the ulcer, and separated from it by sound tissue. One of these is shown in Figure 25 at d. The virus is carried by the lymphatics and may be planted beneath the peritoneum at a distance from the ulcer. You are probably aware that tubercular ulceration of the intestine can usually be made out before opening the bowel, by inspection of the peritoneal surface, localised collections of tubercles indicating the existence of ulcers beneath.

It may be added here that necrosis of the peritoneum

sometimes occurs in a similar way to that of the pleura, and sometimes leads to perforation by the separation of the slough. This, however, is a very unusual accident compared with pneumo-thorax; the intestine usually acquires adhesion

FIG. 25.—Microscopic section of small tubercular ulcer of intestine. In the middle there is a crater-shaped ulcer (a) with overhanging edges. The mucous membrane beneath and around is infiltrated with round cells, in the midst of which tubercles are visible. The muscular coat (b) is interrupted by the infiltration. At d there is a small sub-serous tubercle. × 16.

to neighbouring folds so that such an accident cannot occur. In very acute cases, however, there may be numerous necroses of the intestinal peritoneum, as happened in a case which I examined a few weeks ago.

TUBERCULAR ULCERATION OF THE STOMACH.

This, as already mentioned, is a very unusual consequence of phthisis pulmonalis. I have only seen one case of it, and as the condition is so unusual, I may venture to give a fuller account of it. The case was one of advanced phthisis with very extreme emaciation. The man had been under

my own care in the hospital for three months before death, and even on admission he was already in an extreme condition of emaciation. For the last week or two of his life he was a painful sight on account of the weakness, fretfulness, and utter prostration which he presented.

The ulcers in the stomach were numerous and had no special arrangement. They had the overhanging edges and granular base of the tubercular ulcer of the intestine, but were more superficial, had less prominent edges, and less marked excavation. In these respects they approached in character to the tubercular ulcers of the urinary bladder, presenting a condition perhaps midway between these and the ulcers of the intestines. The peritoneal surface of the stomach presented tubercles, usually of some size, and these were very abundant towards the lesser curvature where they were associated with enlarged tubercular lymphatic glands. Tubercular bacilli were found in the walls of the ulcers as well as in the tubercles and affected lymphatic glands outside.

I have already referred to the probable explanation of the rarity of tuberculosis of the stomach. The gastric juice appears to exercise an inhibitory influence on the bacilli, perhaps because of its acid reaction. It is interesting in this connection to find that, according to Koch, the gastric juice is very inimical to the cholera bacillus. It will be remembered that in his experiments on animals he found that this micro-organism is nearly always killed by the secretions of the stomach, and it was only by altering the reaction of these secretions that he succeeded in making the bacilli pass alive through the stomachs of animals. It is clear that in man the stomach secretions are by no means so inimical to the cholera bacillus as in animals, but Koch is probably correct in saying that it is only in certain states of the stomach that they can pass through alive, and that

even in man they rarely multiply vigorously in the stomach. Similarly, the tubercular bacillus is inhibited, as a rule, in the stomach, and only attacks its mucous membrane when the secretions are altered, as in extreme cases of inanition.

Another difference which the stomach presents as compared with the intestine is the absence of the closed follicles, which are, as we have seen, the seat of origin of the tuberculosis in the intestine. This may have an important relation to the rarity of the lesion in the stomach, and it may also partly explain the more superficial character of the ulceration, as well as the lesser degree of infiltration of the walls. In this respect the stomach has considerable resemblance to the urinary bladder, and indeed to other mucous membranes generally.

EXTENSION OF THE TUBERCULOSIS BY THE BLOOD—ACUTE MILIARY TUBERCULOSIS.

We have already commented on the fact that the tubercular process, as a whole, has very little tendency to penetrate deeply, and more especially that it contrasts with most other diseases depending on micro-organisms, in respect that in tuberculosis the micro-organisms are less liable to pass into the blood. It may be added that there is no evidence that the tubercular bacilli multiply in the circulating blood itself. When they find their way into the blood, they produce their further effects by settling down in the various organs of the body, and there producing the regular tubercular lesions.

While in phthisis pulmonalis the tuberculosis is, in the majority of cases, a local disease, affecting primarily the lungs, but extending, as we have seen, to the bronchial glands, air passages and intestine, it is by no means

uncommon to meet with evidences of a limited extension by the blood. In examining the liver microscopically in cases of phthisis, it is not unusual to find tubercles in its substance, although they are too small to be visible to the naked eye. I have the impression that they are most frequently found in the liver when it is the seat of fatty infiltration, but I am not able to state this with any degree of assurance. The tubercles are minute rounded bodies of the usual structure, and apparently of various ages; many of them are the seat of fatty degeneration, as if of comparatively old standing. The appearances suggest that at intervals a few bacilli reach the liver, and settling there produce the usual lesions, but the numbers are so small at a given time that there are few of the same age. We also occasionally meet with a few tubercles in the kidneys. They are of larger size, and visible to the naked eye, but they are much fewer in number. I believe that the conditions of the circulation in the liver favour the settlement of solid granules or bodies such as the bacillus in it. The blood reaching the liver by the portal vein has already passed through the intestinal or other capillaries, and is at a low pressure. The circulation must accordingly be slower than in any other system of vessels in the body. In acute general tuberculosis, as well as in the condition we are now considering, tubercles are usually most abundant in the liver. It may be remembered also that in syphilis the liver is more frequently affected than any other internal organ.

ACUTE GENERAL TUBERCULOSIS presents very different features from those which have just been considered. In it, the tubercular bacillus is present so abundantly in the blood that it is deposited in many organs and produces a simultaneous crop of tubercles in a number of different

positions. This is not a common result of phthisis pulmonalis, but it is one which occasionally occurs.

In acute miliary tuberculosis there is a simultaneous outbreak of innumerable tubercles, and the clinical symptoms have a correspondingly acute character. The phenomena closely resemble those of an acute fever, which ends fatally in a few weeks. As a matter of fact the cases are often mistaken for cases of typhoid fever. From the simultaneous outbreak, we must infer that within a limited period of time there has been the passage into the blood of a large supply of bacilli.

In cases of this disease an older tubercular lesion is always to be found. There is a tubercular structure caseating somewhere. The seat of this is, in the majority of cases, in the lungs, but it may be in the kidneys, in the suprarenal capsules, in the brain, in the lymphatic glands, or elsewhere. The mere discovery of such a caseating centre, however, is not sufficient. It indicates, indeed, that an older source of tubercular infection has existed, but we know that such isolated tubercular lesions may persist for years without leading to acute miliary tuberculosis. It is exceptional for a phthisis pulmonalis to develop into an acute general tuberculosis ; Weigert estimates that it occurs only in about three per cent of such cases.

The first attempt to account for the sudden loading of the blood with tubercular bacilli was based on the discovery by Ponfick of tuberculosis of the thoracic duct. A tuberculosis of the lymphatic glands at the root of the lung, such as we so constantly find in phthisis, may extend by the efferent lymphatics of these glands to the thoracic duct ; and if a tuberculosis of the duct itself occurs, then we may have sufficient virus launched into the blood to produce acute general tuberculosis. Observation has shown,

however, that this tuberculosis of the thoracic duct is not common in cases of acute general tuberculosis, and it was left for Weigert to discover the most common source. If we have a caseating tuberculosis of the wall of a vein, not leading to occlusion by thrombosis, then, if the tubercular bacilli are abundant in it, we may have a sufficient supply of the latter to account for the multitudinous outbreak of miliary tubercles.

I may here state the conditions which, according to Weigert, must be satisfied, before we can draw the conclusion that tuberculosis of a vein is the source of a general tuberculosis. These are—1. The tuberculosis of the vein must, by its structure, and especially by its size and caseous condition, indicate that it is older than the miliary nodules in the organs. 2. The tuberculosis must be in a vein or large lymphatic vessel which remains unobliterated. 3. The virus must extend to the surface of the lesion, and so be in a position to get into the blood. It will be seen that a case of my own, which I now proceed to describe, fulfils these conditions.

Margaret G., aged 7, was admitted to the Western Infirmary on 2nd February, and died on 6th February, 1886. The fatal illness began on 25th January, although she had been failing in health for six weeks previously, as shown by loss of appetite, loss of flesh, paleness, &c. She remained at school, however, till about the 19th January, when a doctor said she had "inflammation of the left side, the right also being affected." She improved on taking to bed, but became suddenly much worse on 25th January. On admission she was affected with dyspnœa, with duskiness of the face, and herpes on lips and nose. The pulse was weak and rapid (148), and the temperature 102·2°. There was little cough and no spit. She was very restless,

tossing about, gnashing her teeth, and crying out. There was a trace of albumen in the urine. Evidences of consolidation were found at the left apex, most marked in front, with abundant fine mucous râles all over the chest. She gradually sank, and died four days after admission.

We have here a history of a preliminary illness extending over six weeks, and after four weeks a diagnosis of an inflammation of the left side. Then there is the sudden development of an acute illness which proved fatal in twelve days. During life, evidences of consolidation were made out at the apex of the left lung.

Post-mortem examination.—"The body is rather spare. The right ventricle of the heart is somewhat dilated, and in both ventricle and auricle there are several globular thrombi, one at the extreme apex of the ventricle of considerable size and softened in the centre, and another of larger size in the anterior part of the auricle, this latter being three-quarters of an inch in diameter. The tricuspid orifice is rather wide, admitting three fingers, while the mitral admits one.

" Both lungs are non-adherent, and on section present, from apex to base, innumerable miliary tubercles, which are so closely set as, in some parts, to be almost continuous. In the upper lobe of the left lung the tubercles are very close, giving place in fact to a tubercular condensation, and there is here also caseous transformation. On making the original section of this lung, a blood-vessel is laid open, which is distinguished as a branch of the pulmonary vein about the size of a large crow-quill. In the wall of this there is an elongated yellow caseous body, which, in its outline, somewhat resembles a thrombus. It is smooth on the surface, and there is no layer of fibrine between it and

the calibre of the vessel, which is here unimpaired, except by the projection of the yellow mass.

"The liver shows on the surface innumerable minute white nodules, some of them supported on little tags of connective tissue. On looking closely minute nodules can be seen beneath the capsule. On section there are tubercles visible, and with the microscope they are found to be very numerous. The kidneys present numerous very small tubercles, many of which are visible at the surface. The spleen presents on its cut surface a number of yellow nodules."

The brain was not examined, as the friends objected.

The facts of the *post-mortem* examination so far agree with the clinical record in respect that we have evidences of an older caseating lesion in the upper lobe of the left lung, and in lungs, liver, kidneys, spleen, evidences of a more recent eruption of tubercles in immense numbers. The tuberculosis of the pulmonary vein, it will be observed, satisfies the conditions which Weigert has laid down. The lesion is of some size and it is caseous, and hence of older standing than the general eruption; it is in a comparatively large vein, in free communication with the blood, the vessel being here unobliterated. It satisfies also the third condition, inasmuch as the tubercular bacillus is abundantly present in the caseous structure occupying the wall of the vein. I have placed under the microscope examples of the tubercular bacillus, and it will be seen that it is in great abundance.

There was in this case, as in some others recorded of a similar nature,* direct evidence of the passage of the

* Weichselbaum : Ueber Tuberkelbacillen im Blute. *Wiener Med. Wochenschr.*, 1884, 12 and 13.

tubercle bacillus into the blood, and its presence there in considerable quantity. In the blood-clot found in the heart after death unequivocal bacilli were found. I only made two cover-glass preparations from the clot, but I found them in both. It required, indeed, considerable searching to find them, but, when we consider the large mass of the blood in relation to the minute fragment of clot used, the discovery of even a few implies that very large numbers were present. They were, however, much more readily detected in the blood in the capillaries and arteries of the lung. In this relation it is interesting that Koch, after injection of large masses of tubercular bacilli directly into the blood of animals, has found the bacilli present inside the white blood corpuscles.

The presence of the tubercular bacillus has been determined during life in the blood of persons affected with acute general tuberculosis. This has been done by Meisels, Lustig, Rütimeyer, and Stricker.*

It will have been inferred from what has gone before that other veins, besides the pulmonary, may be involved in tuberculosis and give passage to the bacillus. Weigert's own cases comprise three with the splenic vein involved, two with portal vein, one with hepatic vein, one with azygos, two with anonyma, one with left internal jugular, one with suprarenal, and one with thyroid. In each case there was an old caseating tuberculosis which had involved the wall of the vein. It appears that in about half of the cases observed the pulmonary vein has been the vessel affected.

It will obviously be henceforth proper, in all cases of acute general tuberculosis, to examine carefully the veins

* See references in a paper by Bergkammer, *Virchow's Archiv*, vol. cii, p. 397.

throughout the body to see if tuberculosis of any of them has occurred. Doubtless there will be a certain proportion of failures, but Weigert's results seem to indicate that the proportion should be small if the search be thorough. It is to be remembered also that, according to an observation by Koch, and a similar one by Bergkammer, the bacilli may extend from lymphatic glands directly to the blood-vessels and so into the blood of the glands. In these cases the walls of the small arteries and veins were infiltrated by bacilli, which sometimes extended through into the calibre, more particularly of the small veins.

We may perhaps infer that, in the cases in which a few tubercles are present in the liver and kidneys, the bacilli have got into the blood by the bronchial lymphatic glands in the way just referred to as having been observed by Koch.

AMYLOID DISEASE IN PHTHISIS.

Phthisis pulmonalis furnishes distinctly the largest proportion of the whole sum of cases of amyloid disease—it is the principal determining cause of that condition.

Turning in the first place to my own cases, we may obtain from them some estimate of the comparative frequency of amyloid disease in phthisis. This is shown in the accompanying table :—

TABLE VII.

	Total Cases.	Amyloid. No.	Amyloid. Per Cent.
Caseous, ...	155	32	20·6
Fibroid, ...	27	10	37
Total, ...	182	42	23

It appears from this that, taking the whole cases of phthisis, amyloid disease was present in less than a fourth.

The figures also show that it was proportionately more frequent in the fibroid than in the caseous form, being present in about one-fifth of the latter, and considerably over one-third of the former. It may therefore be inferred that in the duration of the disease the cases in which amyloid disease has developed approach more nearly to those of the fibroid form than to those of the caseous. This appears in the following table, which is a repetition of one already given, with the addition of the amyloid cases :—

TABLE VIII.

DURATION AT DEATH.

DURATION.	CASEOUS.		FIBROID.		AMYLOID.	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
0 to 3 months,	10	7·15 ⎱	1	4 ⎱	2	5 ⎱
3 ,, 6 ,, .	31	22·15 ⎰	3	12 ⎰	7	17 ⎰
6 ,, 9 ,, .	31	22·15 ⎰ 70·73	2	8 ⎰ 40	5	12 ⎰ 53
9 ,, 12 ,, .	27	19·28 ⎰	4	16 ⎰	8	19 ⎰
1 to 1½ years,	10	7·15 ⎱	3	12 ⎱	4	10 ⎱
1½ ,, 2 ,, .	12	8·58	4	16	7	17
2 ,, 3 ,, .	8	5·7	1	4	4	10
3 ,, 4 ,, .	5	3·57 ⎰ 29·27	1	4 ⎰ 60	2	5 ⎰ 47
4 ,, 5 ,, .	1	0·7	1	4	0	0
Over 5 ,, .	5	3·57 ⎰	5	20 ⎰	2	5 ⎰

Here it is seen that while over 70 per cent of the caseous cases died within a year, only 40 per cent of the fibroid did so; the amyloid cases occupying an intermediate position with 53 per cent.

A similar result is obtained when we take the average duration of the whole cases in each of the two forms, and compare it with the amyloid cases.

TABLE IX.

AVERAGE DURATION IN MONTHS.

Caseous, ...	11·87
Fibroid, ...	35·08
Amyloid, ...	20·

In the number of females affected the amyloid cases, as shown in Tables X and XI, do not show the same tendency as those of the fibroid form, the proportion of females being even greater than in the caseous form. This table brings out, indeed, that both in the caseous and in the fibroid form the number of females affected with amyloid disease is proportionately greater than in the general run of cases of each form. This is especially so in the fibroid form.

TABLE X.

PROPORTION OF SEXES.

	NUMBER.		PERCENTAGE.	
	Male.	Female.	Male.	Female.
Caseous, .	106	48	69·4	30·6
Fibroid, .	22	4	84·6	15·4
Amyloid, .	28	14	66·6	33·3
Total cases, .	128	52	71·1	28·9

TABLE XI.

NUMBER OF AMYLOID CASES IN THE TWO FORMS ACCORDING TO SEX.

	Male.	Female.
Caseous, ...	21	11
Fibroid, ...	7	3

It would appear from these tables as if amyloid disease were more prevalent in the female sex than the male.

The figures are too small to establish a definite conclusion, but it is certainly striking that while our total number of female cases of the fibroid form is only 4, amyloid disease occurred in 3 of them, whereas among 22 males in whom the fibroid form of the disease was present, there were only 6 amyloid.

The following table exhibits the ages at death in the caseous and fibroid forms as compared with the amyloid cases :—

TÁBLE XII.

AGES AT DEATH IN THE TWO FORMS AND AMYLOID CASES.

Age.	Caseous.		Fibroid.		Amyloid.	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
0 to 5 years,	1	0·6 ⎫	0	0 ⎫	0	0· ⎫
6 ,, 10 ,, .	2	1·3	1	4	1	2·5
11 ,, 15 ,, .	3	2·	1	4	0	0·
16 ,, 20 ,, .	22	14·75 ⎬58·35	2	8 ⎬48	5	12· ⎬56
21 ,, 25 ,, .	23	15·5	3	12	4	9·75
26 ,, 30 ,, .	36	24·2 ⎭	5	20 ⎭	13	31·75 ⎭
31 ,, 35 ,, .	16	10·75 ⎫	3	12 ⎫	6	14·75 ⎫
36 ,, 40 ,, .	22	14·75	2	8	6	14·75
41 ,, 45 ,, .	11	7·4 ⎬41·65	2	8 ⎬52	5	12· ⎬44
46 years and upwards	13	8·75 ⎭	6	24 ⎭	1	2·5 ⎭

It will be seen that in regard to age at death the amyloid cases are considerably nearer those of the caseous than those of the fibroid form. This is particularly seen in the higher ages. It appears that, taking the deaths above 40, there were 32 per cent of the fibroid cases who

survived that age, 16 per cent of the caseous cases, and only 14·5 per cent of the amyloid.

We may perhaps sum up these results by saying that amyloid disease is distinctly more frequent in the fibroid than in the caseous form; that the duration of the disease is intermediate between that in the two forms; that it is distinctly more frequent in the female than in the male sex; and that the age at death is nearly the same in the amyloid cases as in those of the caseous form, being very distinctly lower than in those of the fibroid form.

THE ORGANS AFFECTED IN AMYLOID DISEASE.

It is not our function here to enter into the whole pathology of amyloid disease, but it may be proper to pass briefly in review the relation of this process in cases of phthisis pulmonalis as it affects the various organs.

It may be said that the principal organs affected by this condition are the spleen, liver, kidneys, intestine, and lymphatic glands. It will be found in all these situations that the structures chiefly affected are those of the vascular apparatus, and that the disease mostly begins in the walls of the arteries. Hence in the minor degrees in any organ it will often happen that the arteries alone are amyloid. It can also be frequently seen that the amyloid disease affects primarily the middle coat of the arteries, beginning evidently in the muscular fibre cells, so that the arteries frequently present, when stained so as to bring out the amyloid condition, very striking transverse markings, sometimes with the muscular cells distinctly mapped out.

As a general rule, amyloid disease can be readily detected without any reagent in the spleen, even when slight in degree. It produces such characteristic changes there that

the eye of an experienced person is at once attracted. It is my habit at the time of the *post-mortem* to test for amyloid disease by making moderately thin sections of the spleen, liver, or kidney, and treating them with a watery solution of iodine and iodide of potassium (iodine, 10 grs.; iodide of potassium, 20 grs.; water, 4 ounces). This brings out a deep brownish-red colour in the amyloid substance, while producing in the unaffected structures a yellow staining. The same solution is poured on the mucous membrane of the intestine, when the brown colour is soon developed on account of the nearness of the affected capillaries to the surface. Where amyloid disease is regarded as present, the observation is controlled by microscopic examination. Sections for microscopic examination, after being cut by the freezing microtome, are stained in a watery solution of methyl-violet or gentian-violet, and mounted in a solution of grape-sugar. All amyloid structures take on a bright red or rose colour, while the other structures are stained violet.

THE AMYLOID SPLEEN.—There are two forms in which amyloid disease affects the spleen, and it is a remarkable fact that the structures affected in the one form are almost exactly the converse of those in the other. In the one form the pulp of the spleen is affected, and in the other the Malpighian bodies, while in both the smaller arteries are liable to be involved. The pulp of the spleen consists of a congeries of vascular sinuses, and it constitutes the greater part of the mass of the spleen. The Malpighian bodies are small lymphoid structures occurring at intervals around the arteries, and forming localised expansions of the sheaths of the latter. They appear to the naked eye as small white bodies visible on the cut surface of the organ.

In the form of amyloid disease affecting the pulp the lesion is homogeneously diffused throughout the organ, and the latter is much more strikingly changed than it is in the other form. The spleen is enlarged and rendered remarkably solid. The cut surface is smooth and presents a distinctive translucent waxy appearance. This form in fact suggests the name waxy or lardaceous disease, and may be distinguished by these terms. It may also be designated diffuse amyloid disease of the spleen. A microscopic section stained with methyl-violet gives a very beautiful picture—a close net work of rose coloured trabeculæ occupying the greater part of the section, with only here and there an artery with slightly amyloid walls, surrounded by the round cells which constitute the Malpighian bodies, these latter being unaffected and stained of a blue colour, contrasting with the red of the affected structures.

In the other form the disease localises itself in the arteries and Malpighian bodies, the pulp being unaffected. The amyloid condition, therefore, occurs in isolated spots, which, in the early stages at least, are comparatively small, and are usually separated from each other by considerable areas of pulp. The clear translucent amyloid matter gives somewhat the appearance of grains of boiled sago, especially when, by hardening in alcohol, the tissue of the spleen generally has been rendered opaque. It is from this appearance that this form is usually distinguished by the name *sago spleen*. (See Figure 26.)

In the sago spleen the smaller arteries are usually more completely amyloid than in the lardaceous spleen, their coats being sometimes completely replaced by the translucent amyloid substance, while their calibre is diminished by the resulting swelling. This affection of the arteries, however, may be comparatively slight, and the amyloid

change may be almost limited to the Malpighian bodies. In advanced cases, especially after hardening in alcohol, it is impossible to determine what structures of the Malpighian body are affected. There is simply a rounded area of a convoluted or lumpy appearance, which with methyl-violet gives the characteristic colour. I have frequently embraced the opportunity in less advanced cases of examining sections in the recent state. When such sections are cut with the
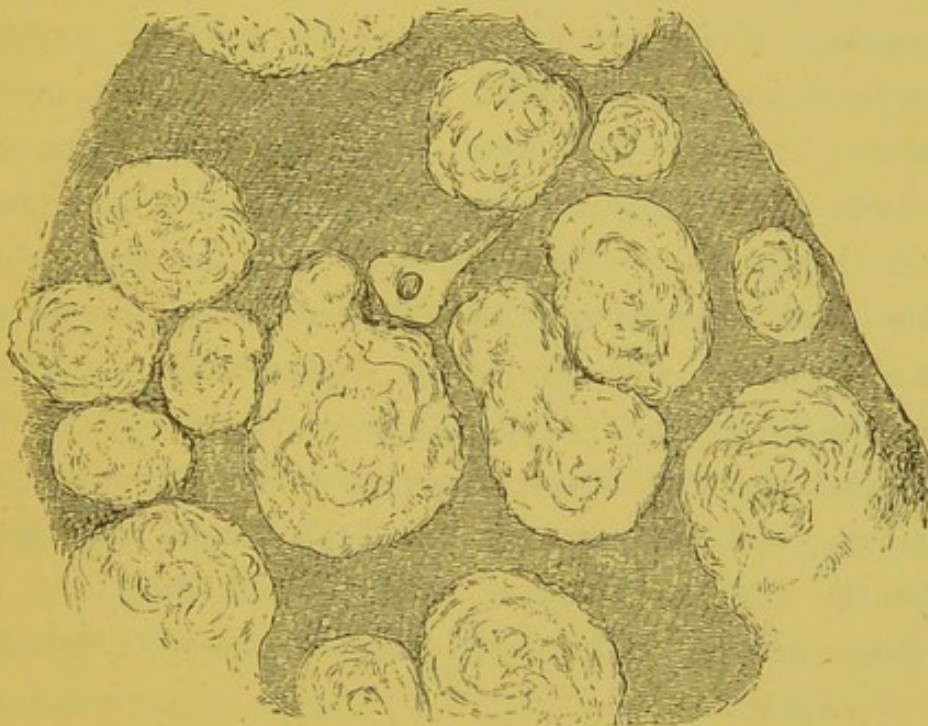
FIG. 26.—Section of sago spleen. The enlarged and translucent amyloid Malpighian bodies are seen. In the middle there is an artery with walls similarly altered. × 20.

freezing microtome and stained with methyl-violet, it is readily seen that the amyloid transformation is present, not in the round or lymphoid cells of the Malpighian body, but in the reticulum between the cells. This reticulum forms in the stained sections a very fine intricate net work of a striking and beautiful character. When sections are made after hardening in alcohol, this fine net work almost completely disappears. In more advanced cases, even when examined in the fresh state, there is much less appearance

of a fine net work, and it is evident that by the swelling
of the amyloid structures the round cells of the Malpighian
body undergo atrophy.

I am unable to say what circumstances determine the
occurrence of the sago spleen and the lardaceous spleen
respectively, but from the record of my cases there can be
no doubt that sago spleen is the form almost universally
met with in phthisis. In my 42 cases of amyloid disease
following phthisis pulmonalis there were 37 in which the
typical sago spleen was present, and there were 3 in which
the spleen was not amyloid. One of the two remaining
cases was doubtful, the record being rather indefinite as to
the form of amyloid disease. There remains only one in
which there was a slight degree of the lardaceous form.
In this case the presence of advanced kidney disease may
have determined the exceptional character of the amyloid
disease in the spleen. In the 37 cases of sago spleen there
were great varieties of size and weight, the largest weighing
16 ounces, and two others 14 and 13 ounces respectively,
while the smallest weighed only $2\frac{1}{4}$, and this in a man
44 years of age. The usual weight, however, was from
5 to 9 ounces.

In three cases, as already mentioned, there was no amy-
loid disease of the spleen, while other organs were affected.
It is worthy of note that in these cases the liver was highly
amyloid, the heaviest liver in the whole series (138 ounces)
being in one of the cases. In the other two the liver was
considerably enlarged by amyloid disease, weighing 92 and
73 ounces respectively. In one of these the amyloid con-
dition was absent from the kidneys as well as from the
spleen. This variability in the distribution of the amyloid
disease in the different organs is a very striking fact which
we shall see exemplified in the case of the liver and kidney

as well as in that of the spleen. It may be here added that, while perhaps in most cases the amyloid condition was in a somewhat similar degree of intensity in all the organs, yet, there were numerous exceptions apart from those in which it was absent from the spleen. In the case of very slight amyloid disease, for example, in which the spleen weighed only $2\frac{1}{4}$ ounces, the liver is noted as very amyloid.

THE AMYLOID LIVER.—In many cases of amyloid disease the liver is not altered, so far as the naked eye appearances are concerned; but under the microscope sections stained with methyl-violet will usually show that at least the branches of the hepatic artery are involved, and, it may be, also the walls of the veins, and to a slight extent the capillaries. In the typical amyloid liver the organ is enlarged and heavy, and its margins are rounded. On section it has a striking waxy appearance, like that of the lardaceous spleen, but is paler in colour, and often with some fatty markings interrupting the homogeneousness of the surface. As the amyloid matter has a greater specific gravity than the proper hepatic substance, the liver weighs more than its size would indicate. It used to be thought that the hepatic cells undergo the amyloid change, but the frequent examination of cases in which the disease was comparatively recent has convinced me that, when it extends beyond the arteries and veins, it is the walls of the capillaries which are attacked. By the swelling of the walls of these vessels, atrophy of the hepatic cells which lie between them is brought about. In advanced cases we may have, on examination of microscopic sections, very little visible beyond amyloid material and the connective tissue supporting the vessels and ducts, the proper secreting tissue having entirely disappeared before the enlarging capillaries.

In my 42 cases of amyloid disease following phthisis pulmonalis, the liver was unaffected in 4. The remaining 38 may be roughly divided into those in which the affection was slight and those in which it was advanced. In the latter category there were 14. The greatest weight presented by an amyloid liver was 8 lbs. 10 oz. (138 oz.), and there were others weighing 132 oz., 117, 96, 94, &c. The lowest weight recorded is 40 oz.

THE AMYLOID KIDNEY.—The structures affected in amyloid disease of the kidney are, at first, the arteries and Malpighian tufts in the cortex, and the straight vessels in the pyramids, but the disease frequently extends to the capillaries of the cortex, and in advanced cases to the basement membrane of the tubules. In my cases there were only 2 in which it was noted that amyloid disease was absent from the kidneys, and in one of those the spleen was also unaffected while the liver was highly amyloid. The greatest weight of a single kidney was $16\frac{1}{2}$ oz., and the least 4 oz. In almost all cases of amyloid disease of the kidneys there were some evidences of inflammation in the structure of the organs, and in a considerable proportion these were such as to give the appearances of a combination of Bright's disease with amyloid degeneration. As the association of disease of the kidney in various forms with phthisis pulmonalis will come up for discussion immediately, it will not be necessary to dwell upon it further here, as the connection between amyloid disease and Bright's disease will be then considered.

AMYLOID DISEASE OF THE INTESTINE.—In almost all cases of amyloid disease, in whatever form, the arteries running in the sub-mucous tissue of the intestine are affected. In

sections treated with methyl-violet, they come out very strikingly, being enlarged and often tortuous. In addition, the degeneration extends to the superficial capillaries, and more particularly to those of the villi, perhaps also, to some extent, to the basement membrane of the mucous coat.

Tuberculosis in the form of ulcers or enlarged follicles (the latter only in one case) was present along with the amyloid disease in nearly half the cases of amyloid disease, which is a similar proportion to that in the cases of phthisis over all.

AFFECTIONS OF THE KIDNEYS IN PHTHISIS.

Albuminuria is well known to be of frequent occurrence in phthisis pulmonalis, and we have now to consider the changes in the kidneys which give rise to it.

There are cases in which the albuminuria may be regarded as symptomatic. The mere acuteness of the fever, implying as it does the presence in the blood of products absorbed from the affected pulmonary structures and their discharge to some extent by the kidneys, induces albuminuria without producing any apparent anatomical change in the kidneys. We may have this kind of albuminuria in various febrile diseases.

TUBERCULOSIS OF THE KIDNEY is an occasional concomitant of phthisis, and it may contribute to the production of albuminuria. I need hardly say that there are two forms of tuberculosis of the kidney. In one of these it is merely a part of a general tuberculosis, and as such I have already referred to it. As the virus is present in the blood, it is carried to the kidneys where it gives origin to numerous small lesions scattered throughout both organs, but having their seats largely in the cortical substance. These lesions appear to the naked eye as small white nodules, and under

the microscope they have the characters of miliary tubercles, generally presenting in their central parts the characters of caseous necrosis. The existence of numerous lesions of this kind may give rise to albuminuria.

The other form is a proper LOCAL TUBERCULOSIS, which is often described as RENAL PHTHISIS, and presents many points of resemblance to pulmonary phthisis. It is frequently met with as an independent disease, a true primary tuberculosis, but it is not an uncommon concomitant of phthisis pulmonalis. In my whole cases of phthisis pulmonalis it occurred 6 times, or in about 3 per cent. All the 6 cases were males. In one of these cases it was associated with an old healing phthisis pulmonalis in which there was a cavity and some cretaceous matter, but no evidence of recent tuberculosis, but in all the others there was an active caseous phthisis, and the tuberculosis of the kidney was of secondary importance to that of the lung. In order to a local tuberculosis of the urino-genitary organs the virus must be conveyed by the blood, and we may presume that in the case where this disease actually develops, these organs are specially susceptible by inheritance or otherwise to this particular form of infection. When an active caseous tuberculosis is progressing in the lungs, then the tubercular bacilli will pass to some extent into the blood, in the way we have already considered, and they will be likely to settle in the kidneys if these organs are predisposed in this direction.

Tuberculosis of the kidney is essentially an ulcerative process. As in the case of other tuberculoses there is first the formation of grey tubercles, which takes place apparently in or near the pelvis or calyces. The tubercles undergo caseous necrosis, and as the lesion advances at its margins there is an increasing caseous area. This softens and dis-

charges into the pelvis or calyx and a caseating cavity is formed. This cavity or ulcer is a progressively advancing one and gradually eats into the kidney tissue, so that the whole organ may be converted into a large caseating cavity. The disease is, in the majority of cases, unilateral, but it is not uncommon to find it advanced in one kidney and at an early stage in the other. In most cases it extends to the ureter and bladder, and in the male, as already mentioned, to the genital tract. In the urinary bladder it produces superficial ulcers, usually of a circular or serpiginous outline, and with small white tubercles at their margins.

BRIGHT'S DISEASE is commonly regarded as including AMYLOID DISEASE of the kidney, and it will be proper for us to consider the relation of the two. It is not to be supposed that amyloid kidneys present lesions altogether distinct and apart from those of the other forms of Bright's disease. If we use the term Bright's disease as indicating either an acute or chronic inflammation of the kidneys, then it may be said that the amyloid kidneys, unless the lesion is very insignificant, always present evidences of the existence of one or other of the forms of nephritis. It would probably be more correct, therefore, to say that amyloid disease of the kidney is usually associated with Bright's disease, than to describe it as a form of the latter.

The changes in the amyloid kidneys indicative of Bright's disease are usually those of chronic interstitial inflammation, the extreme stage of which is seen in the contracted granular kidney, but it is not by any means uncommon to find evidences of active inflammation, the epithelium of the uriniferous tubules being enlarged and fatty. It is indeed difficult at times to determine which is the predominant lesion, the amyloid or the inflammatory.

A similar difficulty is often experienced in the clinical distinction. There are cases in which the urine is large in quantity and albuminous, but without the ordinary evidences of inflammation in the kidneys, and the diagnosis is that of amyloid disease. But there are other cases in which the urine is not in excess, but is highly albuminous, and contains leucocytes and casts in such numbers as to suggest sub-acute nephritis.

While in all my cases of amyloid kidney in phthisis pulmonalis there were evidences on microscopic examination of inflammation in the kidneys, generally chronic interstitial inflammation, there were at least nine out of a total of forty in which the inflammatory changes were so pronounced as to warrant the assertion of a combination of amyloid disease with Bright's disease. Most of these, also, in their clinical features presented the characters of Bright's disease, usually with a history of an acute attack, but sometimes passing into the more chronic form. Among the nine cases we have considerable variety in the condition of the kidneys, but in most there was evidence of tubular nephritis chiefly manifest in the existence of cloudy swelling and fatty degeneration of the renal epithelium. I append a brief summary of each of these nine cases.

CASES OF LARGE SMOOTH KIDNEY WITH FATTY EPITHELIUM.

1. Kidneys weighing $16\frac{1}{2}$ and $14\frac{1}{2}$ oz. highly amyloid. There was the history of an acute attack of vomiting and dropsy about $2\frac{1}{2}$ months before death, the chest symptoms going back $4\frac{1}{2}$ months. The lungs showed numerous cavities and condensations.

2. Kidneys weighing $10\frac{3}{4}$ and $9\frac{3}{4}$ oz. Moderately amyloid, but with extensive cloudy swelling and frequent fatty degen-

eration. History of chest complaint for a year. Anasarca 7 weeks before death. Urine scanty, highly albuminous, and with abundant hyaline and granular casts. Case regarded as phthisis pulmonalis with parenchymatous nephritis. Lungs found to contain many cavities and condensations.

3. Kidneys weighing 10 oz. each. Slight amyloid disease and marked fatty degeneration of the epithelium. History of anasarca beginning 3 months before death. Urine scanty, and highly albuminous, with granular and hyaline casts. Chest symptoms for at least 12 months. In lungs large cavities and condensations, also emphysema.

4. Kidneys weighing $9\frac{3}{4}$ oz. each, only slightly amyloid. In addition the renal epithelium showed frequent fatty degeneration. The history is imperfect, but in addition to chest symptoms, there was anasarca, with highly albuminous urine, in which tube casts were very abundant, many of them containing fatty epithelium. In the lungs there were numerous cavities and extensive condensations.

5. Kidneys weighing 7 and $5\frac{3}{4}$ oz. Slightly amyloid with fatty epithelium. History of phthisis for 2 years. Dropsy latterly. Urine, with considerable albumen and hyaline and fatty casts. Lungs showed very large cavities and extensive condensations.

CASES OF CONTRACTED KIDNEYS.

6. Kidneys weighing 6 oz. each, highly amyloid, but with granular surface and marked interstitial nephritis. Albumen detected in urine 3 months before death. Urine abundant, of low specific gravity and with abundant albumen. Lungs showed numerous cavities and condensations, also emphysema.

7. Kidneys weighing $4\frac{1}{2}$ oz. each. Surface irregular, but

not properly granular, and capsule slightly adherent. Cortex greatly thinned. Kidneys highly amyloid, especially straight vessels of pyramids. Also interstitial infiltration and fatty epithelium. History, that of Bright's disease, beginning with anasarca more than 2 years before death, recurring several times. Urine persistently albuminous. Pulmonary symptoms not of consequence. After death fibroid phthisis not very advanced, but with greatly thickened and adherent pleura and pigmented condensation in lung.

8. Kidneys weighing 4 and 4½ oz., highly amyloid, especially pyramidal portion; cortex irregular and frequently thinned. Surface irregular but not properly granular, capsule non-adherent. History of very chronic phthisis. Anasarca about 5 weeks before death. Had several general dropsical swellings during some of her pregnancies. Lungs showed cavities and condensations.

9. Kidneys weighing 3½ oz. each. There was only slight amyloid disease, but in addition marked fatty degeneration of the renal epithelium. The surface was smooth and the capsule non-adherent. The history is rather that of Bright's disease than of phthisis—namely, anasarca, weakness, scanty albuminous urine. The lungs contained several cavities, mostly of small size. This case was that of a diminutive female, 16 years of age, and the kidneys, although reduced in size, are not so much so as their weight would indicate. It is a case of chronic parenchymatous nephritis, with some contraction of the kidneys.

This frequent association of amyloid disease, with proper nephritis can be no accidental circumstance, and one is inclined at first sight to believe that the explanation must be that the presence of amyloid disease induces, by interfering with the circulation, the phenomena of inflammation.

This is indeed the view taken by Weigert and adopted by Hilton Fagge. The former believes that the amyloid change leads to fatty degeneration of the epithelium, which is followed by the other changes of Bright's disease. I do not think, however, that this relation can be accepted as of universal application. I find one case, for instance, in which, with a prolonged history of Bright's disease, the kidney was found to weigh only 3½ oz., and there was only slight amyloid disease. While assenting, therefore, to the general proposition that amyloid disease is likely to induce inflammatory changes in the kidneys, we may perhaps further assert that the conditions existing in phthisis pulmonalis are in some respects favourable to the development of nephritis. There must be considerable absorption of morbid products in the diseased lungs, as evidenced by the existence of fever, and these products will act, to some extent, on the kidneys. The histories of these nine cases, indeed, would indicate for the most part a sudden onset of nephritis, such as would result from the over-loading of the kidneys with morbid products. We may say therefore, that, while the existence of amyloid disease in the kidneys undoubtedly predisposes to nephritis proper, yet the acute attack is due to some more direct irritation.

Among my cases of phthisis pulmonalis there is only one in which the kidneys showed definite evidences of Bright's disease without amyloid disease. This was a case in which the kidneys were greatly contracted, weighing only 3 oz. each, and they presented the usual lesions of chronic Bright's disease. This case differed from those already given in respect that there was no history of an acute attack. The urine was albuminous but there was no anasarca, and the symptoms were mainly those of phthisis pulmonalis. In fact the case seems to have been a regular one of the granular

contracted kidney complicating an undoubted case of ordinary caseous phthisis.

FATTY INFILTRATION OF THE LIVER.

This is a condition of very common occurrence in phthisis pulmonalis, and it is one of the most difficult to account for. The normal liver contains comparatively little free fat that can be detected by means of the microscope; the hepatic cells are seen to be free from obvious oil drops. In most cases of phthisis, however, there is fat present in the hepatic cells, sometimes in considerable quantity.

FIG. 27.—Fatty infiltration of liver. Hepatic cells with drops of fat in them. × 350.

The fat is in the form of oil drops lying free in the protoplasm of the cell (as seen in Figure 27), and these often assume considerable dimensions. It is clear that the fat is introduced into the cells from without, and brought to the liver by the portal vein. The fat is always first deposited in the cells at the peripheral parts of the hepatic lobules. The obvious explanation of this is, that the circulation of the blood is from the periphery to the centre of the lobule, the capillaries running from the terminal branches of the portal vein, which are distributed around the lobules, to the radicles of the hepatic vein, which occupy their centres. In a comparatively early stage of this condition, this localisation of the infiltration to the peripheral parts of the lobules leads

to a very striking demarcation of these. This is shown in a microscopic section in Figure 28, and it is quite visible to the naked eye, on the surface of the organ when one looks through the capsule, and on section. There are yellow figurings which give the outlines of the lobules. The liver is pale in colour as a whole, in proportion to the amount of fat, and it is also enlarged. Sometimes the enlargement is very marked, and the organ in such cases has a soft, greasy or doughy feeling.

FIG. 28.—Fatty infiltration of liver. Osmic acid preparation as seen with a very low power. The peripheral parts of the lobules are demarcated by the fatty infiltration. × 16.

This accumulation of fat in the liver is a very remarkable fact in phthisis pulmonalis. It is the more remarkable as it is associated with a reduction of the amount of fat everywhere else, a reduction which in some cases is very extreme. I know of no satisfactory explanation of this circumstance. Cohnheim has suggested that it may be related

to a diminution in the function of the liver. Fat is used in the formation of the fatty acids and the cholestearine of the bile, and as the bile is deficient in quantity in phthisis, the fat, being unused, may accumulate. This is not a sufficient explanation, especially in view of the local distribution of the fat at the peripheral parts of the lobules. It seems to me that for some reason the fat absorbed from other structures is accumulated in the liver.

In the artificial production of fatty liver in geese, in order to obtain the delicacy *pâté de foie gras,* the birds are kept in a dark place with little opportunity of moving about, while they are overfed with a paste made of farinaceous food. The consumption of carbo-hydrates within the body is thus reduced to a minimum, while starchy food is richly supplied. It is stated by Larrey that even without food the confinement in close hot cages will induce fatty liver, while emaciation occurs in the rest of the body. These facts would support Dr. Wilks' suggestion, that the fatty condition of the liver in phthisis may be related to the fact that the patients have generally been confined to bed for a considerable time before death. While this may be so, it does not give any explanation of the accumulation of fat in the liver either in phthisis or in the animals treated in the way referred to.

Another view, and one having a considerable appearance of plausibility, has been suggested by Naumann (*Reichert and Du Bois Reymond's Archiv,* 1871, p. 41). It is based on the fact that liver fat is more oxidisable than ordinary fats. It is suggested, therefore, that it may be a function of the liver to prepare the fats for oxidation, and that in phthisis the accumulation of fat in the liver may be with a view to the supply of more oxidisable fats for the purposes of respiration. In support of his view Naumann cites the fact that

the size of the liver in the various orders of vertebrate animals is very nearly in inverse proportion to the activity of respiration, being largest in birds and smallest in fishes. The well known therapeutic use of liver oils, especially of cod liver oil, is another argument in favour of Naumann's view.

It may be added that the fatty and amyloid conditions of the liver are by no means mutually exclusive of each other. The amyloid affection, being in the capillaries, may coexist with a fatty condition of the hepatic cells. In extreme cases, however, in which, as we have seen, the hepatic cells are atrophied, the fat will, of course, disappear with the cells.

OTHER COMPLICATIONS.

TUBERCULAR PERITONITIS.—This also is an occasional accompaniment of phthisis pulmonalis, having occurred in five of my cases. In this form of tuberculosis the bacilli may find access to the peritoneum from tubercular ulcers in the intestine. This, as we have already seen, is a very unusual mode of extension, as is shown by the fact that it did not occur in more than five of our cases. It presents the usual features—namely, large flat yellow tubercles in the midst of adhesions composed of new-formed connective tissue. In the female, the tuberculosis commonly extends to the Fallopian tubes and uterus and it did so in two of my cases.

TUBERCULAR MENINGITIS.—This does not frequently complicate phthisis pulmonalis, unless as part of an acute general tuberculosis. I find, however, that in two cases it did so without there being any signs of general tuberculosis. In these cases as in those connected with general tuberculosis the bacilli must be carried by the blood to the meninges.

FEVER.

As fever is one of the most constant concomitants of phthisis pulmonalis, its consideration can scarcely be omitted in studying the pathology of this disease. It is very variable in its degree in different cases. In very chronic cases, especially of the fibroid form, there is little or no elevation of temperature, whereas in acute cases the temperature may be constantly above normal, and it sometimes runs very high.

It is a familiar and striking fact that the evening temperatures in phthisis are nearly always higher than those of the morning; the latter may be normal, the only febrile ones being those of the evening. The common evening exacerbation is, however, subject to exception. I had under my care in the Infirmary a man affected with phthisis, whose temperature for a number of weeks was regularly high in the morning and low in the evening. As this man was a sailor and had to attend to his watches night and day, it is possible that the periodicity of his bodily functions may have been altered by his habits.

In endeavouring to account for the pyrexia in phthisis, it is necessary to bear in mind certain facts in connection with the causation of fever in general. I do not here go into the question of the exact nature of pyrexia, whether it is really a curative process, the temperature being sustained at a higher pitch in order to get rid of extraneous matters, or whether it is the direct result of the presence of extraneous matters in the blood. It is sufficient for our purpose that in all cases of febrile elevation of temperature, with the exception of obscure cases in which lesions of the nervous system are concerned, the cause of the fever is to be found in the presence of some matters in the blood which are extraneous to it. Fever may be most readily induced in

animals by the injection of septic material, and we have in man many cases in which a similar mode of production can obviously be traced. The fever accompanying acute suppurations, for instance, is due to the absorption of septic products formed at the seat of the suppurations. These products are formed by the micro-organisms which are to be found in all cases of acute suppurative inflammation. It is by no means necessary that the micro-organisms themselves should be absorbed, it is really the chemical products evolved by them which induce the febrile phenomena. In such cases the fever can usually be stopped by providing a free outlet to the pus, and so hindering the absorption of the products. Fever arises in erysipelas in a similar fashion. Here also we have micro-organisms, which confine themselves for the most part to the skin and subcutaneous tissue, but their products, passing by the lymphatics into the general circulation, lead to pyrexia.

The agents causing fever need not be the products evolved by micro-organisms. There are other extraneous matters which, when absorbed into the blood, will induce fever. This is somewhat strikingly brought out by the fact observed by Volkmann, that in simple fractures of bones there is very often febrile elevation of temperature. This observer found that in fourteen cases of ordinary fracture of the thigh, eleven had fever. In five it lasted for several days between 39° and 40° C. (102°-104° F.) and in four it continued for ten days. There is also the fact pointed out by Volkmann, that fever is not always absent in the case of wounds which have been successfully treated by Lister's method, in which there could be no question of micro-organisms or their products. In fractures and in wounds the tissues are torn and blood is effused amongst them, and we must relate the elevation of temperature to the absorption of the products resulting from

the changes in the effused blood. These products are extraneous to the circulating blood and induce fever.

In the case of phthisis pulmonalis we have to consider what kinds of extraneous matter find their way into the blood. For the most part it cannot be said that the tubercular bacilli themselves, by their presence in the blood, lead to fever. We have seen that in ordinary course very few of these get into the blood, and that there is no reason to believe that they are capable of multiplying there. In the case of acute general tuberculosis the fever is to be related to the extension of the bacilli by the blood and to the occurrence of multitudinous centres of tuberculosis in various organs. It may be inferred that it is the multitudinous local tubercular processes which induce the fever and not the mere presence of the bacilli in the blood. The fever in this case, moreover, has a somewhat different type from that in ordinary phthisis. It shows much less of the hectic character, the morning and evening temperatures presenting a much less degree of difference than in ordinary hectic, and the fever having more of the aspects of an ordinary continued fever.

We must look for the cause of the fever in phthisis to the local processes, and consider what extraneous matters may find their way into the blood from them. I have already spoken of pleurisy as due to the action of the products of the tubercular bacillus, diluted more or less as they are removed from the immediate proximity of the bacilli. These products also find their way, more or less diluted, into the blood. They are drained off chiefly by the lymphatics. We know that the tubercular process itself extends by the lymphatics, and that the bacilli are regularly carried to the bronchial lymphatic glands, where they lead to tuberculosis. The lymphatic glands usually serve as filters, stopping the further progress of the bacilli; but they

do not hinder the passage of dissolved products, which are carried on in regular course to the thoracic duct, and so into the blood. It is clear that, the more acute the process, the more abundant are the products evolved by the bacilli, and consequently the greater is the fever.

Besides this, which we may consider to be the regular mode in which fever is produced in phthisis, there are several other subordinate methods. When once cavities have formed, their contents may undergo various kinds of decomposition. I have placed under the microscopes a number of preparations of the sputum in phthisical cases, prepared so as to show, not the tubercular bacillus, but other more common micro-organisms. It will be seen that abundant examples of a considerable variety of kinds are present. It is not to be inferred that none of these have grown since the sputum left the lungs, but yet there can be no doubt that, in cavities which stand in open communication with the air, we may have decomposition of the contents in the ordinary way, and absorption of the products may occur. It is not uncommon, indeed, to find the sputum with distinct traces of a putrid odour, and this may even be so strong as to suggest gangrene of the lung. These remarks apply to bronchiectatic cavities, such as occur in the fibroid form, as well as to those of caseous origin, and perhaps even more to the former, as the wall of a bronchiectatic cavity is probably in a more fit state for absorption than that of one originating in caseous necrosis.

Another frequent subsidiary cause of fever in phthisis is acute pleurisy, which we have seen to accompany the process so frequently. The pleurisy is associated with effusion of fluid which deposits fibrine on the pleural surfaces; pus corpuscles are present in the fluid as well as in the fibrine. These products of inflammation are liable, in various states of change, to absorption, and they add to the causes of fever.

It is not necessary that the pleurisy should be septic in its character, I believe that it is seldom so; it is enough that extraneous matters pass into the blood.

Of similar import is the fever which usually follows hæmorrhage. We have seen that blood is often present in the lung alveoli, and here as elsewhere it is liable to changes resulting in its disintegration. The resulting products may, as in the case of simple fractures, induce fever. It is a well known fact of observation that an accession of fever commonly follows hæmoptysis, and we may find an explanation of this in what has just been stated. I am convinced, however, that hæmorrhage into the lung alveoli is of frequent occurrence without any appearance of blood in the sputum, and it is not improbable that in the ordinary course of phthisis, small hæmorrhages have a considerable share in the production of fever.

EMACIATION.—The loss of weight, which is so striking a feature in phthisis pulmonalis, is, for the most part, to be directly related to the fever. The emaciation is generally the more rapid the more acute the case, and the higher the temperature. I may, indeed, venture the apparently paradoxical statement that, taking the conditions as seen *post-mortem*, the shorter the duration of the case, the greater is the emaciation. This statement, however, is to be received with considerable reservation, because cases which are of long duration very often become acute towards the end, and in that case emaciation may be very marked at the time of death. Indeed it is very seldom that emaciation is not markedly present in the body at *post-mortem* examination.

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