Tubercule.

Tubercule dans les
organes à organisation
compliquée : glandulaire

Poumon,

— : Testicule,

— Rein : Foie.

Le Tribunal militaire
dans le roman.

Von einer nennenswerthen
Zeitigen expectoration der
Alveolarsepta ist bei
der Viruspneumonie
Kinderspneumonie so
wenig die Rede wie
der Croupoy. Pneumon.
der Erwachsenen.

Physiologie. f. 35 a.

Rein. — mémoire — 1.1.40

— Grauthe. — développement dans les gaines lymphatiques.

— Maltosky. le début se fait dans la gaine lympho conjonctive qui entoure le conduit séminifère.
— en même temps la lumière du conduit se bouche par des accumulations épithéliales.

— dans le Rein. c'est autour des tubes urinifères

— dans le foie — autour des canaux biliaires et des vaisseaux.
— Ils sont interlobulaires.

— fig. 1. pl. II. de Harvey
chez un cobaye 3 mois —
— par 2 cellules géantes.

Cornil. 6.723.

— Les granulations grises occupent habituellement les alvéoles d'un infundibule —

Les alvéoles sont remplis par la néoformation, tandis que les cloisons se laissent encore reconnaître par leurs faisceaux de fibres élastiques

— L'infundibule forme un nodule dont le centre est déjà en voie d'atrophie caséeuse.

— en dehors les alvéoles présentent les signes de l'[illegible] catarrhale.

comment se fait l'envahissement des cavités :

1°, Épaississ^t des cloisons
2° Desquamation de l'épithélium
3° Leucocytes émigrés } ?

— Mais comment démontrer sur le bourgeonnement — figure à la périphérie d'une nodosité tuberculeuse — alvéol capillaires remplis —

7e Série : fig. XXVI et XXVII

Enfant mort de Tuberc. miliaire aiguë

— Le tissu pulmonaire qui circonscrit le tubercule représente à du tissu adénoïde

Les cavités alvéolaires sont occupées par des cellules géantes qui sont en connexion avec les septa alvéolaires cicatrisés.

— Les septa consistent pr. la plupart en un tissu fibreux avec de nombreux noyaux

à un fort grossissement ce sont des cellules rendues dont la plupart sont fibreuses.

— La fig. 27. Représente la cellule géante avec ses lieux d'adhérence à la paroi.

Rindfleisch. p. 364. dern. colum.

Le tubercule miliaire ressemble à la forme au voisinage d'un vaisseau. (fig. 144.)

— c'est une masse de la cond. complètement molle.
— en partie de 3 à 7 ans.
— Il y a de 1 à 20 Tub. sur un point carré.

— Siège c'est le tissu conjonctif des poumons. — qq. auteurs que Deichler l'a vu ce sont exclusivement les gaines des petites artères et veines — c'est le tissu conjonctif interalvéolaire et le tissu interlobulaire

Pour la structure analog. avec les tubercules miliaires des autres organes.

— Dans les plus gros nodules on trouve plusieurs centres de développt, autour desquels les cellules tuberculeuses ont un arrangt concentrique, tandis que les autres cellules forment des tractus intermédiaire entre ces centres.

à la périphérie des nodules l'épaisseur des parois alvéolaires est remarquable. Il est clair que quand le processus se développe, ce n'est pas par remplissage des cavités alvéolaires, mais par infiltration des parois alvéolaires.

Les lumières des alvéoles quand il n'y a pas de communicat. catarrhale sont seulement plus ou moins aplaties, ou comblées par l'envahissement du processus tuberculeux. Dans un gros tubercule il y a certains un certain nombre d'alvéoles ainsi oblitérés. Mais cette oblitération n'est pas le fait capital comme dans la Pn. catarrhale.

- Dans les parois aussi que plus de tous les parois des vaisseaux qui semblent jouer le rôle capital. - Voir la planche de Colberg. Tous les petits vaisseaux sont comblés de cellules qui prendraient des parois des capillaires. -

Il peut y avoir eu une affection concomitante catarrhale des alvéoles ou ... d'où une pneumonie catarrhale lobaire. De telle sorte que la tuberculose y avait été là une cause provocatrice.

Emphysème: Bronchopn. p. 225

La tuberculose miliaire aiguë généralisée, est en 1re ligne un signe de malad. constitutionnelle.

De plus le tuberc. disséminé, produit dans les organes où il se développe des transformations pathologiques — mais dans la 2e série, dans les poumons il est un excitant des inflammations d'alvéoles et des membranes.

— Dans le poumon. emphysème et emphysème —

déjà à l'œil nu les tub. miliaires ont là des contours anguleux — ces angles sont les septa interalvéolaires qui sont en état ± prononcé d'inflammat. scrofuleuse — l'injection ne pénètre pas dans ces espaces alvéolaires anémiés.

à leur surface on voit souvent des Riesenzellen. Sur chaque coupe de tubercule on trouve de 5 à 7 de ces cellules, qui ici, sont des cellules, sont dans la lumière de capillaires superficiels.

— Si nous pénétrons dans l'intérieur des petites masses, nous voyons les septa interalvéolaires infiltrés, se prolonger dans une 2e série, un 3e rang, d'alvéoles également infiltrés et au centre on trouve une petite branche de l'artère pulmonaire.

Ainsi les parois alvéolaires épaissies jusqu'au point d'oblitérer la lumière de l'alvéole forment la partie essentielle de la néoformation.

Le tissu périvasculaire, participe

a La Pneumonie.

Le tubercule pulmonaire est donc un petit foyer circonscrit d'inflammation cellulaire.

— Le contenu des cellules granuleuses. Le poumon autour des artérioles pulmonaires.

Le tubercule miliaire n'est donc qu'un petit foyer d'inflammation chez un scrofuleux. —

Lindfleisch 0.245 = Manuel.

Dans le poumon

Chaque tubercule du poumon contient une ou plusieurs coupes de conduits aériens – bronchioles ou cond. alvéolaires.

Ces coupes sont au centre de 2-5 divis. secondaires qui donnent un contour lobulé.

Les parois de canaux sont épaissies par un infiltrat en zones concentriques de cellules, gg. grosses cell. scrofuleuses.

— Pas de masses purulentes – Il y a une homogénéification particulière de l'infiltrat sorte de dégénérat. vitreuse des cellules analog. à l'amyloïde.

Il est probable que c'est une dégéner. analogue à l'amyloïde – En caséification les contours des cellules se fondent entre elles –

Les cellules meurent –

— Plus en dedans vers la partie centrale c'est une trouble grenue, ou caséification des infiltrats.

Quant au ramollissement on peut dire que plus le diamètre du nodule est grand et plus il a acquis ce diamètre rapidement plus est à craindre le ramollissement rapide.

— Le nodule ordinaire formé par la réunion de plusieurs nodules allant jusqu'à la grosseur d'une noisette avant qu'il y ait ramollissement

— le processus de ramollissement est dû à une simple dissociation aqueuse – les cellules mortes et leur détritus sont suspendus dans une certaine qté de liquide qui apparaît au milieu du nodule.

Je pense qu'une partie des corps albuminoïdes subissent par une lente digestion à 37,5 [illegible] qui les rendent solubles et prennent alors d'autant [illegible] d'eau qu'il en faut pour leur solution –

— Cela commence au milieu du noyau caséeux. Il se forme une boue comme purulente.

— Die Riesenzellen der Tuberkel „verunglückte Gefässanlagen – gewissermassen das angehäufte Baumaterial für eine entsprechende Zahl „nicht entstandener Gefässchlingen.

— de là résulte la non vascularisat. du tubercule

Note sur le siège et la structure
des granulations tuberculeuses du testicule.
Malassez. archives de Physiologie. 1876. 1er janv.

- Tubercules aigus.

1°. granulations les petites { élémentaire primitive. renfermé avec 1 seul tube ou deux-trois seulement
tandis que les plus grosses, il paraît peu [illegible] un groupe de tubes.

A. Petites — au niveau de granul. la cavité du tube séminifère oblitérée —
cellules épithél. propres dégénérées — paroi infiltrée de petites cellules rondes
— la paroi dégénère également — mais on la reconnaît. Ces granul., parce que
la tubes. conjonctive résiste mieux aux processus dégénératifs.

— la pér. périphérique est composée de petites cellules rondes, dégénérées dans les
parties les plus centrales

— on sait que tubes séminifères revêtus d'un épithélium analogue à [illegible] —

B. Plus volumineuses —
— voisinage immédiat de granulation —
Zone péritubérculeuse. —
{ a. Tubes séminif. aplatis — lumen remplie par
épithélium atrophié
paroi épaissie, infiltrée d'élém. cellulaires
b. Tissu interstitiel — petites cellules et
tissu conjonctif de formation nouvelle.

3 Zone périphérique — zone jaunâtre —
— formée de cercles clairs entourant des centres plus foncés — il y en a 12 à 15 par
granulations — ces cercles = tissu conjonctif fasciculé — ses rapports avec la
zone péritubérculeuse qui est peut être plus riche en cellules —
au centre — cellules épithélioïdes
ce sont des tubes séminifères altérés.
Parois — au centre cellules petites
— plus grosses — cellules épithélioïdes, forme
de l'épithélium.

La Rosengelle. tout [illegible] au mélange des centres dégénérés.
comme une avant-garde.

— Centre dégénéré — presque pas inerme —
— au centre une figure en Q. cercle en ellipsoïde.
— au centre, tout du granulat. prennent complètement d[illegible]

Tubercule d'enfant de II mois, plusieurs.
— au pourtour. petites cell. rondes. et qq. part. de tissu conjonctif — au milieu
cellules géantes. altérées.
En dedans, [illegible], et dégénérat. granuleuse.
— au centre plusieurs cellules [illegible] altérées.

viscera were healthy. The child was deaf, but very intelligent.

The other two children, aged four and two years respectively, presented the enlarged gums and slight cutaneous affection, and the elder child had only recently exhibited an enlargement of the end of one of the fingers. This child presented also certain peculiar mental and moral phenomena, and gave little promise of speaking well.

The disease in all three cases had commenced a few months after birth. The eldest child of the family, a boy ten years old, presented none of the symptoms found in the others. The parents were first cousins, but healthy. There was no evidence of syphilis or scrofula in them or in their history. The children's grandmother was stated to have died of consumption, but with this exception the members of the family were long-lived and remarkably healthy. There was no similar affection to that of the patients in the parents or any of their relations. The three affected children were born in a wretched and damp dwelling, and exposed to bad hygienic conditions most of their life. The eldest child of affected child resided with the rest of the family, but was during the early part of his life not subjected to the same unfavourable hygienic conditions as the patients. The whole family had, however, for the past year lived in a good habitation and otherwise comfortably, still the disease advanced in all the affected children. Several of the subcutaneous tumours had been removed, and exhibited microscopic characters allied to fibroma, with cartilaginous-looking structure in parts. Dr. Murray considered that the disease should be placed in the group of molluscum fibrosum. The enlargement of the gums and ends of the fingers, the deafness, mental and moral symptoms present in one or more of the cases, the symmetrical character of the affection, and particularly the occurrence of three cases in one family, were remarkable and in many respects unique features in the disease. He considered that the bad hygienic condition in which the patients had been placed may have acted as an exciting cause of the affection, but he thought the predisposing, if not the sole factor, to be the blood-relationship existing between the parents.

REPORT OF A CASE OF "MOLLUSCUM FIBROSUM" OR "FIBROMA," WITH OBSERVATIONS.

BY GEORGE POLLOCK, F.R.C.S.,
SURGEON TO ST. GEORGE'S HOSPITAL.

A woman aged thirty-three was admitted into St. George's Hospital with tumours of the skin, such as are known as "molluscum fibrosum," or "fibroma," and consisting apparently of excessive hypertrophy of the connective tissue. The patient had been the subject of these growths from childhood; they were small in their early condition, but had been slowly growing and increasing in number. The tumours occupy various positions; there are three large ones, and over one hundred smaller ones of various sizes, in different parts of the body; some as small as a split pea. One large one is attached to the back of the head, and is of the size of a small melon. Another occupies a space over the right shoulder, between it and the root of the neck. The most remarkable and the largest commences on the right side of the neck, by a pointed extremity, and is seen to extend below the umbilicus. Its attachment extends from the above point to the upper margin of the right mamma, and increases in breadth as it is traced from the neck to the breast. It consists of a long, thick, and broad pendulous flap of skin, about eighteen inches in length, the anterior surface is thrown into several folds, which give it somewhat the appearance of coils of intestine. The general colour of the skin covering the larger tumours is darker than that of the rest of the body; its surface coarser and more rough, and pretty uniformly marked by obstructed orifices of sebaceous follicles. Sensation over the larger part of this large mass is impaired. A slight touch over the greater portion of the surface is not detected, but more severe handling is readily felt. In addition to these three large masses, various other tumours occupy the trunk; some solitary, some clustered; some with broad base, others pedunculated; one may be seen on the forehead almost flattened, while on the forearms are many suspicious spots of commencing future growths. One rather larger on the front part of the neck was partially removed, but has since increased in size.

When admitted the patient was in a very low state of health, and the removal of the portion alluded to was followed by a good deal of suppuration; but her health being greatly improved since then, removal of a large portion of the largest growth was proposed, and was much wished by the patient.

A microscopic examination by Dr. Whipham of the portion removed, as also of a second small tumour which was snipped off, gave the following results. The tumours were entirely covered by layers of epidermis and rete mucosum, resembling in every respect that of healthy skin. Immediately beneath and closely connected with the rete was a layer of wavy, well-defined fibrous, mixed with a small amount of yellow elastic tissue, which in many places exceeded in thickness that of the epidermis and rete together. Proceeding inwards, the fibrous tissue which constituted its bulk became split up into separate wavy bands, varying considerably in thickness; and between these bands was an abundant growth of small, round, or oval cells, which were closely aggregated in large groups, or arranged in lines between delicate strips of fibrous tissue. In the central parts of the tumour, fibrous tissue, mixed with yellow elastic tissue, abounded; the fibrous tissue was less dense and more wavy, was split up into more distinct bands, separated from one another by wider interspaces, which interspaces were either empty or occupied by the cell-growth above mentioned. The tumour was well supplied by blood-vessels. In the larger piece sebaceous and sweat glands were present; occasionally, though rarely, a hair was found, always presenting a healthy appearance. The growth was due to excessive hypertrophy of the connective tissue, and partly to abundant cell-growth occupying interspaces between the bands of fibrous tissue.

Virchow has published an engraving of a remarkable case of this disease, which represents numerous small growths over the whole body, and one very large one, which hung over the hip, and weighed some thirty-seven pounds. A somewhat similar case is to be found in the catalogue of Guy's Hospital museum, illustrated by wax models of the tumours of the body; and in this case there was also a tumour at the back of the head, and one hanging down from the nates some sixteen pounds in weight. This man died over eighty years of age. Another remarkable case is recorded in the Transactions of the Pathological Society, vol. xvi., in which the growth of the tumour occurred from the neck, and hung down below the umbilicus, very similar to the case of Mr. N. Hughes. There was also a large secondary tumour attached to the back part of the head and neck.

Dr. J. C. Warren has also described a peculiar case of skin tumour in his work on tumours. The tumour resembled a coil of intestine, and occupied the right side of the neck. It was removed, but returned in the course of some eighteen months.

It is worthy of remark that there is, to a certain extent, a correspondence in the position of the tumours in the cases alluded to. The trunk is most frequently the seat of the larger number of tumours, while the upper and lower limbs are often free. Little is to be said with respect to treatment, and, though there is a prospect of a recurrence after operation, it is proposed to remove the larger portion of the anterior flap, as the patient is anxious to be relieved from the inconvenience she suffers from it.

Dr. LANGDON DOWN said he was sorry Dr. Murray had not paid more attention to the physical peculiarities of the eldest boy's head. He was of a strumous diathesis, which was often associated with congenital imbecility. He had a history of convulsions during infancy, but showed no traces of imbecility. Dr. Murray thought the disease was due to the consanguinity of the parents and bad hygienic conditions; he would say rather to struma, of which there was a history on both the father's and mother's side. Some years ago he had attempted to show that the marriage of cousins was the great cause of neuroses, but as he went on with his paper he found from the statistics it was not so, for if the first cousins could be selected, we should have a healthy race.

The PRESIDENT, referring to Mr. Pollock's case, thought the proposed operation would be of advantage to the woman, but as the growth depended essentially on a blood disease, it was questionable if the benefit would be permanent.

Dr. A. P. STEWART said he had seen a somewhat similar case some years ago in a dressmaker who had undergone

Wilson Fox, on tuberculosis

great privation and much mental anxiety. There were a considerable number of tumours like those just described. A large pendulous mass had been removed from the hip. The tumour never returned, and the cicatrix was healthy. There was one large pendulous tumour on the right temple and a number of small ones scattered over the trunk and the back. After a time symptoms of phthisis appeared and rapidly increased, and she died. The lungs presented the ordinary appearances of phthisis. On examining the thoracic viscera, he found all the lacteals of the mesentery closed by a concrete yellow substance; the glands were enlarged, and full of the same material.

MR. SPENCER WATSON said he had met with two somewhat similar cases to Mr. Pollock's, one in an old gentleman aged sixty-eight, the other in a young girl.

Dr. DUCKWORTH thought Dr. Murray's case was unique. He went a short time ago into Essex to trace out a similar case. There were many tumours on the legs, and one the size of an orange had been removed by Mr. Hutchinson. The cicatrix was healthy. The woman lived in a most wretched manner—bad food and bad hygienic conditions.

Dr. JOHN MURRAY said, in reply, that he had recognised the peculiarities in the insertion of the ears and the shape of the head of the eldest boy, but did not admit that the interpretation of them was correct. He thought that the evidence of a scrofulous origin rested on very slight grounds. The family had really been a very healthy one, the only exception being the death of the patient's grandmother, when young, of consumption. The children, brought up in poverty, were almost singularly free from the ordinary signs of scrofula. On the other hand, the arguments in favour of the consanguineous origin of the disease, as expressed in the mental and moral phenomena in one child and in deafness in another, were strong. Moreover, the symmetrical character of the affection was in favour of nervous origin.

MR. POLLOCK agreed with the President as to the benefit the patient would derive from the operation. He questioned if the advantage would be permanent.

PATHOLOGICAL SOCIETY OF LONDON.

TUESDAY, MARCH 18TH, 1873.

SIR W. JENNER, BART., K.C.B., PRESIDENT, IN THE CHAIR.

THE PRESIDENT said that, according to the announcement at the last meeting, Dr. Wilson Fox would now open a discussion on the Anatomical Relations of Pulmonary Phthisis to Tubercle.

DR. WILSON FOX.—In introducing this subject at the request of the Council, I will spend as little time as possible in preliminary observations. One would not willingly, however, enter such an arena without certain cause being shown, the first being in this case the desire of the Council that this discussion should take place. Personally I can offer no apology, except that I have done a certain amount of work at the subject. One other point on which I would ask for some indulgence is the comparatively superficial manner in which many important points are treated. As you said, Sir, the subject is illimitable, and my hope will be that the Society will hear from other gentlemen elucidation of points which time prevents me from considering. The last point on which I must appeal for indulgence is that I must pass by, without remark, the opinions of others. The debates on phthisis have extended through nearly two centuries, and to offer a balance of opinion between the author and another, the remainder of the session, as you remark, would hardly suffice. There are few opinions of the present day which are not held by various members of the Society, and those views can be far better represented by those gentlemen in the expression of their own views than by me. Therefore, Sir, as regards historical or controversial opinions I have no statements to offer. I have simply to bring forward some results of personal observation, which may, I hope, at least serve as the basis for discussion. On the history of the question, then, I shall merely have to say that which will, in my opinion, help to elucidate its present position. I have also for one moment to apologise for the special point we have to discuss to-night. For that I am responsible. When the secretary asked me to open this discussion, I felt at once that the main interest surrounded the question of phthisis, and that is an interest which will never cease in relation either to humanity or to the many controversial questions of the present day. The etiology, therapeutics, and prognosis of phthisis all hang upon this point—how far tubercle is concerned in the constant morbid anatomy of phthisis. I allude to this more particularly, because a most eminent German writer, Prof. Niemeyer, has stated that every question connected with tubercle bearing on the etiology and prognosis of phthisis must be given up on account of the very small part which tubercle bears to phthisis proper. A large part of phthisis has nothing to do with tubercle; tubercle is superadded to phthisis, but it constitutes an almost fatal complication of the disease. I am not quoting Prof. Niemeyer's words, but those are the practical conclusions from some of his most important statements. On the question whether there are clinical varieties of phthisis I am not going now to enter. For my own part, I believe most may be traced to the variations of development of, and subsequent changes in, certain bodies in the lung, to which, up to the time of Virchow, the name of tubercle was almost universally given.

Regarding, then, the history of the question, I would only state that, commencing with the definitions of Hippocrates, that phthisis was ulceration of the lung, through the greater part of the seventeenth century there was a gradual recognition that bodies which the authors of those days called tubercles, and designated alternately or simultaneously by the names of "Scirrhosa" and "Caseosa," or "indurated" and "caseous," formed the anatomical basis and essential constituent feature of phthisis. Chronologically, it is impossible to trace this question accurately, for the opinions of different writers merged into one another during the greater part of this century.

During the greater part of the eighteenth century the discussion ranged further on a recognition of the similarity of those changes to what takes place in scrofulous glands, and the main debate was whether these tubercles, as they still continued to be called, were lymphatic glands allied to scrofula or were anything else. In the present day this aspect of the discussion has very little altered from that of two centuries ago. That discussion was continued to the time of Portal, who supported the same opinion; but in the early part of the present century began another phase in the history of the discussion, when Bayle regarded what he still termed tubercle as a product *sui generis*, and the result of a special dyscrasia. Even before him arose a controversy regarding the possible inflammatory nature of the growth, and that of the present century has mainly been between the theories of a special dyscrasis, or inflammatory changes. The theory of scrofula was maintained in part during this period by eminent foreign and English writers, but whether this was maintained or not, the question of tubercle being an inflammatory product, or the result of a special dyscrasis, a deposit from the blood in a morbid state, was the main controversy in the whole history of medicine. Into this controversy, each side of which has been maintained with great ability, I cannot now go; but a fact to which for a moment I would call the attention of the Society is that whatever theory was held concerning the origin of these growths, deposits, masses, whether grey, yellow, hard, or soft, they were always called "tubercles."

The history of what is now called tubercular infiltration, speaking not very accurately, but within chronological limits, was practically begun by Bayle. He described grey and yellow uniform infiltrations of a large part of lung, with somewhat amorphous-looking material, and pronounced it to be tubercle, and it was continued to be thus described in the same terms to the time of Laennec, who may be said to have solidified the previous floating ideas of tubercle, which had before received the stamp of homogeneity by Bayle, with the one exception of the grey granulation.

What Laennec and Bayle considered to be a grey infiltration of tubercle, analogous to the grey granulation, was since been usually considered to be pneumonic, but the yellow still continued to be called tubercular, and the yellow infiltration or yellow masses in the lung were finally taken to be the type of tubercle, until what Bayle had doubted to be tubercle, the grey granulation, was asserted to be tubercle by Laennec and Louis, because it became yellow. The occurrence of this transformation was considered to be the test of tubercle. Whatever became yellow, caseous, dry, and friable, was considered to be the most typical form

of tubercle, and, with the exception of Dr. Addison—into whose views, with the most profound respect, I shall not here enter, partly because there are many here better qualified than I to do so, and partly because of the limits I have already laid down not to enter into particular views—with that exception, yellow tubercle was regarded as the most characteristic form until the time of Virchow.

With Virchow began the main variation which the discussion has undergone in our day. He announced that yellow caseous matter might arise from the degeneration and inspissation of many other products—pus, cancer, and other matters; and he therefore pronounced that caseous matter was not the type of tubercle. Seeking for another definition of tubercle, he considered the typical form to be the grey granulation of Bayle—the granulation which Bayle himself for a moment doubted to be tubercle at all. This definition was to a certain extent a new one. It excluded a large amount of what had previously been called tubercle. He also expressed his belief that a large part of what was termed infiltrated tubercle was, in fact, inflammatory; that these infiltrations and caseous matters, when not assuming the form of grey granulation, were a "lobular caseous pneumonia," or "scrofulous pneumonia," and that they arose from the inspissation of inflammatory products.

There is no one here, Sir, who has a more profound respect for Prof. Virchow and his labours than I have. A large amount of the work of my professional life has been due to the desire to follow out some of his teachings in this respect, and if I have on some points arrived at different conclusions, they are chiefly these—viz., that in the large proportion of cases where tubercle arises in the course of phthisis, that origin is not due solely to the inspissation of inflammatory exudations or products, but to the destruction of vessels by a new growth; and that again the typical isolated grey granulation is not the sole form in which tubercle appears in the lung.

In Germany those views, coming with the authority of such a teacher as Professor Virchow, have obtained large acceptance, and in this country they have been accepted to a large extent. They find their latest culmination in the text-book of Professor Niemeyer, which I specially allude to because it is generally known in this country. The logical deduction from Niemeyer's teaching appears to me to be that phthisis, as a disease, has nothing whatever to do with tubercle. The destructive changes in the lung are the result of the inspissation of inflammatory products, caseous changes in pneumonia, and that tubercle when found in the lung is merely an accidental product; and is to be regarded according to a second theory, which we also owe to Germany, to Professor Buhl, and which has found many supporters, as the result of infection. The question is thus absolutely changed, inverted from the old doctrine, and tubercle is an accidental complication of phthisis, in no respect its primary and chief anatomical distinction. I think I am not guilty of any exaggeration when I say that that is the logical deduction from Professor Niemeyer's views.

Fifteen years ago I came back from Germany strongly impressed with the opinion that phthisis might be divided into many absolutely diverse diseases, and with a certain ambition, such as a young man may entertain, to aid in working out their definitions from a clinical point of view. I had many terms at the end of my tongue—broncho-pneumonia, caseous pneumonia, and scrofulous pneumonia—which sometimes young men use more freely than their teachers. Coming to the work of teaching myself, I long felt great hesitation in speaking of tubercle in a practical point of view, and found great difficulty when face to face with the changes in the lung, in saying what was not, and still more what was, tubercle. I believe that many others in the same manner can speak of caseous changes and lobular pneumonias, but find it difficult, when brought face to face with the lung, to say what the particular change is. Before setting to work at the clinical aspect of these cases, I found it, therefore, necessary to deal with the anatomical question which they involved, and I have to thank my colleagues for giving me large opportunities of examining lungs in the post-mortem room. I found it necessary, after noting the appearances in the lung, to have a good drawing made of them (and for these I have to thank Mr. Tuson), and to make microscopic observations on what the drawings showed. The results of these I have tabulated. I had then to go over the ground again, for I found the same appearances crossing one another in every possible manner, and a large number of them in the lung not corresponding to what I had imagined to be the type of the typical grey granulation. I decided therefore to take the lung in the general disease universally recognised as tubercle—acute tuberculosis,—and see what changes accompany the disease in which grey granulations occur in every part of the body, which grey granulations really produce in the lung. I have to thank my friend Dr. Gee, and my lamented colleague and friend, the late Dr. Hillier, through whose kindness I was able to obtain from the Children's Hospital many lungs from cases of generalised tuberculosis.

It was necessary to take either an arbitrary tubercle, or arbitrary idea of tubercle, as derived from section in serous membranes, or take all the appearances of the disease, and see wherein they differed from other diseases, or wherein they corresponded, and whether any general definition could be found; and the results of those observations I wished to apply to cases of phthisis. As the result of my inquiry I found in the lungs of patients dying of phthisis changes almost identical with those in the lungs of children dying of acute tuberculosis, with such variations of anatomical change as can, I think, be pretty clearly traceable to lapse of time. Tubercle

The following are the chief appearances which I found in the lungs of children dying of acute tuberculosis:—The semi-transparent granulation of Bayle; white granulations, neither absolutely semi-transparent nor absolutely opaque, but still presenting a certain degree of translucency; granulations like the semi-transparent granulation of Bayle—firm granulations, but more or less caseous in the centre; yellow soft granulations, somewhat prominent, easily crushed, but not easily removed from the pulmonary tissue, varying in size from that of a poppy-seed to a mustard-seed, or still more rarely to that of a split pea; distinct caseous granulations, dry, opaque, friable, sometimes with, sometimes without a semi-transparent zone of induration surrounding them; groups of granulations, semi-transparent or opaque, two, three, or more in number, reaching the size of a split-pea or bean; indurated pigmented granulations, surrounded by a zone of pigment; and lastly, tracts of indefinite extent, one, two, or more inches in diameter, irregular in outline, prominent above the cut surface, granular on section, which pass sometimes insensibly into the so-called grey infiltration; cavities, from infinitesimal specks to the size of hazel-nuts or more; sometimes granulous softening into cavities, either the softer, the white or the yellow. The semi-transparent granulation, *per se*, as far as I have seen, is not found softening into a cavity without some intermediate change. Tracts of grey semi-transparent gelatinous appearance, what we know as either the grey pneumonia, or gelatinous pneumonia, or gelatinous grey infiltration, or gelatinous infiltration of Laennec, and red pneumonia, bronchitis, injection, punctiform extravasation, and dilatation of bronchi, and some other appearances which have in this moment very little to do with the subject I have to bring forward. The point on which I wish specially to insist is that these things are very seldom found alone, that the grey semi-transparent granulation of Bayle is sometimes found isolated, scattered thickly through a whole lung, but that is the rarer appearance of the two. As far as my experience has gone, you most frequently find commingled with one of these. Many granulations are larger than that little pin's-head semi-transparent point—are more opaque and more friable. In the lungs of eleven cases in which I have taken minute and accurate notes, they either co-exist with the caseous or with the white and soft and caseous, or the soft and caseous alone.

The point on which I wish to insist is, that the grey granulation of Bayle, the typical tubercle of Virchow, does not exist alone in the majority of cases of acute tuberculosis of the lung in children, but that it is almost always found with other forms of change, which in a large proportion of cases so predominate, that the grey granulation is the rarer appearance, and is even difficult to find. In this I think I shall be borne out by anyone who has minutely examined with the microscope. The grey granulation of Bayle in such lung: is not only difficult to find with the naked eye, but even to find typically under the microscope. What appear to be grey granulations are peri-arterial or peri-bronchial thickenings, and exist in the septa, but in the lung-tissue the typical grey granulation is comparatively a rare product.

Cavaliers
Intergranular
Grids
reticulum

By the word grey granulation I allude to (as represented in the diagram) a body comprised of minute cells, about the size of a white blood-corpuscle, or smaller, with nuclei smaller than a red blood-corpuscle, in older forms separated by a delicate reticulate network. In many of these diagrammatic representations has been exaggerated by the artist. The reticulum is found by high powers of the microscope in most forms of tubercle except in the most recent granulations, in which these nuclei and small cells crowd upon one another, and no reticulum can be seen. I must apologise for not now going more fully into the structure of typical tubercle; it would involve too long a discussion beyond the points I have dwelt on, and would occupy more than the time that remains at my disposal. A body isolated and composed of small cells massed together, generally separated more or less by reticulum, or only not separated by reticulum because so densely massed, may be taken for the moment as the typical structure of tuberculosis, with certain exceptions, on which I shall have presently to dwell. There are often much larger cells, and sometimes, though these are comparatively rare in the lung, very large cells, $\frac{1}{125}$ to $\frac{1}{250}$ of an inch in diameter, with many nuclei—the Riesenzellen of Virchow. I have placed under the microscope one or two of them. I have never been able to isolate those cells. They have been described by Koelliker and Luppol. I have never seen them isolated. They are as large as the myeloplasts of a myeloplastic tumour. Virchow described them in the omentum. I have frequently torn up preparations of tubercle from the omentum to seek for them, but have never succeeded in isolating them. Whether groups of nuclei, surrounded by protoplasm, or real cells, I cannot say. Between these and the typical small cells are various other forms of large cells which I have not represented in the diagram. Those characteristic of tubercle in the lung, as I take it, are the small cells, most of them the size of the smaller cells of lymphatic glands, commonly smaller than the white blood-corpuscle with a nucleus, sometimes nearly filling up the cell; or, what is more common, dense masses of apparent nuclei, whether in a reticulum or not, which may be best described in the terms of Dr. Lionel Beale, as a formed material produced by the protoplasm of these cells indurating in its external margin till finally what we used to call the cell-wall blends with the reticulum, so that when broken up only nuclei can be isolated.

The grey granulation, I said, was hardly ever found in the alveolar structure of the lung; what appear to be grey granulations there, being growths of tubercle in the peribronchial sheath, or peribronchial and inter-alveolar tissue. In the alveolar tissue this typical growth becomes mixed with other products. I do not say you never find among the alveoli these rounded bodies; you do find them, tending to assume a rounded form; only, most commonly, in their centre and at various points, you meet with, more or less, the appearance of epithelial cells, large cells which correspond to those of the interior of the alveoli; and I believe that they are the result of epithelial proliferation. I do not wish to dogmatise for the moment upon the word epithelial. Whether there be epithelium in the lungs or not, I believe the cells I allude to are the result of the proliferation of cells in the interior of the air-cells, which assume a larger form, give rise to a rapid development of nuclei, and die. But together with them, but in the walls of the vesicles, you find, in large tracts, the same reticular growth, and multiplication of nuclei and small cells, identical in appearance with those of the most typical grey granulation. The proportion between the epithelial cells and the denser nucleated growths varies in nearly every specimen of granulation that you can find in the lung in acute tuberculosis. That, and not the typical grey granulation of the peritoneum, is the most common appearance in the lungs in acute tuberculosis in children. A large number of the granulations that appear yellow or owe their colour to two sets of causes. In many of them the change is an acute caseous change. Nothing is to be seen but round yellow caseous spots, surrounded by more or less fibrillated material of inter-alveolar septa or inter-lobular septa surrounding the caseated interior of the alveoli.

The next most common change is to find the air-vesicles filled with cells, but the vesicles themselves separated more or less widely by a growth passing between them. Lastly, you find intermediate stages between that and another condition, in which the interior of the air-vesicles is occupied by a caseous material, and the wall at the margin of these spots of infiltration is occupied by a similar infiltrating growth.

Lastly, in those large tracts of caseous infiltration you meet with one of two things. I am merely stating my own opinion. You meet either with large tracts entirely occupied by the two appearances last described, or with only another set of changes—universal thickening of the walls of the alveoli until the interior of the alveoli is almost obliterated. You may find that change where the caseous material blends into the tracts of grey infiltration. That thickening is produced by a small-celled or nucleated growth similar in character to that occurring in the softer form. You find, besides, little infiltrations of small-celled growths into the sheaths of the arteries and bronchi, and also similar ones around the bronchioles—those terminating in the alveolar tissue, and these may be termed peri-bronchial tubercles.

Everything I have now described occurs in acute tuberculosis, and I would also state that the infiltrations, when traced in an injected specimen, are found to occur in an almost similar manner in those occupying large tracts to what is observed in the granulations, and they gradually occlude the blood-vessels. In the network of blood-vessels larger masses of nuclei may sometimes be found, but by their growth the bloodvessels become obliterated; and I am inclined to think that—to correct my former statement—the reticulum in the lung is partly formed of débris of the pulmonary capillaries, from the nuclei of which the growths may also arise. It is a hypothetical question, having little bearing on the subject I am discussing. The point on which I would insist is, that in their progress of fine-celled growth, vessels almost invariably disappear.

Now I venture to describe, as presenting similar characters, different appearances in the lung of acute tuberculosis, excluding for the moment pneumonic infiltrations. I have described a growth in the wall of alveoli, the interior of which is occupied by all products differing but little from those of ordinary pneumonia, producing extensive thickening of the alveolar wall. According to my observation, Sir, these and the grey granulations have all the same structure. I venture, therefore, to affirm that the grey granulation is not the all-essential typical distinguishing feature of tubercular formation in the lung, for in the most typical acute tuberculosis you have the same kind of growth occurring in larger areas. If one of these changes I have described is pneumonia or scrofulous pneumonia, or caseous infiltration, you must have in acute tuberculosis two diseases which start from a growth the same to all microscopic appearance, one of which is hitherto unnamed, or you have one disease which may occur in two forms, the grey granulation and the wider infiltration. I dwell on this because it is one of the questions constituting a great part of the difficulty about phthisis. If tubercle is only grey granulation, tubercle in phthisis is not only comparatively rare, but its demonstrable share in the destruction of the lung is small. If tubercle be an infiltrating growth, destroying capillaries, passing into caseous change, the part tubercle takes in phthisis is large.

I wish to say one word about caseous change. You will find large tracts completely caseous, and presenting appearances which I have called tubercular pneumonia, a term, in my own opinion, meaning pneumonia complicated with a growth analogous to that existing in these granulations in acute tuberculosis. Wherever you have this growth thickening the wall of the air-vesicle in injected specimens the capillary circulation is found to cease, and even in non-injected specimens it is easy to see that there are no capillaries there, and that this growth is occupying the place of the capillaries. In this respect, as the figures before you show, it is scarcely distinguished from specimens of acute pneumonia. In acute pneumonia the capillaries are still there, and the growth in the wall of the air-vesicle is not there, as far as I have been able to observe, in a considerable number of specimens.

At this point I must become, for a moment, hypothetical. The death of the part is due, as I believe, to the destruction of capillaries, which may take place slowly in one case, or acutely in another. In one case you have a growth capable of a further development, concerning which I shall have to say a few words in a moment—a fibre change. In the other case you have simply a complete destruction de-

pending on the acuteness of the process. As a question of pathology, new formations of rapid growth under abnormal circumstances of irritation have a short life in proportion to the intensity of the irritation present. I believe that that explanation of the acuteness of the inflammatory process with which this inflammatory growth concurs—I will not say originates, because that would be begging the question,—largely determines the rapidity of the caseous change, of a growth which would proceed to an infiltration, producing thickening of the wall, if it had sufficient vitality, but which dies on the first destruction of the capillary circulation if the irritation is acute. To recapitulate: the distinctive and vital characteristics of the growth are in two directions—in the possibility of development, and, in the other, of death. Development may take place because we know that minute capillary circulation is not necessary to permanent tissue, as is seen in cartilage and in large tracts of fibro-cartilages and fibrous tissue. You may have a considerable development of the lower form of fibrous tissue without the intervention of any discoverable capillarity, except from a distance. Accordingly, the fibre change may take place in contradistinction to the destructive change; and in young children where the process has become more or less chronic in one lung, whilst it is still acute in the other, you see bands shooting in, almost like what is seen in cases of periosteal ossification. Around all tubercles as they advance in age a more or less fibrous capsule forms, and these bands tend to traverse them in all directions; and even where in early childhood the process of tuberculisation has been more or less acute you see those bands passing into the tubercle. I need not dwell on the proof that these grey granulations pass into fibre change: there is nothing new in this. That tubercle can indurate, become obsolescent, become hard without becoming caseous, has been known from almost the earliest ages of pathology. It is only difficult to explain the relation of this to phthisis.

The point which I wish to lay before the Society is, that all these changes—caseous infiltration, large pneumonic product, mingling of pneumonic products with reticular growth, induration of the reticular growth, infiltration of that reticular growth throughout large portions of the lung, acquires a more or less caseous appearance—all concur in the most typical forms of acute tuberculosis. In some cases that infiltration, when it occurs in previously emphysematous tissue, gives to the tissue a peculiar worm-eaten appearance—an almost exact repetition of one specimen before me.

Now, Sir, I will dwell briefly on two or three propositions which I venture to lay down as corollaries to these points. Under what condition do we meet with tubercle of the lung in phthisis, especially in relation to the question of inflammation? Speaking in the abstract, as I stated in the proposition laid on the table, tubercle may occur without inflammation in the elements of the tissue in which it is developed. We must admit that in the liver and kidney in certain blood states you may have a development of tubercular growth as part of the signs of the general acute disease or constitutional affection, in which we find no inflammation of the part in which it occurs. Secondly, I also stated that tubercle is found in addition to inflammation of the tissue in which it occurs. In many cases of acute tuberculosis the evidence is very strong that you may have inflammation of serous membranes (pericardium, pleura, and meninges), in which, at any rate, no naked-eye tubercle can be discovered. Without quoting statistics, there is a fair percentage of instances recorded by Empis and Colin and others. In these cases I think it is fair to believe that the inflammation precedes the tuberculosis. At a later period, wherever you find inflammation you commonly find granulations added. In the intestine you may find intense congestion and hyperæmia proceeding to ecchymosis, but no tubercle, that ecchymosis and hyperæmia sometimes surrounding Peyer's patches and the solitary follicles. I have been induced to regard this as a preceding inflammatory process, antecedent to the formation of secondary tubercle. Of all things you find most commonly and constantly the two, inflammation and tubercle, mixed together; at any rate, in the mucous surfaces and serous membranes that combination is almost constant. The question is, what part each plays in the lung, and can we distinguish the one from the other? Again, Sir, in the lung, as far as we know at present, the implication of the alveolar wall is the most constant and typical character. I would for a moment beg to exclude a certain number of diseases which I have already recapitulated, ulcerative bronchiectasis, indurated pneumonia, ulceration after pneumonia, disease of the lung from inhalation of dust, cancer, and syphilis. With these exceptions, I have only in my life found three cases of phthisis which did not present the grey granulation or the soft granulation which I have described, or such caseous change or infiltration, in greater or less combination. I may appeal, Sir, to my unbiased opinion, because it was my anxious wish to find something in my day and generation to distinguish a great many varieties of phthisis, and it was only after long search and inquiry I came to this conclusion.

Sir, I have not yet given anything like a definition of tubercle, nor am I going to give a dialectical definition, but as far as we know of tubercle at present, especially if I may allude to the researches of my friend, Dr. Sanderson, it may be regarded as a lymphatic overgrowth produced by irritation. We do not know at present its relation to the walls of the air-vesicles. Buhl has lately stated that the lining membrane has its analogue in the endothelium of the lymphatic sheath. On that point it would be difficult to give an absolute opinion; certainly lymphatics have been traced in sufficient number in the walls of the air-vesicles to allow us to believe that lymphatic tissue must play an important part in the changes of the lung. Perhaps this wall is one of the richest lymphatic plexuses in the body, but that is not positively proved. At any rate we do find tubercle most commonly in the lung, and when you give rise to irritation, in cases where the lymphatic irritability is excessive, under those circumstances you may get tubercle. You may therefore get it from a blood-state in which that relation to lymphatic change is abnormal, or you may get it locally by exciting inflammation. As to the last question, the question stated by Laennec and Louis, whether tubercle gives rise to inflammation—it is as difficult to answer as the first two. One can give some answer from recent observation. I have said that it arises from lymphatic tissue; the other theory, by exudation and deposit, I take as far granted as for the moment, been abandoned. So far as my own observation goes, I believe that caseous change is the result of this growth. That it originates from white corpuscles I doubt; certainly on the other hand no proof has been given. All this has some bearing on the question of phthisis proper, apart from tuberculosis.

To sum up, I would say that the causes of inflammation may be simultaneous with the causes of tubercle. Inflammation and tubercle may be set up and arise, even in the lung; or inflammation once set up may give rise in its extension to a secondary growth in the alveolar wall, and that secondary growth is the cause of ulceration.

These characters of tubercle, as I have described them, are distinctive, but they cannot be defined now, and even for the purposes of distinction they require to be taken collectively. It would be difficult if not impracticable, even with the microscope, to distinguish in all cases tubercle from certain growths which resemble it, as those of leukhæmia, typhoid, glanders, and even some forms of inflammation. Time will not permit me to discuss the resemblances to the former. Cases where any close similarity exists between simple inflammation and tubercle are, however, rare, and even then it is rather a superficial resemblance than an identity of structure. The dense massing of tracts, with cells, and nuclei with an interposed reticulum seen in tubercle, is seldom found in ordinary inflammation of other parts. The boundary line, as has been remarked, between tubercle and inflammation cannot be accurately defined. Tubercle is the result of irritation of a particular set of tissues under certain circumstances, and we must therefore expect to find, and I believe do find, formations which resemble it, arising under other similar but yet really diverse conditions of origin.

I have limited myself very much to the question of acute tuberculosis. Coming to phthisis, in my opinion, subject to the criticisms of the Society, it may be divided mainly into two forms which merge insensibly into one another—the acute and the chronic. The acute forms are multiple, but are mainly characterised by yellow granulations, grey and opaque granulations, and grey infiltration, in which caseous spots exist. Regarding what is termed scrofulous

pneumonia, I would put the matter briefly thus. There are large caseous infiltrations in what is termed caseous pneumonia. When they exist in large tracts, immediate death of the contents of the air-vesicle takes place, and generally death of the wall at the same time. The caseous change most commonly occurs in isolated spots in the grey infiltration, dotted through it, the size of pins'-heads or larger, distinct and circumscribed from the surrounding grey infiltration; and on this, Sir, I bestowed an amount of time which I will not inflict on you. I will only summarise my conclusions briefly, by saying that they are almost all identical with two of the drawings before you. In all these caseous spots is a new growth in the wall of the air-vesicle of a nuclear reticular structure, which I have never been able, with any microscope I could use, to distinguish from the infiltration and nuclear growth that occurs in the softer granulations of acute tuberculosis. That the caseous change which occurs in the middle of the grey infiltration arises from any mere inspiration of pus or accumulation in the interior of the air-vesicle, I would distinctly deny. It is absolutely different from any accumulation of puriform matter that takes place in lobular pneumonia or in simple pneumonia. This drawing by Sir Robert Carswell shows clearly the distinction between lobular pneumonia and these caseous spots which occur in acute tuberculosis. The part dies because the circulation in its wall is cut off by the new growth, and the death is acute in proportion to the acuteness of the inflammatory process surrounding it or accompanying it. If the grey infiltration sets up around old tubercles, in a large number of cases they die at once, and yield those caseous matters; but in other cases the growth takes place simultaneously with the infiltration, and caseous spots form in proportion as the infiltration does so.

It is only where little growths take place in the middle of an infiltration that these spots occur. To say that they are mere inspissation is a fallacy, and leads to the second, and, in my opinion, a fallacious theory, that caseous change takes place from an inspissation of catarrhal pneumonia, and that is the origin of tuberculosis. In the form which it has become the habit of the present day to call *confluent* pneumonia, the process is identical, step by step, with the majority of the processes which occur in acute tuberculosis.

Chronic phthisis, as distinguished from the acute, presents the characteristic of induration of the lung, and its chronicity depends upon the slow induration of these growths, rather than upon their acute destruction. The change by which tubercle becomes obsolescent may affect the infiltrated tissue—if I have already established that there is such a thing as tubercle extending over a non-circumscribed area,—as well as it may affect an isolated granulation; and it also affects groups of granulations. In the groups of granulation you get tracts of tissue which are more like masses of fibro-cartilage than anything which can be called tubercle, and you can trace the progress by stages of gradual induration through any amount of fibre-growth into the most intense tracts of fibroid induration. These processes of induration have been called lately by some German pathologists, peri-bronchitis, peri-arteritis, and indurating peri-alveolitis. I would venture to say they are not peri-bronchitis in the sense that they affect large bronchi; but they affect bronchioles, and commence with a growth identical in character with that which I have described. Whether peri-bronchitis or not, it does not alter the main character—it is a peri-bronchitis; or, as Virchow has termed it, a peri-bronchitic tuberculosis. In that sense I would venture to say that in every indurated form of phthisis—and those I have classed in the table as chronic phthisis come under that category—the indurating tubercles are mixed up with forms of alveolar type. One word about the origin of all this. I have already spoken of the origin of phthisis in pneumonia, and I shall say no more on that point. We have lately had some dogmatic assertions as to the origin of phthisis from pneumonia and scrofulous pneumonia—assertions so dogmatic that one is surprised that more of it is not seen. It is rather rare to see commencing disease of the lung that terminates in phthisis. We only see certain forms of induration at the apex. I have seen two such cases, and in both there was grey pneumonia with soft granulations. The argument from later stages—and that is the only one possible for us, as I would specially insist, as giving a harmony lately broken to the idea of phthisis—is that of a similar growth antecedent to the destructive change, whether in granulations or in infiltration. Finally, I must apologise to the Society for the amount of its time which I have taken.

The PRESIDENT suggested the adjournment of the discussion to the next meeting, and said the Council would be obliged if gentlemen proposing to speak on the subject at the next meeting would give their names to the Secretary, and at the same time give him some idea of the amount of time which they desire the Society should give up to them.

Reviews and Notices of Books.

Researches on the Action and Sounds of the Heart. By GEORGE PATON, M.D. pp. 64. London: J. & A. Churchill. 1873.

Few matters depending upon direct observation, or, perhaps we ought to say, upon the interpretation of auditory and visual impressions, have led to such differences of opinion as the actions and sounds of the heart. Physiologists have even been accused of mistaking the systole for the diastole; whilst the varieties of opinion in regard to the causes of the sound have been as extreme as, we might say, existed between those who, listening to an orchestra, should maintain the entire volume of sound to be produced by the vibration of one string of the basses and those who should attribute it to the simultaneous action of every note of every instrument. Dr. Paton is by no means exclusive in his views; but he has done what many others neglect to do—listened and observed for himself, and is therefore fully entitled to be heard.

Dr. Paton describes more clearly and, we think, more correctly than has hitherto been done, the variation in the phenomena presented, according to whether the heart is acting slowly, with moderate vigour, or very energetically. In the former case the ventricles become fairly filled during the first stage of the diastole, and only completely distended by the comparatively feeble and late contraction of the auricles. In the second case the auricles act sooner and more vigorously, but the ventricles have still received a moderate supply of blood during the first stage of the diastole. In the last case there is no first and second stage of diastole, but the auricles are promptly filled and promptly discharge their contents into the ventricles; the contraction of the auricles is, in fact, synchronous with the diastole of the ventricles, and *vice versâ*. Dr. Paton has not, however, we think, been happy in his phraseology, where he says (p. 19) that the auricles exert a force which converts the movement of relaxation into one of *active dilatation*, "because the power with which they contract may not only excite the dilatation, but impart a stimulus to the relaxing parietes of the ventricles which renders the diastole the first and most prominent movement, and the systole its first sequence." We think Dr. Paton would have done better to have employed the term forcible dilatation rather than active dilatation, which, indeed, he does not really mean. The term active dilatation expresses the phenomenon presented by an ordinary elastic ball after it has been compressed and a considerable suction power is exerted, but here the walls of the ventricular sac are only forcibly separated by the propulsion of the contents of the auricles.

Again, we think the following statement very doubtful, and, even if it be correct, we think better language might have been selected. Dr. Paton says (p. 31) that in the heart of a turtle exposed, and beating thirty-two times per minute, it was observed that as the auricle projected the blood into the ventricle it swelled out, and dilated till it attained the point of distension; it then immediately *contracted*, its parietes *expanding* with considerable force, resisting the pressure of the fingers applied to them. Ac-

tinued in it with varying force through the year 1856. On the other hand, the typical periods of rest were from April, 1851, when it ended in Jamaica, till September, 1852, when it broke out in the Bahamas; from February, 1853, when it ceased there, till it appeared in Nevis and St. Thomas and Jamaica in December, 1853; from March, 1854, when it ceased there, and burst out in Barbadoes in May, the Grenadines and Trinidad being subsequently infected until October; shortly after that reappearing in the north in St. Kitt's, which it occupied through the next dry season, and ceased in January, 1855; and, lastly, with the intermission of the entire wet period of the year, it visited Puerto Rico in December in the early part of another dry season.

This remarkable series of sequences of epidemic intensity and intermediate rests observed in a long chain of islands are, to my mind, conclusive in proving the truth of the proposition, that cholera epidemics show a very remarkable relation to the seasonal phenomena in hot climates; the dry season being that of epidemic intensity, and the wet season that of dormancy or of comparative freedom from activity.

A strongly pronounced feature in this correlation of cholera epidemics and the dry season of the Antilles is its contrariety to that which exists between the natural epidemic disease of the region—yellow fever—and the seasons; the period of its development commencing with the early hot moist season in May, attaining its climax in July and August, and ceasing in October, when the long dry cool season sets in; whilst cholera commences towards the end of the hot moist season, reaches its climax, and exhausts its epidemic force during the dry season.

These observations have reference to the relations of cholera epidemics to meteorological phenomena. I shall now proceed to examine their relations to restrictive measures by means of quarantine and lazarets.

The history of cholera epidemics in insular positions offers, it is true, negative instances in which the disease has not prevailed after free intercourse with infected places, and others in which it has broken out in spite of quarantine restrictions, so that some difficulty arises in asserting beyond question that these are certain means by which islands are to be rendered secure from invasion. There are, however, so many examples of epidemics arising subsequent to the arrival of infected vessels, or of those from infected localities, as to place it beyond question that the disease was imported from without. Thus, the Mauritius was infected from India in 1819 and 1856, and Bourbon from it in 1819, and from Africa in 1859; Madagascar and the Comoros from Africa in 1859 and 1869; Sicily from Italy in 1837 and 1867; Malta from Sicily in 1837, from Tunis in 1850, from Egypt in 1848 and 1865; Gozo from Malta in 1837, 1850, and 1865; the Grand Canary from Eavannah in 1851; Fogo in the Cape Verds from Italy in 1855; Madeira from Lisbon in 1856; the Bahamas from New York in 1852; Nevis and St. Thomas from England in 1853; Jamaica from Chagres in 1850, and from St. Thomas in 1854 and 1855; the Grenadines from Barbadoes in 1854, and Guadaloupe with its minor dependencies from France in 1865, to which may be added St. Nicolo and St. Antonio from St. Vincent in the Cape Verd group in 1856. These instances suffice to show at least that the neglect of quarantine has been followed by the most deplorable results.

Other instances may be cited to show that the presence of vessels in port undergoing quarantine has been the antecedent of outbursts on shore, as at Palermo in 1837, Malta in 1837, 1848, 1865, and 1867; and Trinidad in 1854.

A series of sequences so numerous as these, must have, I consider, much weight in the decision of that most important question whether the importation of cholera is preventable by quarantine, inasmuch as they show that without it, and even during its performance within a limited radius, there is a great liability to its epidemic outbreak in insular positions which, from their geographical conditions, present the best of all circumstances for testing the efficiency of exclusive measures.

Looking around for instances in which islands have seemed to be protected by quarantine, which many regard as positive evidence to that effect, but which we will assume to have been only remarkable instances or coincidences of quarantine and of exemption—a *post hoc* rather than a *propter hoc*,—we find that Bourbon is said to have escaped in 1856 and in 1862, through keeping up quarantine against Mauritius from which it had been infected in 1775 and in 1820, before quarantine for cholera was thought of. Another French colony in that ocean—Mayotte, one of the Comoros—holding a peculiar relation to the labour-traffic between Africa and Bourbon, but keeping up a rigid quarantine in epidemic seasons, was unaffected by the African epidemics of 1859 and 1870, when other islands of the same group, under Arab government, were infected, and Bourbon itself underwent a severe invasion in 1859, through its labour-traffic with Africa.

Among the islands of the Mediterranean, Sicily, the greatest of them, was exempt in 1848, 1850, and 1865, concurrently with rigid quarantine restrictions, when Malta, holding by a very easy quarantine, was always infected. Sicily and Sardinia escaped till 1867, while Italy was scourged from 1865, through 1866 and 1867. On the other hand, in 1867, when Sicily underwent an epidemic of it, Malta, having recourse to strict quarantine with Sicily, escaped notwithstanding its previous proneness to the disease.

In the Atlantic, the Grand Canary suffered very intensely in 1851, but every other island of the group was exempted, all communication with it being cut off. This affords the very strongest of contrasts with what occurred in the Cape Verd group in 1855, when the disease appeared in St. Vincent—a packet coaling station,—and two others of the group, St. Nicolas and St. Antonio, were speedily infected by persons migrating from it to them.

(To be concluded.)

Medical Societies.

THE ANATOMICAL RELATION OF PULMONARY PHTHISIS TO TUBERCLE.

THE discussion at the Pathological Society of Dr. Wilson Fox's paper on this subject, adjourned from the 1st instant, was concluded on Tuesday evening last.

Dr. C. J. B. WILLIAMS.—I cannot but think that this debate on tubercle has been about words more than things. A great many things have been shown, and we have had abundant proofs of much diligent labour and careful observation; but the object of all this seems to be more to determine what these things shall be called than to find out what is their real nature. When Dr. Fox and others, after the example of Virchow, call tubercles "growths," they give no more satisfactory account of their nature and origin than the girl Topsy did when, in answer to a question about her nativity, she replied, "S'pose I grow'd." In fact Topsy was a growth in a truer sense than tubercles are; but as this did not account for her origin and nature, neither will the word "growth" explain the origin or nature of tubercles. And I maintain that the term is applicable to tubercles only to a very limited extent. It is one of their most remarkable characters that, except at their first development, they do not grow as other growths and tumours do. They harden by the increasing number and consistence of their corpuscles; and this induration, by depriving them of pabulum from the blood, leads to their ulterior decay, either by caseation or by dwindling. As growths they are insignificant and abortive, and their chief characteristic is early decay. . . . is the foundation of their consumptive character, tending to the destruction of the tissues and the waste of the body. That miliary tubercles are essentially modifications of the lymphatic glandular tissue, I think fairly proved by recent observations in confirmation of the opinions of Portal, Broussais, Abercromby, and others. The similarity of scrofulous disease in lymphatic glands and tuberculous disease in the lungs, and their succession in the same individuals and families, have been generally accepted as strong evidence in favour of their identity, and rendered most probable the views of Portal and Broussais, founded on anatomical as well as on clinical observations, that miliary tubercles have their seat in the lymphatic textures. And

since in recent times the microscope has been brought to bear on the subject, and Virchow first declared miliary tubercles to resemble lymphatic or adenoid tissue in structure, there has been a general concurrence of opinion in the matter, and few now doubt their resemblance, if not their identity. The experiments of Dr. Sanderson and Dr. Wilson Fox on artificial tuberculisation led to the same conclusion; and now we have Dr. Fox in his address emphatically declaring that "tubercle is a lymphatic overgrowth." For my own part I had, forty-five years ago, expressed my conviction that miliary granulations in the lungs owed their constant form and size to their connexion with some elementary part of the lung-texture, and I was quite prepared to conclude, on the new evidence given, that the lymphatic tissue is that element; but that they are mere overgrowths of that tissue I could not and do not admit; nor do I believe that the lymphatic system is at all necessary to the production of other tuberculous formations, not granular. A mere overgrowth of a tissue ought to be an increase of all its parts—of the stroma, of the trabeculæ, and of the lymph-paths, as well as of the corpuscles; and this is what we have in true lymphoma and in the adenoid enlargements of leucæmia. So says Dr. Bastian. So said I long ago. But this is not tubercle. In tubercle you have increase only of the *corpuscles*; and they are not merely multiplied; but they are altered; they are harder; so that as they crowd in their proliferation they form, not soft expanding swellings as in lymphoma, but little hard nodules; and their subsequent history of irritation and obstruction of surrounding parts, and of decay and caseation in themselves, is dependent on this essential character of induration, which is not comprehended in the term "overgrowth." I say, then, that tubercles, if a growth at all, are a bad growth, a cacoplasia as well as a hyperplasia, and the elements rendered are the lymph-corpuscles rather than the whole adenoid substance. Do you ask for my proofs? I refer you to all the best microscopic descriptions, from those of Gulliver, which were the first, to those of the present day, not excepting Virchow, but excluding his fanciful conjectures about connective tissue. Still more strongly I appeal to the evidence afforded in the numerous microscopic specimens brought before you, and which are rather too clear and conclusive, others more confused, and bearing some likeness to the thicket of "growths" in which the minds of their observers may have become bewildered. But more or less distinctly I see in all these microscopies an assemblage of crowded corpuscles of small dimensions, with more refractive granules shining out within and among them. These corpuscles present essential resemblance to those of adenoid tissue, and to the pale blood-corpuscles, "leucocytes," as they have been named improperly, for they are not essentially cells at all; therefore I call them "sarcophytes," flesh-germs. Crowds of such corpuscles, but without their colloid and amœboid properties, form the bulk of recent miliary tubercle, with little or no reticulum or stroma. When they get close and do not coalesce, they appear among them and around them; about these I shall have something to say presently. But it is the corpuscles, like those of the lymphatics, that mainly constitute miliary tubercles, wherefore Dr. Sanderson and Dr. Wilson Fox call tubercles adenoid growths. Dr. Cayley objects to this, because this same so-called adenoid tissue may be produced in any part of the body by simple irritation, as I myself did in the margin of a hard chancre, in the liver in the early stage of cirrhosis, in the lung textures by the presence of irritating dust, as in grinder's phthisis. I think that Dr. Moxon had a preparation showing the same adenoid appearance in a blood-clot. I quite agree with these gentlemen, and I thank them for the illustrations which they give of my views. The appearances are the same and the corpuscles seem identical, but their origin is different. The corpuscles of miliary tubercle are lymphatic, being developed by infection in the adenoid texture. The corpuscles of inflammatory irritation are the sarcophytes from the bloodvessels, the pale blood-particles migrating and forming the corpuscular exudation-matter of scrofulous and other low types of inflammation. And as, according to von Recklinghausen, "the lymph-corpuscles are universally admitted to be identical in all their characters with the colourless corpuscles of the blood," so we find the same resemblance in appearance and the same unity in nature and history in the multiplied corpuscles of diseased lymph in miliary tubercle, and in those of inflammatory exudations in scrofulous subjects. And thus, in brief, you have my key to the twofold seat and origin of tubercle, or rather of consumptive disease:—1. Lymphatic, miliary, infective, scattered. 2. Inflammatory, diffused, local. Thus we get explained the identity and yet the difference of all the chief elements of consumptive disease—phthinoplasms, as I call them—granular and diffused, differing in their form and seat, but alike in their corpuscular composition and in their proneness to decay. Dr. W. Fox says that he doubts that caseous tubercle originates from the exudation of white corpuscles. If he means that they exude in the caseous state, I doubt as much as he. But neither I nor any reasonable man can now doubt that white corpuscles do exude from inflamed bloodvessels, and we have abundant proofs that they form a corpuscular lymph which may turn either to pus-cells in suppuration (which is a process of excretion), or to a fatty disintegration in caseation, which is the condition of yellow tubercle. Suppuration results from continued inflammation, which involves a chemical change, a further oxidation of some of the protein of the corpuscles into a liquid tritoxide; but caseation results from lowered vitality, and is a process of decay. And now to conclude with a few words on *fibroid phthisis*, which, in opposition to Dr. MOXON, I hold to be a reality in both tenses, present and past, and yet, differing from Dr. Bastian, to be but still a variety of phthisis. In some of the microscopic views of tubercle which we have seen, in addition to the prevalent corpuscles, there have been an admixture and intertwining of fine fibres. And these are found not only in old miliary tubercles, but sometimes in recent granulations, and still more frequently and largely in the red and grey consolidations of chronic phthisis and pneumonia. Dr. has been the fashion to talk of these fibres in high Dutch, and to call them connective-tissue growths. I prefer the plain English notion of them which would call them fibrous, and trace their origin to the primordial fibrils found in clots of blood or liquor sanguinis, and forming part of the inflammatory exudation of serous membranes and other textures. I suppose my friend Dr. Beale will condemn me in using the word *fibrillation*; nevertheless I do apply some such term to the reality which is exhibited in these microscopic sketches of my friend Mr. Gulliver. They show the primordial fibrils which are formed in the spontaneous coagulation of liquor sanguinis, independently of cells, nuclei, or any other tissue element. There are distinct, fine, even fibrils, of uniform size and considerable length, crossing and interlacing each other—in short, just like those of connective tissue; and I do not see how we can avoid the conclusion that they may be the primary material of such tissues. In ordinary nutrition, and even in hypertrophy, the growth of tissues may be effected by the activity and proliferation of their proper cell-germs or bioplasm; but in inflammation and in similar states of vascular excitement there is an overflow of plasma, through the coats of the bloodvessels, with nascent materials, ready to form, without the aid of any cells or germinal matter of the old tissue. The result is the *coagulable lymph* of John Hunter, in all its varieties; the *fibrinous and croupous lymph* of Rokitansky; the *fibrinous and corpuscular lymph* of Paget; the *plastic and cacoplastic exudations* of your humble servant. These may account for the relations of tubercle or phthisis to inflammation more satisfactorily than the maze of growths in which the North Germans mystify the subject. The more fibrinous and less cacoplastic exudation becomes organised into a tough contracting tissue called fibroid and the like chronic consolidations. The more corpuscular and aplastic degenerates into cheesy matter, to soften and decay; this is diffused yellow tubercle and the results of scrofulous inflammation. These are the products of inflammation, confined to the inflamed part; but, by infecting the lymphatics, the matter may contaminate other parts in the form of scattered miliary tubercles. And all these phthinoplasms, whether they wither and dwindle like fibroid, or caseate and decay as tubercles and corpuscular indurations, represent different degrees of the same consuming disease, which brings the life-giving and flesh-forming materials of our body to premature decay.

DR. GREEN.—In listening to Dr. Wilson Fox's most able introduction of the present discussion on the relations of pulmonary phthisis to tubercle of the lungs, I think that there

were two main points upon which he especially insisted: firstly, that the anatomical changes in the lungs in acute miliary tuberculosis are precisely similar to those met with in pulmonary phthisis; and secondly, that the development of tubercle plays the most important part in the production of the latter disease. With regard to the first proposition—that the changes in the lungs in acute miliary tuberculosis are precisely similar anatomically to those met with in phthisis—I have but little to say. The recognition of this similarity is so important in studying the pathology of phthisis that I think, Sir, pathologists owe very much to Dr. Fox for the prominent way in which he has brought it before the notice of this Society. These changes, which have been so fully described by Dr. Fox, may be briefly stated to be of two kinds—the one an accumulation of large epithelial-like cells within the pulmonary alveoli; the other, the development of a small-celled adenoid growth in the alveolar walls, or in the interlobular tissue. The former of these growths Dr. Fox regards as an inflammatory product, whilst the latter he describes as *tubercle*. What, Sir, I am desirous of bringing before the notice of the Society to-night is the pathological relations which appear to me to subsist between these two kinds of growths. In order to state my views as briefly as possible, I would venture to express my belief—1st, that the accumulation of epithelial-like cells within the pulmonary alveoli, and the development of the small-celled adenoid growth in the alveolar walls and in the interlobular tissue, are both the anatomical results of the same pathological process—a process which comes within the category of what we understand by inflammation; and 2nd, that the predominance of the one or of the other of these anatomical changes depends mainly upon the intensity of this inflammation. That the growth in the alveolar walls and that within the alveolar cavities are both the results of one common cause appears to me to be evident from several considerations. In the first place, in a large proportion of cases of acute tuberculosis these two kinds of growth are so intimately associated—the nodules of induration consisting partly of the one and partly of the other—that it seems to me exceedingly unjustifiable to assume that they stand to one another in the relation of cause to effect. Then again the fact that in other cases the nodules consist entirely of the small-celled adenoid growths, and that this growth is sometimes so markedly fibroid that its development must evidently have extended over a somewhat lengthened period, clearly shows that this growth by no means necessarily causes any proliferation of the alveolar epithelium (endothelium). For these reasons therefore, Sir, I venture to submit that in these cases it is not a question whether the tubercle caused the pneumonia or the pneumonia the tubercle, but that both these products are the result of the same irritating agent. The point, however, to which I would especially venture to direct your attention is to what I believe to be the cause of the preponderance of the one or of the other of these anatomical changes in acute tuberculosis and in phthisis. Upon studying the alterations in the lungs in these diseases, and comparing them with those which result from inflammatory processes in other organs, I am led to believe that the greater the intensity of the inflammatory process, the more does it tend to produce proliferation of the large cells contained within the alveoli; the less its intensity the more does its influence tend to be limited to the elements in the alveolar walls and interlobular tissue. Further, that whilst the large epithelial-like cells invariably undergo retrogressive changes, the small-celled adenoid growth in the alveolar walls or in the interlobular tissue very frequently undergoes progressive development and becomes densely fibroid. In the most acute cases of tuberculosis and of phthisis, the principal anatomical change is an intra-alveolar one. In those cases of acute phthisis which have been termed pneumonic phthisis, the pulmonary consolidation consists almost entirely of the alveolar accumulation, and I must confess that in many of these cases I have failed to detect any marked change in the alveolar walls. The intensity of the inflammatory process not only determines to a great extent the anatomical characters of the pulmonary consolidation, but also the subsequent changes which take place in the small-celled growth in the alveolar walls. The large intra-alveolar elements, as already stated, always degenerate. If the intensity of the process be very considerable the small-celled growth also dies, but if less intense and more chronic it undergoes progressive development and becomes fibroid. I would here say one word respecting the death and caseation of the new tissue. This Dr. Fox regards as in great measure due to the obliteration of the capillaries by the tubercular growth. The death of the large epithelial-like cells which have accumulated within the alveolar cavities, as I have already stated, appears to be in great measure owing to their apparent inability to undergo further development, and it can be explained, I think, quite independently of any such interference with their nutritive supply. The non-absorption of the retrograde products, on the other hand, and the resulting caseous metamorphosis, is I think mainly due, as stated by the late Professor Niemeyer, to the interference with the circulation in the alveolar walls which is caused by the pressure exercised upon the capillaries by the intra-alveolar accumulations. It is in the most acute cases of phthisis, those which have been termed "pneumonic phthisis," that this death and disintegration of the consolidated lung occurs so rapidly, and it is just in these cases, I venture to think, that any adenoid growth in the alveolar walls, which might be supposed to interfere with the circulation, is almost entirely wanting. From the few observations I have ventured to make I wish to be understood to express the belief that the various anatomical changes met with in the lungs in phthisis are the result of inflammation, and that the difference in their anatomical characters and in the subsequent history of the newly-formed elements is mainly due to differences in the intensity and duration of the inflammatory process. With regard to the question as to what part *tubercle* plays in the production of phthisis, it appears to me, Sir, that the ground for attempting to make any pathological or etiological distinction between the small-celled adenoid growth which is developed in the alveolar walls and which is called by Dr. Fox tubercular, and the intra-alveolar growth which is termed by him pneumonic, are somewhat insufficient. Anatomically, as Dr. Fox has stated, it is often impossible to distinguish the typical miliary tubercle from certain other chronic inflammatory growths. For my own part, I must confess that I am unable to distinguish the small-celled reticulated structure of the grey miliary tubercles from that often met with in some portions of the indurated tissue of a cirrhosed liver. I cannot help thinking that the prominent part which the production of this adenoid tissue plays in these chronic inflammatory processes in the lungs is to be explained by the anatomical peculiarities of the pulmonary texture. This adenoid tissue is not only largely met with, as shown by Dr. Sanderson, in the neighbourhood of the minute bronchioles, but the recent investigations of Buhl and others seem to show that the alveolar walls are intimately connected with the lymphatic capillaries, and that the large cells lining them correspond with the lymphatic endothelium. In conclusion, I would say one word upon the question—What constitutes tubercle? In the first place, I think it is impossible to frame a definition of "tubercle" upon a purely anatomical basis. The small-celled reticulated structure which makes up the greater part of the miliary nodules in the lungs and in other organs, is met with in parts of the indurated tissue produced by many chronic inflammatory processes. If there be any anatomical peculiarity which might serve to separate tubercle from other chronic inflammatory growths in tissues closely related to the lymphatic system, I cannot help thinking that it must be looked for in the giant-cells which have recently been so prominently brought under notice by Dr. Schüppel. Respecting the significance of these cells, I will hardly venture now to express an opinion. I will only state that I have found them in the indurated tissue of a phthisical lung in which there was no naked-eye appearance of tubercle; and I am rather inclined to regard them simply as the results of chronic inflammatory processes in tissues intimately associated with the lymphatic system. On these grounds I cannot help thinking that the use of the term "tubercle" tends to cause confusion amongst pathologists; and I would again venture to express the opinion which I did in another place more than a year ago, that it would, on the whole, be advantageous to discontinue it.

Dr. CRISP said, in the remarks he was about to make he should endeavour to touch upon a novel line of investigation, not pursued by any of the previous speakers. He believed if we were permitted to look forward to the proceedings of this Society fifty years hence the question of the anatomical relations of tubercle and other diseases would not be confined to the study of the disease in man, but the question would be, In what kind of organisation was the lesion first met with, and how did it differ from that in the human subject? If this ascending line of investigation had been pursued, he believed that many of the discrepancies and differences now existing would have been removed. He would give a striking example of ignorance on this head. M. Villemin, in 1865, made this

extraordinary assertion: "It is necessary to state that tubercle in the lower animals is excessively rare, and excepting man, who has unfortunately a special aptitude for phthisis, there are only the ape, the cow, and perhaps the rabbit and some other analogous rodents, that are really susceptible of becoming tuberculous." Others who have pursued the investigation have been equally ignorant of their general occurrence among foreign animals in confinement. He had met with tubercle in more than 100 different species of animals, quadrupeds, birds, and reptiles, and he believed that there was scarcely a vertebrate animal that might not under certain conditions become tubercular. Tubercle was common among our London cows when sanitary conditions were less considered. He had examined nearly all our British wild animals, many of the species several times, and likewise many foreign animals in spirits, but had never met with tubercle in an animal in a state of nature. In 1854 there was a great mortality among the sparrows at the Regent's-park Gardens. He examined many of these and found the livers, spleens, and intestines tuberculous; these birds may be considered half-domesticated, and they moreover fed with a large number of tuberculous animals. In support of the conclusions he had come to, he showed numerous drawings and preparations, many of them made twenty years ago, for the purpose of throwing light upon this important question. He would now try the ascending mode of investigation he had spoken of. In the vegetable kingdom there was nothing strictly analogous to tubercle: we have abnormal cell-growth, and abnormal cell-contents, and subsequent death, but nothing exactly resembling tubercle. The nearest lesions to miliary tubercle were the nodes and excrescences produced in leaves and stalks by the *cynipidæ* and other insects. In the animal kingdom the effect of various irritants, such as mercury, stone-dust, and other extraneous bodies, was somewhat similar. But the specimens of grey, semi-transparent tubercle in the lungs of sheep, from a gordian worm, bear a greater resemblance. Here we have no transmutation into amorphous and caseous matter. Among invertebrate animals he knew of no lesion that resembled tubercle. Among fishes (especially pond fishes), he had sometimes met with hard fibrous tumours, with yellow caseous softening in the centre. Reptiles in confinement, especially the ophidians, are very subject to tubercle in the liver, intestines, spleen, and lungs; large masses of tubercle blocking often the whole of the alimentary canal. The liver is studded with small tubercular growths, and the lungs less frequently; nodules are present on the intestines; the spleen is frequently enlarged and tuberculated. Among the saurians he had frequently found tubercle, but up to the present time he had not seen tubercle in a batrachian reptile. Birds are in confinement very liable to tubercle, and the liver, spleen, and external coat of the intestines are much more frequently the seat of the lesions than the lungs; indeed, tubercle in the last-named organs is comparatively rare. He had observed four stages in fowls—first, the hypersemic, congestive, or inflammatory, when the spleen and liver are greatly enlarged; second, the deposit of cell-growth in the connective tissue, especially round the smaller branches of the portal vein and Malpighian corpuscles of the spleen. In the former organs, the liver, the tubercular nodules, when the organ has been immersed in water for a few days, can be seen attached to the primary branches of the portal vein. The third stage is that of softening or cell-destruction. The fourth the cretaceous stage; the tubercles become hard, and contain more than one-half of cretaceous matter. In other examples in birds the tubercular deposit is of a softer character, and assumes more the form of the lesion seen in some quadrupeds. In mammals, especially in the quadrumana, there is a nearer approach to the disease as seen in man, although here even there is an important line of demarcation, for the liver and spleen are more frequently affected than the lungs. There is one practical and important point that he had long since pointed out—viz., that the vegetable feeders, as in birds, are more liable to tubercle; that the deposit arises from faulty nutrition and impure air; that it is hereditary, and in the lower animals, he believed, contagious. Other distinctions he had also enumerated—the comparative absence of cavities in the lungs, the non-occurrence of hæmoptysis. Other differences are, the absence of tubercular meningitis—the comparative absence of cough and of wasting of the tissues—the absence of perspiration—the longer duration of the disease—the frequent tuberculisation of the liver and spleen, so rare in man—the larger amount of fibrous and cretaceous deposit, &c. Histologically the differences were not important. There is one important matter that bears especially upon the question as to the identity of miliary and caseous tubercle discussed by Dr. FOX. In the lower animals these two stages of tubercle are frequently found together; thus the miliary or semi-transparent grey granular tubercle is often present in the membrane and in the intestines, whilst the caseous or softened tubercle exists more frequently in the liver, lungs, and spleen. It seemed to him a matter of little importance whether miliary or, grey tubercle was only a stage of the caseous, or whether it was a distinct affection. One conclusion he thought was tolerably clear—that in all, or in nearly all cases of miliary tubercle in man, the disease was disseminated by inoculation—by the transmission of tubercular matter from some focus, or nidus, that has existed previous to miliary deposit; but as far as he knew, no such contamination could be traced in yellow tubercle, and this led him to the important question of inoculation. Unfortunately, those who have practised it appear (as in the case of M. Villemin) to have neglected to inquire into the anatomical, physiological, and pathological peculiarities of the animals experimented on. Thus neither the rabbit nor the guinea-pig, although often kept under conditions that would favour the development of tubercle, were either of them liable to it. He had examined a large number of guinea-pigs, but had never found one tuberculated. This was also the experience of Mr. Bartlett, of the Zoological Gardens. This led him to the question mooted by Dr. Bastian, Is the disease produced by the inoculation of human tubercle and by other extraneous bodies, true tubercle? He confessed that, looking to what may be called spontaneous tubercle in the lower animals, looking also to the morbid appearances and to the fatal termination of the disease, the results partake more of the nature of *pyæmia*. "Let me give two recent examples which I recorded in the recent volumes of our Transactions. A child under my care had a sore head; pus was absorbed, and a fatal termination was the result. In about twenty-one days I inoculated a guinea-pig with healthy pus from my own finger, and the animal died in thirty days with tubercular deposit in the liver, spleen, and mesenteric glands. If these cases are carefully studied, the difference is not so great as at first sight appears, and when the various anatomical and physiological differences that I have enumerated are considered, the conclusion that *true* tubercle is the result of inoculations becomes a very questionable point. Many of the animals inoculated die within the period of thirty or forty days from the time of inoculation, whereas, in spontaneous tubercle, the duration of the disease may be prolonged to a considerable period, sometimes I believe to many years." He had endeavoured in the short space allotted for discussion to point out a few facts connected with this disease in the lower animals that had, he thought, an important bearing upon the question at issue, and especially upon the effects of tubercular inoculation. "These are not German importations, but they are the result of many years of hard work and patient labour, and I prognosticate that the line of investigation that I have chalked out in the study of this, and of other diseases, is one that will be hereafter universally adopted."

Dr. JAMES E. POLLOCK considered that at this stage of the debate it might be well to review the doctrine which had been displaced, and that which it was proposed to establish in its place with reference to the applicability of the theories to actual clinical facts. Laennec, whose pathological teaching reigned supreme for forty years, had at least the great merit in his theory that he propounded a specific entity, and described a uniformity of progressive morbid actions; and it was found in practice that, while his theory was easily remembered and understood, it really did conform itself, as far as it went, to the observed features of phthisis in the living subject. This fatal disease, he said, has for its element an infiltrated morbid product, which, once deposited in the lung tissues, is never absorbed, but undergoes degenerative changes, involving the surrounding tissues in ulcerative destruction. Successive crops of these small bodies appear, till the lung becomes impacted, local inflammatory congestions take place, and the patient is carried off by the febrile disturbance caused by the softening of the infiltrated masses. A later pathology, under the guidance of Virchow, has taught us that the infiltrated product is not always found where there is phthisis or ulcerative wasting, and that the mass of what we do find in the lungs of persons who have died in advanced consumption is composed of cheesy and fatty degenerations common to products of inflammatory origin, or more special diseases, as syphilis. Both the French and the German master have recognised a miliary grey

deposit which might never soften, but impact the lung, as well as other organs, and is accompanied by acute febrile symptoms. Now, when Laennec called his supposed infiltrated product tubercle, he did so from its well-recognised physical properties; and before the days of microscopes these were aptly enough represented by the name. Histological investigation has recognised epithelial products, fibroid overgrowth, and lymphoid overgrowth, in every case of advanced phthisis. Virchow especially guarded us against studying tubercle by its properties after it became cheesy; for it then possessed characters common to pus, to cancer, and to sarcoma. In other words, varied inflammatory products were by Laennec mistaken for softened and aggregated tubercles: by a not unnatural transition the German school came to the conclusion that in the vast majority of cases of advanced phthisis the only appearances found were the products of inflammation and degenerative changes. Tubercle became limited to the miliary grey translucent deposit, and it was asserted that it only appeared as an incident in the course of advanced phthisis. In this transition of opinion we gradually lost the identity of the disease; its specific character vanished. Neither in the dissecting-room nor under the microscope could the so-called deposit be verified; all unity went with it, and we were left to the vague conclusion that not one but a multitude of affections might lead to ulcerative destruction of the lung. With the loss of identity and specific character all speculations as to the influence of heredity and other causative agents of course became vain; for why seek for special causes for a multiform affection? Dr. W. Fox, in his opening remarks, described his mental distress at finding himself thus perplexed, and by his propositions it appeared to Dr. Pollock that he had restored to us the unity which we had lost. The disease which all agree to call phthisis presented a remarkable conformity to one type, although it had many varieties. He was therefore inclined to accept the offering gladly, and present solution for phenomena which could not be accounted for by the theories of Niemeyer. The question of the nature of tubercle was no doubt still in a transition state; but it appeared to him that Dr. Fox gave a description which included every variety of phthisis as seen in practice, excluding only the acute general tuberculosis, pneumonia, and indurative pneumonia; all the elements found in the lungs of phthisical patients were included in the list of morbid appearances observed in the acute tuberculosis of children. Tubercle may have no individual features by which it might be infallibly recognised, but its vital and histological tendencies were unmistakable. The constant presence of lymphoid or adenoid tissue in the so-called tubercle might hereafter be disproved, but at present it was our nearest approach to truth, and Virchow and Latham still earlier pointed out the almost complete correspondence between the corpuscles of tubercle and the lymphatic glands. It was proposed to renounce the word tubercle, but if the disease in which it played so prominent a part remained a unity, it was better to retain the term, which in itself contained nothing contradictory of the latest composition assigned to it. There was no doubt we might have ulcerations of the lung without grey granulations, but it was very rare to find advanced cases without them. The new word "granulia" had not so much to recommend it as tubercle. Regarding the question from a clinical point of view, Dr. Pollock entirely believed in a miliary acute tuberculosis which need not of necessity proceed to ulcerative changes in the lung. But from a like clinical standing he must deny the accuracy of Niemeyer's pathology. It may not be that Laennec was entirely right, but the experience of large numbers of cases of phthisis contradicted the statement that the disease arose from a catarrh, although an epithelial impaction might be found to block the alveoli after death. Neither had he ever seen a case in which hæmoptysis preceded phthisis. The truth is, this branch of the German school constructed a picture of phthisis out of theoretical materials. The disease was to conform to the theory, and because blood-clots were subject to cheesy degeneration, and cheesy degeneration was asserted to be the causative agent in producing grey granulations, therefore the hæmoptysis was asserted to be the cause of the phthisis. But who has ever answered the question, What caused the hæmoptysis? Again, out of the débris of Laennec's pathology arose the theory of fibroid phthisis. Every case of phthisis which went beyond a very early stage contained the fibroid element, and, as had been well remarked by Dr. Moxon, fibroid phthisis was only old phthisis in which the contractile element was developed. Regarding the inoculation of tubercle and its propagation by infection of the system, the experiments of Dr. B. Sanderson and others were of the highest interest, but he doubted if a similar state to that which had been produced in the rodentia could occur in man. Finally, all the facts drawn from observation of phthisis, the powerful influence of heredity, the marked obedience to one type of all its cases, with diverging features in the individual instances, pointed to a unity in the morbid appearances as a causative agency, while the variety in the features of the disease could be accounted for by the preponderance of one or other of the morbid anatomical elements observed in advanced cases. For instance, an acute typical miliary tuberculosis with high febrile symptoms destroyed the patient often before the degenerative changes had time to occur, and therefore the latter were not observed post mortem. The chronic ulcerative destruction of lung where the vessels were blocked and strangled by the aggregation of the granulations afforded after death an abundance of cheesy degeneration and epithelial impactions. The attacks of pneumonic congestion to which the phthisical are so liable may be coincident, not only with increased inflammatory products, but with fresh crops of grey tubercles, possibly derived from secondary infection. The strumous variety of phthisis will be found to exhibit more of the true lymphoid growth, while fibrous overgrowths abound in very chronic cases with retracted chest walls and great shrinking of the pulmonary tissues. Out of the number of morbid products which Dr. Fox observed in chronic phthisis, there might thus be a basis for the several varieties of the disease as seen during life. Dr. Pollock desired to admit that, while a tubercle was a typical pathological entity which might be called tubercle more properly than anything else, the disease called phthisis was the manifestation of various morbid agencies, lymphoid, inflammatory, and degenerative, which in their various evolutions constituted the several varieties of the clinical affection.

Dr. BURNEY YEO said that Dr. FOX and the speakers who had followed him had based their remarks upon carefully observed post-mortem appearances. All were agreed as to the accuracy of these facts, but the difference lay in the interpretation to be given of them. He thought that the anatomical evidence would be better understood if all the circumstances in the history of the case were considered. It had been argued that what was called tubercle was not specific because it had no specific structure, that it was adenoid or resembled lymphatic gland tissue. But, as Dr. Beale has said, "histological similarity was no ground for specific identity." No one argued that the embryonic cells of a whale were really identical with the embryonic cells of an oyster, though histologically they might not be distinguishable. It was therefore from the life history of a morbid, as of a normal product, that we must judge of its specific character. It had been maintained that all the morbid deposits in the lung which had been regarded as tubercular were "products of inflammatory origin." This might have the merit of simplicity, but he thought it indefinite and unsatisfactory. These masses of deposit and infiltration might as easily and as reasonably be regarded as of tubercular origin. It seemed to him impossible, looking at anatomical details in the light of clinical experience, to admit the applicability of the theory of chronic inflammation to all cases of phthisis. It was equally impossible to regard them as exclusively of tubercular origin. Pulmonary phthisis might be separated into two main forms—the tubercular and the inflammatory. Both were complicated by secondary changes, which tended to confuse the anatomical characters. In the first, which he assumed to be hereditary, three chief circumstances determined its clinical course and anatomical results. 1. The degree of intensity of the original taint. 2. The influence of secondary complications. 3. The effect of time favouring changes in the morbid products. When the original taint was intense we had acute tuberculosis, and the grey granulation generally developed; if the taint was less we had acute phthisis, and in proportion to its duration we found it complicated by the products of chronic inflammation, the pro-

duct of lymphatic irritation, &c. In this way acute phthisis was directly related to acute tuberculosis. Such cases for the most part commenced with indications of a general constitutional affection, and often only after some weeks signs of local mischief were observed. Was this the least like the course of an extending catarrhal inflammation? In chronic phthisis they met with cases of the second class; he believed the most of these were of inflammatory origin. They began with definite signs of local inflammation, and often with no symptoms of constitutional affection. In chronic phthisis you met with the grey granulation radiating, as it were, from an infecting focus of caseous deposit; this, he thought, required further investigation. He agreed with Dr. Cayley that it was premature to give a definition of "tubercle." The statement that it was a "lymphoid overgrowth" was arbitrary and disputable, and not substantiated by anatomical observation. Looking carefully at the facts that had on all sides been adduced, it appeared more consistent to regard "tubercle" as the cause rather than the consequence of lymphatic irritation; and from this point of view he was anxious to hear what Dr. Wilson Fox had to say in answer to Dr. Cayley's remarks about these multinucleated giant cells, which some observers considered the most essential element of tubercle. He thought it would be rash in the extreme to sanction, as had been suggested by Dr. Bastian, the substitution of any such term as "granulia" for "tubercle." It would do nothing to remove the state of suspense which, if they desired to be honest and logical, they were compelled to adopt in regard to the precise relation of that deposit which is generally called tubercle to the larger infiltrations, concerning which there is dispute.

Dr. WILSON FOX.—In replying to the many able arguments which have been addressed to the elucidation of this discussion, I must apologise for the fact that to answer extempore those which we have heard this evening is by no means a simple or an easy task. To some which have been before us a fortnight I have been able to devote some consideration, but those heard for the first time demand, from the authority of the speakers, a careful reflection, in order to appreciate fully their bearings on this intricate subject. If, therefore, I fail to pay the attention which they deserve to some of these points, I trust that those gentlemen will not regard it as evidence of any want of respect on my part to the views which they have expressed. I cannot but feel grateful to one and all for the generous indulgence which the Society showed me on the introduction of this subject, and also for the great courtesy and kindness with which my remarks have been treated by successive speakers. I am glad also to observe that we have arrived to that medical facts at least, there is a considerable unanimity of opinion in this Society. That there should be considerable differences respecting their interpretation is no cause for surprise. When I introduced the subject I felt that some points which I was obliged to deal with somewhat summarily would be regarded as requiring further elucidation, and in this I have not been disappointed. I was, however, obliged to state conclusions, rather than to support them by argument, and for this reason I trust that the Society will permit me to explain in some measure the grounds on which some of these conclusions are based, and to enlarge on a few points on which some members of the Society have asked me for further explanation. As I stated when I introduced this subject to the Society, my object was to investigate whether there were such essential anatomical differences in phthisis as to justify the classification of its different forms as distinct diseases. In the investigation of the lungs of phthisical patients, in addition to pneumonic and fibroid changes, I found one common feature in the whole class—viz., a growth of small cells or nuclei, in some cases imbedded in a fine reticulum, while in others this reticulum was less apparent; but in all the cells or nuclei were densely massed, and were of the same character. In some parts this growth formed round masses, corresponding to the grey granulation in serous membranes, in others it was diffused through large tracts of the tissue of the alveolar wall and bronchioles. In the latter case it was usually mingled with pneumonic products, and in a very large proportion of what appeared to the naked eye as granulations it was also mixed with pneumonic products—that is, with epithelial proliferation in the interior of the alveoli. I regarded this growth as the distinctive feature of phthisis, whether acute or chronic, and it appeared to me that when diffused it was of the same nature as the circumscribed masses, the grey granulations. I was, however, long under the conviction that the grey granulation was the typical form of tubercle, and therefore I felt doubt as to what the character of this diffused growth really was. I therefore determined to investigate the pulmonary manifestations of a recognised tubercular disease—acute generalised tuberculisation in children, and I found here, as I have stated, the same sets of changes—viz., circumscribed and diffused growths of the same nature. I argued, therefore, that in the generalised disease a growth similar in its nature, similar in vital characteristics, and similar in its changes, occurring in the same disease in the same patient, but differing only in being in parts circumscribed and in parts diffused, must be in all probability of the same nature, and that if the circumscribed growth—the grey granulation—was tubercular the diffused growth must also be so, and that if this was tubercular in acute tuberculosis it must also be so in other forms of phthisis. In bringing the subject before the Society I thought the most definite course would be to invert my own procedure, and to inquire first what was tubercle in the lung; and this was the reason why I devoted so large a part of my description to the changes occurring in acute tuberculosis. To Dr. Payne's and Dr. Cayley's inquiry what part of the various morbid changes in this disease I consider characteristic of tubercle, I would reply that in the abstract which I furnished, and also in my description, I categorised all the essential changes of whatever kind, but that I withdrew all the simple inflammatory changes from this category as well as the accidental ones of emphysema, collapse, dilatation of the bronchi, congestion, œdema, and ecchymoses. I endeavoured, however, to show that all the granulations, except some of the earliest spots of lobular pneumonia, had one character in common—viz., this growth in greater or less abundance. Some of the granulations are pneumonic, with, however, this growth superadded in their wall. The epithelial proliferation is not, in my opinion, characteristic; it does not differ from that found in ordinary catarrhal or lobular pneumonia; but the growth, whether diffused or circumscribed, is characteristic, and, from its similarity to it, its identity with the granulation, it still call it tubercular. The products of ordinary inflammation are, in the lungs as well as in the serous and mucous membranes, almost always found co-existing with tubercle; but as in the serous membranes the distinction can be maintained, and as the growths can exist without them, I think that they must be regarded as non-essential in the anatomical sense, though I believe with Dr. Green that in some cases they originate under the same cause, or they may precede and even excite the tubercular growth or follow it. The tubercular growth is something superadded to this, and gives to the pneumonia characteristic features. It causes the prominence even of the softer granulations, and it leads to caseation—necrosis—by destruction of vessels, which does not occur in ordinary pneumonia. I think it desirable that the inflammatory process and the new growth should, from an anatomical point of view, be considered separately, though their relations to one another are so intimate. Anatomically, pneumonia is not tubercular unless this growth co-exists in the alveolar wall. When this is present I think the pneumonia may be conveniently called a "tubercular pneumonia"—that is, a pneumonia associated with tubercle; and this association is often more common in the lung, in acute tuberculosis, than the typical grey granulation—that is to say, a large proportion of the granulations in this disease show some pneumonic changes combined with the growth. I hope, therefore, that it is clear to the Society that I do not consider all the changes in the lungs in acute tuberculosis as tubercular. I limit the term tubercle to this growth. The criticisms that have been directed to the conclusions which I have formed may, I think, be summed up under the following heads:—Firstly, that as the growths described, whether existing as granulations or in a more diffused form, are wanting in absolutely specific histological characters, it is impossible to separate them from other diseases. This point has been distinctly affirmed by some speakers, and appears to me to underlie the argument of others. Some, however, think that the form is distinctive, and would therefore still limit the term to the grey granulation. Secondly, that the dis-

ease known as acute tuberculosis, though presenting the same granulations and growths as ordinary phthisis, is yet so widely separated from it as to form no criterion for an anatomical analogy between the lesions of the two diseases. Thirdly, that the one lesion known as the grey granulation is not necessarily the mark of one disease, but may include several which are distinct from one another. Fourthly, that the grey granulation presents certain cell forms which are sufficient to characterise it and to distinguish it from the diffused growth and other granulations presenting, in other respects, similar characters. I shall endeavour, as far as lies in my power, to deal with these objections *seriatim;* and if I am obliged to enter, in relation to some, into a more abstruse argument than I had originally intended to bring forward, I think that it will be acknowledged that these points must be fairly met in the discussion of this question. I can only endeavour, in spite of the difficulties of some of these subjects, to do so as briefly as possible. I will allude first to the last which I have named—viz., a criticism introduced by Dr. Cayley. If I understand him rightly, I gathered that he regarded the "giant cells," the "Riesenzellen" of Virchow, as characteristic of the grey granulation of true tubercle; at least he stated that I had not found them so frequently as others, because much that I considered tubercle was not really so. My remark was, however, confined to the grey granulations proper in the lung, or at least to such as appear so microscopically. If, however, we examine a number of grey granulations in respect of these cells, we shall find them, at any rate in such sections as we make, in a very small proportion; but the grey granulation, under the microscope, is easily recognised without them, and has been so recognised, before their frequency was observed, by the dense rounded mass of small cells and nuclei, with and without a reticulum. If we exclude all otherwise typical granulations in which we cannot find these cells (I do not say in which they do not exist) from the category of tubercle, we shall, I think, find a very considerable further reduction of the bodies that bear this name. Failing this characteristic, I would assert that, with the further exception of its rounded form, the grey granulation has no feature to distingush it from the more diffused growth, which, as I have just stated, presents in all other respects essentially the same histological structure and vital characters. I must demur to the opinion that the rounded form is sufficient to separate the grey granulation from the diffused growth around. In the first place, it merges insensibly into it; and though Rindfleisch, to whom Dr. Cayley has alluded, makes the distinction in the intestine when he speaks of a diffused growth around ulcers as being inflammatory, while grey granulations also exist there, he admits that in the lungs processes, or irregular growths—such as have been very well figured by MM. Hérard and Cornil,—extend between the granulations. I admit that many grey granulations are tolerably sharply circumscribed, but in many, equally typical in other respects, the same growth extends indefinitely into the alveolar wall; and I believe we cannot then say that the latter is different in essential nature from the former when thus extending from it. Again, when tubercle—what is universally admitted to be tubercle—grows in this sheath of an artery or bronchus, the extension is longitudinal; and though it tends here also to form nodular masses, there is as much a diffusion or infiltration within the sheath as there is in the alveolar tissue of the lung, and the round form is often only an accident due to the section being made transversely. Mere roundness of form and mere circumscription cannot, therefore, be affirmed as being essentially distinctive of even typical tubercle. I believe that, whatever be the real nature of the granulation, whatever characters it possesses are possessed equally by the diffused growth—the same histological structure occurring with it, and having the same vital tendencies. In the specimens which I brought forward of tubercular growth in an arterial sheath extending into the adjacent alveolar texture, you cannot, I think, logically say that the tubercular growth is limited to the former. The growth is the same; and, whatever it is in the arterial sheath, it is, I believe, the same in the alveolar wall. You cannot, when they thus occur together in the lung, call one inflammatory and the other non-inflammatory; you cannot, except by a mere arbitrary definition, call one part tubercular and the other part scrofulous pneumonia, or arbitrarily draw a line of demarcation between the different aspects of a similar growth in the margin of an intestinal ulcer. I do not think that we ought scientifically to separate the granulations which contain some epithelial inflammatory products, but in which this growth occurs, from those which contain none—to call the latter pseudo-tubercle or chronic lobular pneumonia, and limit the idea of tubercle to the former when they both occur together. I lay in this case especial stress on their concurrence, for reasons into which I shall enter presently. The recognition of tubercle without the grey granulation may be and is sometimes a matter of doubt and difficulty, but in the case of acute tuberculosis I believe that we must regard these diffused growths as being of the same nature as the circumscribed form, and on this point I would quote an aphorism of Virchow:—"The form ought only to be admitted as a decisive criterion of new formations when it is conjoined with a real difference in the tissue, and does not result from accidental peculiarities of situation or position." Is there any real difference in the nature of the grey granulations and the diffused growths? In my opinion this is not discoverable by histological characters, nor by the transformations to which they lead, nor by the circumstances under which they originate. This is admitted by nearly every member of the Society who has discussed this point, including Dr. Bastian, to whose singularly able address, with its further details published *in extenso*, I shall have to make more than one allusion. I would, however, remark that I somewhat differ from him in the interpretation which he puts upon the writings of some pathologists when he says that these diffused growths "amidst the shipwreck of the old term (tubercle) were deliberately cast aside" out of this category. He considers that I have lost sight of the fact that the definition of tubercle, as consisting only of the grey granulation, was "confessedly arbitrary," for a certain purpose. Now, "arbitrary" was the very word that I used in introducing the subject; but even in the imperfect revision which I was able to give to the report of my *extempore* remarks, I did not choose advisedly to retain such an expression as applied to any definition of Professor Virchow, as being capable of being understood to imply less of the personal respect and gratitude which I entertain towards him; and, without dwelling on the subject as I would gladly do, I cannot quit it without expressing how great is my sense of the obligation due to him for the stimulus which he has given to pathological inquiry, not only on this but on nearly every important branch of medicine, and how vastly he has increased our actual knowledge. On this subject of tubercle in particular I feel that I should have wanted a guiding clue to most, and these some of the obscurest points in this difficult subject, without his luminous exposition of its morbid anatomy and without the references collected by his profound learning in his history of the "Morbid Growths." But when Dr. Bastian asserts that these diffused growths have been categorised by other authors under the term chronic and interstitial pneumonia, I would, from my own reading, which has been directed somewhat carefully to this point, state my impression that though the presence of chronic pneumonia and induration has been affirmed, especially by M. Lebert, both for these and also for a large proportion of the granulations present (pneumonie disseminée chronique)—though I confess that I am unable fully to understand the distinction which he makes between these and the grey granulations,—neither in this sense nor in that of an "interstitial pneumonia" (a term which, as applied to them, I consider as essentially incorrect and misleading) has their identity of structure with that of the grey granulation, from which they may be seen extending, been distinctly affirmed until recently by Prof. Buhl, and previously by Dr. Sanderson, whose views on this point, as far as I can judge (though in his absence I scarcely venture to quote him), correspond in many important points with those I have laid before the Society. I do not recollect to have found it stated, except by these authors, that these growths are of the same structure and pass through the same changes as the grey granulation, nor do I find anywhere the proposition, as stated by Dr. Bastian, that they must, in spite of this recognition, be avowedly rejected from this category when they occur with it, because we want an artificial definition of tubercle; and form (not only form, but a certain form) and appearance combined are the only very positive criteria at our disposal, and that we must, therefore, for convenience sake,

in our phraseology, draw the line here, and arbitrarily choose to describe these changes, which are apparently similar in nature, in different terms—implying a real dissimilarity,—to call the grey granulation tubercle and all the other growths chronic inflammation. Such a mode of definition thus stated would have been intelligible, but I do not think that it would have stood or that it will stand the test of criticism or of practical experience. What I think was first attempted was to distinguish the grey granulation from caseous change; then grew up the idea that the grey granulation was the only tubercle, and the similarity of these growths was overlooked, and the process by which the caseous change is most commonly produced in phthisis was, I believe, mistaken. Every form of tubercle has been called chronic pneumonia by some authority or another, but to this point I shall presently allude. Nor do I think that Dr. Sanderson or myself can be said to regard some of the "old infiltrations" as being tubercular. These were largely pneumonic, consisting of products occupying the interior of the alveoli. In this sense of the word I agree with Professor Virchow that tubercle is not an infiltrated product; and, owing to the misunderstanding that may arise from this term—which is not very etymologically accurate, though it is, with respect to these growths, as applicable to tubercle (if they be tubercular) as it is to cancer,—I think that they had better (though I have used the former term) be described as "diffused," in contradistinction to the "circumscribed" form. I would now refer to some criticisms which have been directed to the anatomical peculiarities and nature of this growth, and especially to the term "adenoid" or "lymphoid," as applied to them. I used generally the latter phrase, or styled them lymphatic; the word "adenoid" I employed as a quotation from my friend Dr. Sanderson. I greatly regret his absence, because he would have been able to give a much more complete exposition on this point than I am able to do. I had thought that the word "lymphoid," as introduced by Virchow, had become so familiar a phrase, as expressing one of the peculiarities of tubercle, that it required no further explanation. Virchow long ago drew a parallel between the structure of the grey granulation and an isolated lymph follicle, and stated, and I believe accurately, that in some places, as in the spleen, it was almost impossible to distinguish the one from the other. The term is one of resemblance, and does not affirm identity of structure. Since the publication of Virchow's Cellular Pathology our knowledge of the structure of lymphatic glands has been greatly extended by the researches of His and Frey. We know from their researches that all these bodies possess a very complex structure, involving the distribution and reunion of afferent and efferent ducts, and that the glands consist of two parts, a medullary and a follicular portion. The composite structure is only found in the larger glands, and not in the isolated follicles of the intestines, which His believes to be only aggregations of the diffused adenoid growth in these parts. The main tissue is, however, composed of a reticulum in which cells lie imbedded, though these are more developed and the reticulum is less distinct in the follicular portions. It is to the follicular parts of the gland, or to solitary follicles, that typical grey granulations bear the greatest resemblance; but the resemblance is one of tissue, and not of anatomical structure, and it is, after all, only a resemblance, and not an identity. The tissue thus formed has been called by Kölliker and His "cytogenic," and has many anatomical variations and distributions, into which I cannot enter. It is, however, as far as our present anatomical knowledge goes, a derivative of the connective tissue. In this sense I think the likeness of tubercle to these structures may be maintained, as far as concerns the reticular structures in which such cells are imbedded, though in tubercle, as in the lymphatic glands, larger polynucleated cells are not wanting. This tissue is not, however, as a question of normal anatomy, necessarily circumscribed, but it occurs in diffused areas, particularly in the submucous coat of the intestine, and the circumscribed lymphatic masses are only to be regarded as modifications of this structure, with which, except in details of anatomical arrangement, they closely correspond. I am quoting entirely from the researches of His, Frey, and von Recklinghausen, though as far as my observations have gone I can largely confirm their statement in the latter point; but the fact that these two forms exist naturally, and that in embryological development the one proceeds from the other, affords, I think, an important analogy and clue to the nature of some new formations. To return to the description of tubercle as a lymphoid structure, it may be remarked that, as Dr. Sanderson and myself and others have shown, it frequently arises from smaller conglomerates of the natural adenoid or lymphoid tissue. It also arises in the sheath of the arteries and bronchi, which are believed with great probability to be of the nature of lymph spaces. Mixed types of tubercle have, therefore, not only a lymphoid or adenoid structure, but have also a lymphoid origin; and I think, therefore, that this term may be appropriately applied to them. As regards the diffused growths, I have stated my belief, which has been confirmed by other speakers in this discussion, that they have the same structure as the grey granulation. That they have the same anatomical origin in the cases where that of the latter can be shown, is not so easy of proof. I hypothetically stated the possibility of their origin and extension from the lymphatic plexus of the lungs. Dr. Beale doubts the extent of this; and I should place the greatest weight on the criticism of so accomplished an anatomical observer. I have no personal observations to record on this point; my confirmatory evidence was based on the observations of Sikorski, who announces the discovery of a plexus in the air-vesicles, which, from his description, presents the closest analogy to the origin of lymphatics in other parts, and which has be traced also through the bronchioles. Whether this be accepted or not, we may, I think, fall back on the fact that there is a delicate nucleated membrane in the walls of the air-vesicles, which may serve as the origin of this growth, and which is allied to the connective tissue series, this again allied and indeed more than allied to lymphatic structures; it being shown by Sertoli and Schmidt that the latter in their embryological development proceed from the former. I have gone into these details of explanation about the name because, as Dr. Moxon has well remarked, names should not be loosely used. The name lymphoid, as commonly employed—and I observe that it is often employed by many—denotes a resemblance, but not an identity. The degree of resemblance may be a question, and in this respect it varies in different specimens. Even when the grey granulation proceeds from a true lymphatic structure the identity of structure between the new growth and the tissue in which it originates is destroyed; and notably in this peculiarity, that a lymphatic gland is vascular, while a tubercular growth is absolutely or nearly absolutely non-vascular; but the resemblance in tissue to a greater or less degree remains, and sufficiently so, I think, though this may appear in a different light to others, to justify the retention of the name. Dr. Williams thinks that the resemblance is so far destroyed that nothing but an overcrowded mass of corpuscules is present. I should hardly venture to reassert my own opinion that there is a reticulum also, were it not that he has stated that fibres are also found in older specimens, and that Professor E. Wagner has also described that much of what is generally recognised in Germany as tubercle is a reticulated lymphadenoma. This brings me to one of the points involving the greatest difficulty raised in this discussion. I do not mean a personal difficulty, for I do not wish to enter into any special pleading; but a difficulty which meets everyone who attempts to define the series of new formations, in which a somewhat similar structure appears, from one another, if we look merely to their histological characters. It was a difficulty which I intended to express by stating that the characters of the new growths in acute tuberculosis were distinctive but not specific. This phraseology may meet with criticism, but by it I mean to express that their general characteristics distinguish these formations from *simple* inflammatory processes in the lung and other organs; but they are not specific—that is to say, they are more or less closely shared by formations occurring in other diseases—glanders, typhoid, leucæmia, and it is stated in other chronic inflammations, in some syphilitic growths, and in the class of lymphosarcomata, and lymphadenoma. With some of these my acquaintance is but small, as with glanders; but it must be admitted, from Cornil and Ranvier's description, that there must be, histologically, very little difference between the appearances which this disease produces in the lung as

regards the implication of the perivascular and peribronchial sheaths and tubercle occurring in these regions. Of syphilis also I can say but little; but I would say that during the past fortnight I have looked through the drawings and descriptions of many of the authors who have described secondary syphilitic growths, and I can find little or nothing corresponding with any close approximation to tubercular formations in the lung, except in one by von Baerensprung, of probable syphilitic disease of this organ. There are scattered masses of cells and nuclei imbedded in fibrous tissue; but these are less dense than in tubercle proper, they are more widely separated by fibrous tissue, and they occur in little groups. I do not wish to dwell on finer distinctions, though I would make the same remark of the appearance of the base of a chancre. Here, also, at least in some specimens which, thanks to the kindness of Mr. Arnott and Dr. Gowers, I have been able during the past week to compare with the lungs of acute tuberculosis, I find such differences that if I had met with these appearances in the lungs I should have called them tubercle; they are not so fused with the basis substance. They appear more isolated, and again they occur in scattered groups, between which is proliferating connective tissue. Here, again, it is a question of degree, the variations in which is almost impossible to express verbally. Billroth and Wagner have, however, described a true cytogenic tissue at the base of chancres, and I accept their statement. In leucæmic growths, especially in the liver and kidney, there is the greatest difficulty, I would say impossibility, of histologically distinguishing between them and the grey granulations in the same situation. Of the appearances presented by the lungs when affected by this disease I have no experience. In typhoid again, at least in the general infiltration of the intestine, the resemblance to the cytogenic tissue found in tubercle is so close that, histologically, it would, I think, in many cases, be almost impossible to distinguish the tissue at the base of the ulcers, in these diseases, from one another; and the resemblance is still greater from the fact that in typhoid you may have multiple disseminated small growths, as Wagner and others have shown, in the liver, peritoneum, lymphatic glands, and air-passages. Indeed, as Virchow long ago remarked, anatomically as well as clinically, the diagnosis between acute tuberculosis and typhoid may be a matter of the extremest difficulty. Here, even in well-marked diseases, we have a whole group of very similar changes of structure. And I would go further. I would say that in nearly all new formations arising in the so-called connective tissue you may have almost identical appearances, but at different stages; or, as Dr. Beale has well put it, in the earliest periods of growth it is impossible to differentiate one bioplasm from another; and in many instances it may extend to later periods of formation. At any rate, in the connective tissue series, the products of inflammation may at certain periods very closely resemble the processes of morbid growths of very different kinds, and the difficulty of expressing these differences is at times extreme. Hence I said that I would give no dialectical definition of tubercle. I believe it would be almost impossible to frame any definition even of the histological changes in common suppuration, in which the attributes predicated of it might not be equally applicable to cancer; and again I have preparations of cancer of the lung which in some respects present a close resemblance to tubercle; while Dr. Bastian has affirmed the same ascending series, with no line of demarcation, for the peritoneum. No one can affirm more strongly than I do the absence of histological specificity for tubercle. Such absolute specificity is denied also etiologically in every phase of its history, except the hereditary tendency, and how far back this reaches it is impossible to say, for when we know family histories it is traceable further than in statistical hospital inquiry; but that the disease may originate *de novo* under various unhealthy influences, none of which can be called specific, is a fact which I think no one can deny; while as to the diathesis M. Pidoux at least asserts that it may be the expression of any diathetic constitution when the primary manifestations of this are exhausted in successive generations. I do not adopt this mode of expression, but it conveys in some cases an approximative truth. I said in my introduction that I should abstain from etiological considerations, and must still pass them by; but I wish to express my opinion that we have no more right to attribute to tubercle a specific form than to attribute to it a specific structure. At least, if we do, we exclude from the disease a vast number of other wise similar structures, which in some cases predominate in its manifestations. Take the illustration of acute tuberculosis, and I would ask where in the lungs would you draw the line between the varieties of granulations found there. They differ from one another in structure as a whole, but they contain one structure common to all, and common to all the manifestations of the disease throughout the body; nature as the circumscribed masses; and, if this be true, the definition of the grey granulation as the sole form of tubercle is too arbitrary to express the phenomena of the disease. I have no wish to add to the confusion of this intricate subject, but I think that we may as well look the logical impossibility of framing a dialectical definition based solely on the histological characters of any single general disease fairly in the face and admit it. Histological identity is one feature necessary to prove the identity of similar growths at the same period; but it alone does not prove identity, for only the collective characters determine their further changes that enable us to discriminate them, and even here we are often in difficulty. Take multiplicity of anatomical changes, even when combined with structure, and we have it in a vast group of new formations, in some of which, tubercle, it is sometimes apparently of an infective kind, as in leucæmia and lymphadenoma. The tendency, to a greater or less degree, characterises at least nearly all the diseases of the connective tissues, including even the process of suppuration; but yet, as a practical fact, we may distinguish many of these diseases from one another. Where I think we shall be in pushing any single feature to its extreme. Take, for instance, suppuration and tubercle. The resemblance of tubercle, in its constituent elements, to pus, was, fifteen years ago, affirmed by Virchow, and even his classical figure of a typical grey granulation differs but little, except in the size of the cells, from a small nodule of commencing suppuration. Years before that Dr. Williams stated that no boundary line of definition could be drawn between tubercle and inflammatory processes, a proposition which he has recently reasserted, and with which I fully concur. Is there, however, no difference between them? Is every suppuration tubercle, and every tubercle suppuration? The question may be absurd, but anyone trying absolutely to state their differences in all stages would find this, I believe, an impracticable task. He must recognise diseases by their broader features, and this is what I have wished to do in discussing this question of the relation of tubercle to phthisis. I have dealt with tubercle as a disease, and I have affirmed my belief that the sole form of its anatomical manifestation is not the grey granulation, but that it occurs in other forms. Now the question meets us, firstly, is it distinguishable from other and recognised diseases? In the majority of those which I have enumerated, though in some cases the pulmonary manifestations may be similar, we have other criteria in nearly all. But perhaps the greatest difficulty that can meet us will be in syphilis, typhoid, and tubercle. For isolated specimens of the two last-named may, I believe, be found which, when placed under the microscope, would be indistinguishable from tubercle. I admit that the question of the relation to phthisis of these may be at times very difficult; and I at once admit that I have no positive definition to give, partly because I have had few opportunities of examining destructive changes of the lungs occurring in these diseases. In one case, after typhoid, the granulations resembled the softer forms of acute tuberculosis. In all the cases of phthisical patients with a past history of syphilis coming under my observation, the appearances in the lung differed in no respects from those of ordinary phthisis. In one case which I recently met with presenting syphilitic gummata in other parts of the body, there were a few similar bodies scattered through the lung, and these were

very distinct from any appearances seen in phthisis; but there was no destruction of tissue—no phthisical disease. I have not, however, been able yet to subject these to microscopic examination. The argument of the non-specificity of tubercular new formations is, I think, pressed somewhat unduly if we say that the disease is, except in the form of the grey granulation, undistinguishable from other diseases. In the first place, the majority of those quoted are either more or less specific in their origin—as syphilis, glanders, and typhoid,—or have, as in leucæmia, other distinctive features. When we eliminate these, we have little remaining with which phthisis can be confounded except some of the processes of inflammation. I have already here, and I shall have to repeat, how great is the affinity here, but it is an affinity only to be taken practically in a special sense. Inflammation, as a process undergoing evolution, does three things—it either resolves, or it suppurates, or it passes into a chronic stage, sometimes ulcerative, sometimes indurative. The first two may be excluded. The question lies in the chronic stage, and may be answered from two aspects. The first is the anatomical: as regards ulceration in ordinary chronic inflammation, it also is in the main suppurative, and the tissue is infiltrated with cells, large, more loosely packed, and differing in actual appearance from the growths of tubercle. Moreover, it is not preceded by caseation, as in tubercle, and the vessels grow in it and are not so directly destroyed by the growth. As regards indurations, though a nuclear and cell growth appears in these, it is less dense, and passes far more rapidly into fibrous tissue. These, again, do not undergo caseation. When caseation occurs as a consequence of inflammation, it is due, in the vast majority of cases, simply to retention of preformed pus—it is not the first immediate change in the cells of new formation. It is said that caseation is common in the lung because its structure facilitates retention of inflammatory products; but when we look at other glands where retention is even more easy, as for instance, the mamma, the parotid, and the liver or kidney, do we find simple chronic inflammation attended with this nuclear growth and caseous change *combined?* Even pus is probably long before it undergoes the latter change. In most indurations or other nuclear growths of the kind, which we know as chronic inflammatory products, it hardly ever occurs, if at all. I wish to be distinctly understood that I am not speaking of all caseous matters found in the lung—I am only speaking of the changes in the growth which are said to be not specifically definable from processes of ordinary inflammation. Of the origin of the diffused caseous matters in the lung I have already spoken, and shall have again to refer to them; but I do not know, either from my own observation or by that of others, of simple chronic inflammations producing growths of precisely the same nature as those in phthisical lungs with the same dense growths of reticular structure, not suppurative, and with the same fatal tendencies. To call disease in the lungs, therefore, *mere* chronic inflammation, is to state an opinion of their nature little supported by analogy. The boundary line, I admit, may be indistinct, but the broader features on either side differ to a marked degree. That nuclear growth does occur in inflammation, and does occur in some chronic indurations (whose inflammatory nature is less distinct, and is denied by some) may perhaps be considered as proved, as in such cases as early stages of cirrhosis and of the granular contracted kidney, referred to by Dr. Bastian. Such appearances, however, are not common, and certainly are not the predominant features of the fibroid thickenings in that disease, but are on the whole very exceptional to any marked degree in them. They do not undergo caseation; they do not indurate in the same manner, and, as far as I have seen, do not present any true resemblance to those which are found in the walls of the alveoli in acute tuberculosis and phthisis, and the resemblance is only on the side of induration, and not in the tendency to caseation. And I would ask, are we to take these exceptional appearances as an adequate ground for stating that phthisis and tubercle are not a definable disease? Again, there is an etiological side to this question. The mere existence of "chronic inflammations," with the exception of these two diseases last quoted, is comparatively rare without constitutional or local weakness or mechanical or chemical causes. In cases, however, where these are not proveable, as in chronic catarrh of certain mucous membranes—and of these, in relation to the lungs, I will especially quote chronic bronchitis, which in an otherwise healthy person may last for years without producing any growth significant of phthisis,—it is the old argument, as raised by Laennec and Louis, that the most persistent inflammation of this kind was insufficient to produce phthisis. In the stomach the chronic catarrh of hepatic congestion is rarely attended by enlargement of the lymphatic follicles. In phthisical people it is so very commonly. Chronic dysentery presents the nearest analogy, and forms one of those diseases where the boundary line between irritation of the lymphatic apparatus, as seen in tubercular and in non-tubercular conditions, is the least defined; but the vital character of the growth differs, for caseation is the exception in dysentery. There are intermediate stages on either side not accurately defined, for tubercle may soften so acutely as to resemble suppuration, though caseous change is rare in inflammatory products except in its presence. I only point out that the broader features differ, and clinically a great proportion of the chronic inflammations appear in persons to whom that ill-defined condition, a tuberculer or scrofulous condition, is attributable; and when this is not present there is usually some other form of constitutional cachexia; but the inflammations in the former class have characteristics, not absolutely definable, but different from the latter. In the inflammations of the serous membranes we have perhaps one of the best contrasts: we have a chronic pleurisy without tubercle, and a chronic pleurisy with tubercle. A chronic pleurisy in itself always affords the gravest ground of suspicion for a constitutional state in the background,—Bright's disease, or cancer, or tubercle; but the first-named presents no special growth; the latter presents growths similar to the grey granulation, and which, until Dr. Bastian's new position was introduced, have been almost invariably regarded, with the exception of Andral, as being of this character. To sum up, I would say that the so-called chronic inflammations of the pulmonary tissues are most commonly attended with other evidences of tubercle, and are very rare except in its presence, and that they then present many marked distinctions from the processes of inflammation not so associated. Another question remains behind. Do the formations which we recognise as tubercle because characterised by the bodies which we know as the grey granulation, represent pathologically more than one essentially distinct constitutional disease? This is the question which has been raised by my friend Dr. Bastian, and argued by him with such great ability. Now, though I have argued that such bodies are not the sole form in which tubercle can appear, I admit they form its most distinctive character; and therefore I started from its structure in investigating the other changes found in the lungs. I think, also, that we may generally admit that the grey granulation is not usually produced or imitated very closely in the other diseases of the lymphatic class which have yet been characterised, except these we have only what we have hitherto regarded as one. Now this formation appears in the disease which we know as acute tuberculosis, most forms of phthisis, and certain inflammations of the serous membranes, in which it is very seldom found alone. Does its presence signify under these varying circumstances many diseases or one disease? —diseases, I mean, as different from each other as leucæmia, or glanders, or lympho-sarcoma, or lymphadenoma are in turn from one another or from what we call collaterally tubercle. If I understand Dr. Bastian's argument aright, they may. I understand that he means that there are granulations in ordinary phthisis having identical nuclear appearances and histological structure to those occurring in acute tuberculosis, but which have yet an absolutely different pathological signification. He adduces also illustrations of the same character from a granulation disease localised in the peritoneum, hitherto known as tubercular peritonitis, but which he regards as again different—that is, having no necessary pathological affinities to the two former. Dr. Bastian, therefore, questions the consequences of my reasoning from the grey granulation as occurring in acute tuberculosis being applied to define tubercle generally. According to him the grey granulation is only to be called tubercle (if the name is to be retained) when it occurs in acute tuberculosis. The same identical anatomical form and structure occurring under any other conditions may be another disease not yet named, or to be called granulation.

Now I would for a moment call attention to the fact that this is not Virchow's definition. Virchow's definition was of the grey granulation in the abstract, wherever found, and the definition in question is, if Dr. Bastian will allow me to say so, a second arbitrary one engrafted on a previous arbitrary one (using the term arbitrary in the non-invidious sense in which he applies it). The argument is not, however, unfamiliar to me. I have long thought that the largest outcome of Niemeyer's views would be that there is no tubercle except acute tuberculosis, but as an anatomist I have felt the combination of anatomical form with anatomical structure of these granulations, coupled with their pathological affinities and their vital tendencies, to be an insuperable objection in my own mind to accepting this doctrine. The distinctions of acute tuberculosis on which Dr. Bastian relies are mainly these: simultaneity of affection of a great number of organs, and therefore involving multiplicity; and acuteness of course. Now, on all these points I would venture to assert that the disease, as we have hitherto known it, presents very great variations. In M. Empis' book and also Colin's and Wunderlich's cases I would observe that these excellent clinical observers show a series of successive invasions of different organs, often attended with intervals of remission, and extending over comparatively considerable periods of time, and that the characteristics of the disease vary with these variations of site. There is a cerebral form, a pulmonary or acute asphyxial form, and an abdominal form. Secondly, that multiplicity is also very variable. I hardly know what Dr. Bastian will accept as acute tuberculosis, seeing that he excludes Bayle's cases of granular phthisis from this category. Still, however, I would point out that a disease in the adult running the course of acute tuberculosis may, as in a case recorded by MM. Hérard and Cornil, be limited to a single lung; and even in the multiple disease I am acquainted with three other recorded cases where, in addition to other lesions, one lung alone was affected, the other remaining free. What, however, is the degree of multiplicity necessary for the recognition? On this point I would, even at the risk of undue prolixity, quote the results of an examination which I made nearly two years ago, and without any special object, except to illustrate the general pathology of the disease, of 61 cases, illustrating chiefly the pulmonary manifestations of what has usually been regarded as acute tuberculosis in the adult, all being above ten years of age, and only 2 below fifteen, except as ages not stated. In all but one the lungs were affected, the solitary exception being the combination of tubercular pleurisy with tubercular peritonitis. In 7 cases the data are uncertain; in these cases the lung was the sole organ. In 7 two organs alone were affected, in 16 cases three organs, in 12 four organs, in 9 five organs, in 6 six organs, and in 1 seven organs. Dr. Bastian lays stress on the meningeal affection, but in any shape where brain complications are recorded (though in all tubercular meningitis is not described in the current terminology of the present day), the cases amounted only to 28 of the whole number, though in 4 more it was probable but not certain. This is much below the proportion of cerebral affections in the whole class, my data being collected for another object; but they show, I think, that meningeal affection is not necessary to the recognition of acute tuberculosis as a disease—a fact which Dr. Bastian will, I am sure, admit. In respect to multiplicity, again, I would for a moment call the attention of the Society to the data existing for ordinary phthisis, when we know that multiple lesions are common. For a moment I would advert to the cases attended by myself: In acute tuberculosis (8 cases), in 2 cases the lungs alone; in 3 cases, two organs; in 2 cases, five organs; in 1 case, seven organs. In acute pneumonic phthisis (45 cases) there were 10 where the lungs, with or without the pleura and bronchial glands, were affected alone. In the others, reckoning the former collectively as one, there were in 11 cases, two organs; in 11 cases, three organs; in 5 cases, four organs; in 5 cases, five organs; in 2 cases, six organs; and in 1 case, seven organs affected. In 42 cases of chronic phthisis the lungs and pleura were affected alone in 4 cases; in 17 cases, two organs; in 9 cases, three organs; in 10 cases, four organs; in 1 case, five organs; and in 1 case, eight organs. Now, these represent the minimum, for the notes of the examination of all the viscera were not always perfectly recorded; and, in one or two, where the lungs are tabulated as alone affected, I find omissions of the larynx, and even once or twice of the intestines. Very similar data may be collected from other authors: thus Cless, in phthisis in the adult, found the disease limited to the lungs alone, in 35 out of 146 cases, or nearly 24 per cent. Dr. King Chambers, however, found this in 41 per cent. Age, however, exercises a great influence on this; Cless found the same limitation in only 3 out of 20 children, and Barthez and Rilliet in 23 out of 265, or less than 9 per cent. Tubercle in the child is multiple more commonly and to a greater degree than in the adult—a fact which may be explained in various ways, the most plausible hypothesis being, in my opinion, the greater irritability of the lymphatic tissues. I must, however, on these data demur to Dr. Bastian's opinion, that the existence of tubercle can only be affirmed by its multiplicity, or that tubercle cannot affect a single organ. In any organ I believe that the local manifestation may be acute enough to kill the patient without the secondary implication of other organs; and this is sometimes the case, particularly in acute pneumonic phthisis, where the inflammatory lesions largely predominate. How far the meninges can suffer absolutely alone is a point on which I can give no positive assertion without fuller research. Barthez and Rilliet record one such case, and Dr. Gee mentions one, where the minimum amount of cheesy matter found in each lung was the sole other lesion present. Again, as regards duration, I find in 55 cases of acute tuberculosis, from different authors analysed without any reference to this discussion, that 20 extended over more than two months. Empis gives a mean duration of thirty-nine days, the extremes varying from seven to sixty-five days, though Wunderlich records a case of but thirty hours. The possibility of a long duration of a disease characterised by the grey granulations when limited to the lungs was affirmed by Bayle, and numerous instances are given by other authors, one of them lasting nine months, to which I have myself seen a parallel, though here the peritoneum was also affected. I cannot for a moment doubt that a chronicity may attend this affection, while, indeed, in one case by Empis it was shown to lapse into the course of chronic phthisis. Nor is it only acute and fatal. There are a few but tolerably distinct cases of recovery, and some where, after partial recovery, both chief sets of changes which the granulations undergo have been found—in some induration, in others caseation. I admit to the fuller degree the peculiar course often assumed by this disease; it has been enough to cause everyone to classify it as a variety, but I gravely doubt whether it can be precisely defined in children from ordinary tuberculisation. Nor in the adult is it so widely separated from the course of acute pneumonic phthisis. I say this advisedly, since even in my own classification I found great difficulty in determining which cases in some instances to classify in either of these categories. Nor are the granulations in this disease always of the typical grey granulation form in all organs. I will not weary the Society with the other data from my own which I have collected on this point, but in nearly as large proportions as those already stated from my own observation, the yellow and caseous are recorded in the lungs by other observers; and I would therefore only state that these confirm my remark that the grey granulation is not the only, and in many cases not the most common of the granulations found in the lung of what, as a clinical disease must, I still venture to think, be regarded as acute tuberculosis. Dr. Bastian and the Society will, I hope, pardon me for not entering minutely into the discussion of the artificially produced disease in the rodentia. I could only repeat what I have already laid before the profession on the similarity of this disease to acute tuberculosis in man. I would only remark on three points—firstly, that however minute descriptions may differ as to some organs, the granulations in the peritoneum have such an identity of dissemination and structure that, coupled with the multiplicity of the disease, they are in themselves almost conclusive. I would also remark that this disease is not always one slowly evolved, creeping on in recognisable stages from organ to organ. In one of my cases death occurred in six days, with a minimum implication of four organs, and in two others in twenty-eight and twenty-nine days, with a minimum implication of four and five organs, in all ex-

clusive of local effects, or of the implication of the neighbouring lymphatic glands. Dr. Bastian and Dr. Crisp, however, state their objections to considering this disease tubercular on opposite grounds—Dr. Bastian that it is not acute enough, Dr. Crisp that it is too rapid for ordinary tuberculisation. Dr. Bastian appears to think that too much stress has been laid on the histological character of these growths. When I stated my belief in their tubercular nature, I put this, the last, as a question of proof. I asked "that it rested on a broader basis of analogy"—that is, rested on the general or constitutional affection. I stated, "it is not a question of the lung alone, or of the liver alone, or of the lymphatic glands, or the spleen, or the omentum, or the intestines considered simply. It is a question of general diseases producing in various organs growths which, if they occurred in man, would be considered tubercular," and that as no other disease was known producing similar results, I concluded, strange as it might seem, that they must be classed under this category. Into the many etiological questions connected with this subject I cannot enter, except presently to allude to one which has been raised in this discussion, how far indifferent caseation may be an origin of tubercle. To return for a moment to the question of the pathological identity of the grey granulation found in acute tuberculosis and ordinary phthisis, I can only assert my belief in it, and that, though acute tuberculosis differs in many of its manifestations, these differences are in many cases determined by the age of the patient, or by a rapid multiplicity of lesion. In other respects I think that the disease, etiologically and clinically, shows too great an approximation to ordinary phthisis to enable us to classify it separately. There is one other point to which I would allude. To confine the term tubercle to acute tuberculosis is almost to exclude tubercle from the diseases of adult life. It is so rare that even in a clinical hospital admitting a very large proportion of acute cases, one may wait months, I may almost say years, at times without meeting with a typical example, and it is to affirm that the grey granulation when multiple in the child is a different disease when less freely disseminated in the adult. In discussing the presence of the grey granulation in ordinary phthisis, I wish to state distinctly that though I do not regard it as the sole manifestation of the disease, yet it is its most characteristic feature, if we put aside for the moment the theory of its secondary origin from infection, and nearly every observer admits its almost constant occurrence. Thus Rindfleisch, who separates every other anatomical change from it, only found it absent in two cases of acute phthisis, and in a few of phthisis after measles; in relation to the last subject I would quote an older writer, Broussais, who, biased as he was by theories of inflammation, and who, as Dr. Williams has pointed out in his invaluable analysis of his experience in his work on Consumption, had the strongest personal grounds for opposing Laennec, yet sums up his experience in the following words:—"During three years of observation in this immense theatre (a military hospital) I have opened all the men sacrificed by phthisis, and I have only found one with an ulcer of the lungs without tubercle, and this was due to a foreign body. Tubercles, always tubercles! This is the most general and the most constant feature of resemblance." We may demur to Broussais' idea of what tubercle was, but he found something in the lungs of phthisical patients always present, and that something was different from what he found in ordinary inflammation. Allow me for a moment to ask your attention to the tables drawn up by myself of the different kinds of granulations found in the lung. I have omitted nothing that could be called phthisis, except two cases of old fibroid induration of uncertain origin, and one of acutely ulcerative bronchopneumonia. Now of the forty-five acute cases pneumonic infiltrations existed in forty-four, grey granulations in twenty-nine, and other forms of granulation (representing the softer forms found in acute tuberculosis) in fourteen, and visible pneumonia alone in two—("the acute general infiltration"). Now all these last sixteen but one had secondary affections of one or more organs of a nature ordinarily considered to be tubercular; a larger proportion than was found in those where the granulation was present, whilst a secondary affection was absent in five; but in two of the latter and one of the former the state of the larynx is not recorded. Of the forty-two chronic cases induration was present in twenty-nine, and was excessive in twenty; pneumonia was present in twenty-three; recent grey granulations in thirty-one. In eleven cases they were absent, the lesions being indurated and caseous or soft granulations, mingled in some with pneumonia; but in the cases where they were absent a secondary affection existed in all but two, where the appearances were doubtful. They were absent also in two of the cases where grey granulations were present; so that here again, as far as these numbers go, the multiple lesions were scarcely less common in the absence of recent grey granulations than in their presence. Here, therefore, in the absence of the distinct grey granulations, we have strong evidence of a multiple or constitutional affection. Then arises the question, can we not recognise tubercle except as the grey granulation? To say that we cannot is to deny its metamorphosis on one side into fibrous tissue, and on the other into caseous change. I believe that no one will deny either of these, and in some cases of phthisis the production of recent grey granulations shortly before death is a matter of accident. What we have to look to in this class is the probable pathenogenesis, the origin of indurated and caseous granulations; and, knowing the tendencies of tubercular growth in both these directions, we may, I think, conclude that they are tubercular. In fact we know the former as obsolescent tubercle; are we, when we meet with indurations, to designate the fibroid change and call them chronic lobular pneumonia? More diffused indications may, as I stated before, arise sometimes from tubercular growths, sometimes from mere chronic pneumonia; but the latter is different from the former, and I still believe that it is rare except in the presence of granulations of a tubercular character. I would also state that in those where the grey granulation was absent I made in nearly all an examination of the other forms of granulation present, and in all that I did so examine (though I cannot quote the exact proportion of these) I found the same growth that occurs in the grey granulation and in the softer forms of granulation in acute tuberculosis. This is my ground for the statement that in all the cases of phthisis which I have examined I have found, both in granulations and in the diffused form, growths identical in character with those found in acute tuberculosis, and that in the vast majority of cases there was multiple disease affecting other organs. Is this multiple disease different from the multiple disease in acute tuberculosis? It is scarcely so in its multiplicity numerically considered. It is not so as far as the lesions go in the different organs affected. Here, however, we are met with the question of infection, but on this I must dwell briefly. It has been known since the days of Laennec that tubercle tends to multiply, but the question at the present time is, can it be produced in the human subject by indifferent caseous products, or by any inflammatory change not associated with a peculiar liability of constitution? I strongly doubt both. I have already alluded to the caseation which ordinary inflammatory products undergo this change except under special circumstances. The evidence of caseous glands serving as the sources of this infection is, to my mind, after reading Schüppel's observations on the nature of these glands, only evidence of a secondary infection from a primary tubercular change. You may get a tuberculous gland secondary to any common irritation, in a predisposed subject; that is, a carious tooth or a cutaneous disease of the head may give rise to a change in the nearest gland of a tuberculous nature. And I believe that what we thus see externally takes place in the lung: any irritation of the tissue may, in the presence of local or constitutional predisposition, give rise to secondary growths, diffused or circumscribed, which constitute tubercle, and which may be the source of further infection, and that with or without antecedent caseation, though this stage and that of softening are most favourable to the change. I have no wish to ignore the evidence of other non-tuberculous caseous changes acting in a similar way, but then the granulations radiate from this as a focus, and the question is, will they arise without this predisposition? Such cases are, however, few, and in the majority of those where secondary infection is reported, the primary change is tuberculous in its nature, and of this I have seen a marked instance where the bronchial glands became caseous secondarily to an empyema, and acute miliary tuberculosis occurred in the opposite lung. As

regards the caseous changes in the lung which are supposed by many to be the source of the infection, I have attempted to show that these are not a simple inspissation of pus or retained secretion, but a death of tissue due to a particular growth. I do not call the caseous matter tubercle in these diffused areas, much of it is pneumonic, but it is pneumonia running a particular course in the presence of tubercle, and I think it open to the gravest doubt whether it is the caseous matter, as such, or the growth which is the source of the further infection. I expressed this opinion in relation to the rodentia, and it has been more fully expanded by Dr. Sanderson, with whose views in this respect I entirely concur. I would say one word about the often repeated statement that these caseous nodules are often mere accumulations in the smaller bronchi. I have fruitlessly, in earlier days, when I believed this, spent much time in the dissection of bronchi to come upon them; I have tazed the ingenuity of instrument makers for knives and scissors to penetrate to the finest ramifications, but I have not been able to find such conditions in the sense in which they are spoken of as a gradual inspissation of tenacious mucus forming the first stage of this process. A caseous nodule of tubercle surrounded by induration, presents the greatest resemblance to a bronchus; and when it is softened in the centre, a bristle can be passed into the branches, because they necessarily communicate. This appearance, which I also used to describe in the terms often employed, is not in the majority of cases a mere inspissation in a bronchus. It is an area occupied to a greater or less extent by a tuberculous growth, and often including smaller bronchi, but it is not in my opinion a mere inspissation in the interior of these. I had intended to dwell on these points and give some further illustrations when I introduced the subject, but I had to pass it briefly by, and can only give this further explanation now. Inspissations do occasionally occur in large tubercular bronchi—bronchi with a tuberculous growth in their walls; but the majority of caseous nodules found in the lung are not, I believe, due to this cause, but to changes in the lung-tissue. I must demur also to the opinion that this caseation is due to mere pressure. The most intense exudation of acute pneumonia does not produce it, nor does any other pressure with which I am acquainted. I must turn now to another difficult point, perhaps the most difficult in this question—the relation of tubercle to inflammation. But I have little to add to what I have stated as my belief. The question of the origin of the disease in a lymphatic gland represents, briefly stated, to my apprehension, the origin of the majority of cases of tubercle found in the lung. That tubercle may arise from blood changes I have no doubt, but at any rate my belief is, that it is a lymphatic growth, excited by abnormal local or constitutional conditions, or probably, in the great majority of cases, by both combined. I cannot admit on such evidence as we possess that, although these modes of origin are apparently different, the disease itself is essentially so. I would ask whether the disease excited in the lymphatic gland by a carious tooth, in the child of a phthisical parent, is essentially different from the tubercular meningitis in another child of the same parent, arising without apparent exciting cause? For my own part I cannot think it so, and if later in life another member of the same family becomes phthisical after a pneumonia or a catarrh, I confess that I see in all these the manifestations of the same disease. Unless chronic pneumonia in the lung, of which I know almost nothing apart from tubercle, is different from chronic inflammations elsewhere, I would assert that these changes, though with great affinities to a chronic inflammation, have a peculiar but not specific stamp of their own. So great is the affinity that everything which has been known as tubercle has been called simply inflammatory. Broussais did so with a qualification; Cruveilhier, Gendrin, and others have done so with various modifications. Reinhart affirmed it of all forms, and said that even the grey granulation was only an induration of grey pneumonia, and that the so-called tubercles of other organs were only multiple disseminated inflammations. Andral affirmed it also for the grey granulation in the lungs and peritoneum. Empis makes the same assertion, both these authors separating it from what they call tubercle or caseous masses. The modern German school, agreeing with the late Dr. Addison, precisely reverse these opinions, so that what one set of observers call tubercle another set assert to be inflammatory, and what the latter call inflammatory the former call tubercle. Is there no way out of this confusion? I believe that there is but one, except that proposed by Dr. Bastian, to which I shall presently allude. I believe that it is to recognise tubercle as the result of irritation of a particular set of tissues under certain constitutional conditions. We cannot accurately define all the peculiarities of these conditions. This is wanting to our definition of the disease, but on the anatomical side the growths have, I think, characteristic features. The tendency to assume the round circumscribed form is a general feature of these tissues: it is the type of one of their normal physiological developments, and it recurs under pathological conditions; but the diffused form is nearly equally constant though not equally characteristic, and this both physiologically and pathologically. And as we admit their identity in the former case, we must also, I believe, in the latter. We have analogies enough in other constitutional diseases, as Professor Buhl has pointed out. We have a diffused and a circumscribed series of growths in leucæmia. We have the same in syphilis; we have the same even in common suppuration; we have the same in lympho-sarcoma, and even in cancer. It may be said that this is only to return to the definition of Broussais, that tubercle is only an expression of inflammation of the lymphatic tissues; but what Broussais affirmed without the knowledge of these tissues which we now possess, and while he used the term tubercle in a different sense to that in which we apply it, the proposition is proved for a large series. The only question is, does analogy justify its extension to the remainder? I admit that we have not precise proof of its origin in the diffused growths; this yet awaits anatomical elucidation; but I think that we have strong grounds for this belief. This question has been already fully dwelt upon by Dr. Sanderson as well as by myself, and my views, I believe, correspond on this point with his. It is not any inflammation of a lymphatic. No one would call a suppurating bubo tubercle. It is the results of irritation in certain constitutional states which give to the growth its peculiar characters. To my apprehension this idea is not productive of confusion, but the reverse; but to take the alternative, if I may again quote Dr. Bastian—who dreads the chaos which this prospect seems to open to his apprehension,—I would ask, is any chaos greater than the present? We are in doubt, at least after his statement, if we have one disease or many included under those at present classified as presenting the one common anatomical and tolerably distinct feature, the grey granulation. Nay, even I think that on his premises it might fairly be disputed if the one disease we know as acute tuberculosis ought at least to be divided. On his showing no one can say certainly, or feel sure, what the different granulations in the lung in ordinary phthisis signify—whether, even when grey granulations are met with, they are in all cases the same disease, or whether what we have all been calling tubercular inflammations of serous membranes have any pathological affinities to each other or to any of those to which I have alluded. Dr. Bastian proposes to start afresh in the inquiry, by doing away with the word tubercle altogether. The idea is not a new one to me. I persistently adopted it in my own notes for some three or four years, and I still commonly do so in my descriptions of lungs. But I think this procedure hardly necessary, nor even then can we start easily with any common terms. I think we could hardly, even as far as I have ventured to comment on Dr. Bastian's views, define the acute disease "granulia" as one. There are a variety of different appearances in the granulations present which are characteristic. In some cases, or even tissues, one is more common than the other. What degree of multiple affection, or what length of duration, or what combination of symptoms is necessary to constitute it? I have often pitied the "intelligent student," to whom we have appealed as our test of the definitions of our views, fresh from the reading of Niemeyer's text-book, turns to find out in the dead house what is tubercle. I should, I think, pity him still more if I have to tell him, when he asks what Laennec and Louis, Rokitansky and Virchow, Stokes and Williams and Walshe meant by tubercle, that there is no *tubercle*. "Nous avons changé tout cela." And yet still more when he is told that identically the same appearances to the naked eye and to the microscope may mean different

diseases; that there is no pathological affinity between an acute granulie with granulations of varying appearances in the same and different organs, and a granular pneumonia with similar granulations, and with caseous and fibroid products, and a caseous pneumonia with the same granulations and fibroid changes, both associated with caseous or ulcerative or granular changes in the serous membranes, larynx, intestines, liver, and genito-urinary organs; and a granular peritonitis resembling some forms of the complications of the former, yet not the same disease, but passing by insensible gradations into cancer. I believe that if this plan were adopted the first effect would be in the attempt (perhaps *mutato nomine*) to re-establish the unity of most of these affections. For my own part, though I did for the purpose of inquiry cast aside the name, I do not think that we can do so in our literature and description without such a cataclysm of our pathological ideas that I for one cannot advocate it. I do believe that between these diseases there is such a close etiological, pathological, and clinical connexion as to demand at least a terminology implying in some degree their association in a common category. I think it better to express this association, as to my mind it is really expressed, by the word "tubercle" (in use during three centuries), notwithstanding the doubts and obscurity which have hung over it, rather than to seek a new term or set of terms about which for a generation to come there will be even, I think, more disagreement. We may express the phenomena of tuberculisation in the terminology of any current theory. We may call it a neoplasm, an exudation, an inflammation, a deposit, what we will; but it is a *disease*, and in the vent of our pathological ideas it will want a name. To my own ideas its formations are most nearly allied to, though not identical with, the phenomena of inflammation; but you want a term to distinguish it, as I believe it is distinguished, from most common inflammations, and that not in the lung alone, but in other organs. The disease is, I believe, most easily recognised by the presence of recent grey granulations, but I do not believe that it can be defined as limited in its pathological effects solely to this special form. It is distinguished by vital characteristics, by a growth destructive of vessels, and by a consequent tendency to early necrosis, though capable in some cases of more or less permanent development. I have ventured on some remarks adverse to a demand for definitions of anatomical change specific for each disease, which I believe would render all pathological classification, if carried to its full extent, impossible. Imperfectly defined words are the *eidola fori* of science; but too limited definitions, excluding phenomena of identical characters, are not less so; and I believe that we exclude a large part of the phenomena of a tubercular series of formations if we limit the use of the term to the grey miliary formation. We are in this case in no worse position than in almost every other disease—our definitions of disease are only abridged descriptions. There is scarcely one extant with which I am acquainted that precisely excludes the phenomena of other diseases, but they are sufficient for recognition. I have only attempted to give such an analysis of the growths in phthisis as may answer the former purpose. My own position, setting aside the use of a name, was a simple one. I started with the inquiry whether I could find in the diseases classified under the name of phthisis (with the exceptions before alluded to) such differences of anatomical structure as would, in my opinion, justify me in establishing these as the basis of clinical investigation. The result of this research has appeared to me to be negative. I find in all one common series of growths, conducing on one side to destruction, on the other to induration of lung, and I find these corresponding to similar formations in acute tuberculosis; both also forming common phenomena of a disease tending to multiplicity, the manifestations of which, in different organs, present similar characters. If, therefore, many different diseases are included under the name of phthisis, their anatomical classification has (if my observations are correct) yet to be sought for. This position involves, after all, but a slight modification of that previously existing. It is that similar growths occurring simultaneously in the same organ are probably of identical nature, and is in accordance with the aphorism of Virchow, before quoted. No one can recognise more fully than I do the variations in the clinical features and anatomical characteristics of these diseases, but they are all mutually interchangeable, and pass by indistinguishable gradations into one another. Some of these may perhaps be well expressed in different terms. The expressions caseous pneumonia and fibroid phthisis are unobjectionable as expressing certain appearances; but I object to the term scrofulous pneumonia, if this signifies mere caseation without the intervention of a morbid growth, for without this it scarcely exists in the lung—or if it signifies a disease having a different anatomical basis to fibroid phthisis, or if it may not at one time or another pass into the other without changing their essential nature. Both diseases are attended by the same morbid growths, but evolving differently under accidental conditions—in the one into caseation and softening, in the other into fibroid induration. The only objection to either of the former terms consists in the fact that they leave unnoticed the granulations which are almost invariably present; but their close relationship is, I believe, a point never to be forgotten. Among the drawings which I exhibited were one of typically fibroid and another of typically pneumonic form of phthisis, occurring in two sisters, daughters of a phthisical father, who both died in hospital. The one (the younger) had been under my care for years, the other died after a few months' illness. I have seen a very similar contrast and association in two brothers. Neither the whole of the fibroid change nor the whole of the caseous matter is of distinctly tuberculous nature—that is to say, neither in the circumstances directly arises from a tubercular deposit, but in the vast majority of cases both are associated with it. Both simple ulceration and simple fibroid may occur in the pneumonic portions without the apparent ulceration of these growths, but they are, if present, especially the former, of the extremest rarity, except in their presence in other portions of the lung. It is, however, to these parts that a great part of the destructive changes, and a part, but a varying part, of the indurations in the lung, are due. The purely inflammatory changes may vary in extent, and the fibroid may also; but these growths are almost invariably present, and until some further distinctions are established I have felt that I could only regard these growths as of the same nature. Being only to any true extent simulated, as I believe, by the growths in typical tubercle of the child, I venture still to call them tubercular. In this I have no wish to be dogmatic; and, if I have maintained my own reasons, it has been after a full consideration of those of others. There are subjects in medicine which would, I think, so dispel a spirit of dogmatism and exclusive adherence to one's own opinions as the study of the history of phthisis, on which such differences have existed and do exist, among the greatest men of the past and of the present, as may well make any one doubt the value of his own observations, and the accuracy of his conclusions; and much as I felt the honour done me by the request that I should open this discussion, I shrank from it personally, lest what I believe to be the truth should suffer from the imperfect exposition which I should be able to give. I do not regret, and I hope the Society will not regret, that this discussion has taken place. It has, at any rate, elucidated new views of the greatest importance, and though I have ventured to dissent from some of them, their expression will, I believe, stimulate to further inquiry, which, in this Society, cannot fail to lead to the discovery of fresh truth. As regards those which I have stated, I wish to be distinctly understood as disclaiming all priority. I have endeavoured to work out this question as a whole, without any desire for early publication, and much of what I have stated as my opinions have been, in some form or another, expressed by others; but the varying shades of opinion have prevented me from going into the details of these views, or referring to their work in the terms which that work deserved.

THE governors of the Bradford Infirmary held a special meeting last week to elect two members to the hon. medical staff, in the places of Dr. Nicol and Mr. Broughton, resigned. Dr. A. W. Smith, the only applicant, was elected hon. physician. There being no applicant for the post of hon. surgeon, a resolution was carried, leaving it in the hands of the Board of Management to call a meeting of the governors when they might deem it necessary for the purpose. There was some discussion, in which the desirability of an alteration in the rules for elections to the staff was hinted.

Rindfleisch et Virchow.
discussion sur la Tuberculose.

in Berliner Centralblatt.
1873. p. 518 et 519.

Rindfleisch partisan
de l'unicité; analogie avec
les travaux de Thaon et Grancher.

Lebert.

Ueber cultur des Krupes.

im Deutsch. archiv.

12. Bd. p. 42.

1873.

Mr. ARNOTT said there was one point in the pathology about which he should like to speak—viz., the condition of the spinal cord. He had had an opportunity of examining one leprous patient, who had come from the West Indies and died in Middlesex Hospital. Danielssen in his account of leprosy in Norway, speaks always of the spinal cord being diseased, a varicose appearance from enlargements. From the description, this appeared due to a deposition of the same material which Dr. Carter calls hyalin-fibroid. He found the same change in the cord he examined; it was then two or three days after death, and might have been due to post-mortem change. Dr. Carter and all Indian writers saw no change, so perhaps the forms were not identical, and the changes observed in the cord in Norway may be due to the use of fish and fish oil.

Dr. BUZZARD was sorry the author of the paper was not present, as he should have liked to ask him one question. Dr. Carter said the sensory rather than the motor nerve-fibres were affected, and that the nerves were more affected when they emerged from the fascia and became cutaneous than when more deeply seated. Might not this be explained by the fact that the motor nerves—to the muscles—were given off more deeply, and only the last part of the nerve was cutaneous? He brought before the Clinical Society three years ago a case in which the question of heredity was raised. The father and three other members of the family were mutilated by the disease. He thought Dr. Carter was right in saying that the nerve lesion and not the cutaneous affection was primary. It was well known that cutaneous affections followed injuries to nerves, as was exemplified in Paget's case of injury to the median nerve. This was not so if the nerve was only compressed, but perhaps, there was something like a neuritis in leprosy, an inflammatory condition of the nerve producing these changes.

A CASE OF AMPUTATION AT THE HIP.

BY RICHARD BARWELL, F.R.C.S.E.,
SURGEON TO CHARING-CROSS HOSPITAL.

Caroline L——, aged seven, was admitted into Charing-cross Hospital under Mr. Barwell's care September, 1872. She had previously been under the care of Mr. Hancock with severe hip disease, and that surgeon had in the early part of 1871 excised the head of the bone, but during the operation the thigh, a mere shell of bone, had broken in two places. She went out after some months with bony union, but with open sinuses. When readmitted at the above date she was emaciated and feeble; there were several open sinuses; the liver was much enlarged. After watching the case for some time, the operation was decided on, and performed by Mr. Barwell on the 2nd November. Hardly any blood was lost; the limb was almost devoid of muscles, the bone carious and inflamed throughout. The child rallied, and after a time (corresponding with the occurrence of smart diarrhœa) the liver began to diminish in size. On the 1st of February the child went out with the liver much smaller. Certain deductions concerning the states of liver in different phases of disease were given.

Mr. THOMAS SMITH said he should have been inclined to refrain from operating with such a condition of liver. The decrease in the size of the liver after the operation was interesting. He had had a patient in the Children's Hospital who had both the liver and the spleen greatly enlarged, so as almost to fill the whole abdominal cavity. Whilst suppuration was going on the liver became smaller, and returned to nearly its normal size. The spleen, however, remained as large as before. It had been proposed to treat the patient with potash salts, according to the views of Dr. Dickinson of the pathology of this enlargement in suppuration, as arising from dealkalinity of the blood. This, however, was not done, but the liver spontaneously decreased. There was said to be no deposit, only the liver, not going through its nutritive changes as usual, became full of cells in various stages of growth and degeneration; and the size of the liver was due to the retention of these. He would like to ask Mr. Barwell if he has ever held the abdominal aorta when thus operating.

Mr. ARNOTT said he was about to ask a similar question, as he had been struck with the small quantity of blood lost, about one ounce. He had seen the aorta compressed by Lister's abdominal tourniquet, and also by the hand, and little blood was lost; but he would like to know how the hæmorrhage was arrested in this case.

Dr. AWDEN said he had been struck with the remark that the enlargement of the liver in these cases was due to a heaping up of cells. He always thought it presented amyloid characteristics, both as seen under the microscope and by the use of reagents. He should think that when the liver had been enlarged and then decreased, it was due to fatty rather than amyloid degeneration. He could say nothing as to the question of the dealkalinity of the blood. He knew that very little was to be done for the condition.

Mr. CALLENDER said that as the compression of the abdominal aorta had been alluded to, he would like to make a remark as to the mode of its application. The abdominal tourniquet was insufficient for the purpose, for it compressed the vena cava as well as the aorta. This was obviated by placing a narrow pad of lint directed from the right to the left, and then no pressure came upon the vein. If the vena cava was compressed there was great hæmorrhage from the large veins.

Mr. BARWELL, in reply, said that certainly, if operating on an individual with a large and well-nourished limb, he would have the abdominal aorta held. In this case the limb was emaciated, and so Mr. Bellamy passed in his hand under the flap, and at the same time compressed the aorta; only a little venous blood escaped, not so much as two ounces. As to the enlargement of the liver, he thought it was due, as Dr. Anstie said, to fatty degeneration, and that need not preclude any operation. If it depended on amyloid degeneration he should not like to operate. When bones were inflamed and carious, the liver was more likely to be fatty; if largely necrosed, most commonly there was amyloid degeneration.

ANATOMICAL RELATIONS OF PULMONARY PHTHISIS TO TUBERCLE.

THE important discussion on Dr. Wilson Fox's paper "On the Anatomical Relation of Pulmonary Phthisis to Tubercle" was commenced on Tuesday evening last at the Pathological Society.

Dr. MOXON, after expressing the diffidence he felt in following such a speaker as Dr. Wilson FOX, said he was the less anxious to express his views since for the most part he concurred with what Dr. Fox had said so gracefully, forcibly, and pointedly. He would be sorry to see those views weakened by pointing out any divergence in his views. Since he had come almost to the same conclusion, he would be pleased rather to hear what others said who held opposite views. His own views on the subject had been formed for some years, and he had expressed them on previous occasions to the Society. But as the discussion demanded it, he would take the liberty of stating where Dr. Fox's views fell short of the point. Dr. Fox's propositions might, he thought, be summed up thus:—1. That miliary tuberculosis of the lung has neither the anatomical nor histological constancy or peculiarity commonly ascribed to it. 2. That miliary tuberculosis, in short, exhibits all the products found in active chronic phthisis. 3. That all the other products constituting caseous pneumonia under various forms are essentially of the same histological structure which Dr. Fox thinks is characteristic. 4. That the other characters of chronic phthisis are fairly traceable to the effects of time. 5. Due attention has not been given to the developmental changes that occur in tubercle. In short, he did away with the peculiar nature of miliary tubercle by affirming its essential substance to exist in all caseous phthisis. He reasserts the identity of all phthisis as based on this peculiar microscopic matter, and, finally, he excludes common catarrhal pneumonia entirely from phthisis. Shortly, this would be the difference between Dr. Fox's views and his own, that he did not hold with Dr. Fox that the basis of the definition of phthisis should rest on purely histological grounds. He was sorry to see the word "adenoid" brought forward in the discussion: it was a dangerous word; the term was bigger than the meaning, it seemed to mean much, but yet conveyed very little. What did it mean? Shortly, it meant resembling lymphatic gland structure. Did Dr. Fox then say that tubercle resembled the structure of lymphatic glands?

It was only the remotest resemblance, and was far from being identical. There were many other adenoid substances, as of leukhæmia, syphilis, &c., and those described first by Dr. Sanderson as adenoid tissue around the nerves in Addison's disease. The word was vague. What was adenoid in tubercle? All he could find was a number of cells, but they were nothing like lymphatic cells, nor was the intercellular substance. He would have liked to show drawings of adenoid tissue seen in a blood-clot; this appearance was always met with if there were enough white corpuscles. What he meant was this: though adenoid, it was nothing particular, and he would not like to rest the unity of phthisis on such a basis. It was an easy thing to attack the position of others, but he would rather add something positive to the matter which had been brought before the Society. A child was injured by a cab, and brought to Guy's Hospital and died; he examined the body. The medical aspect was interesting. It had had severe bronchitis (at least he assumed that), and there was caseous enlargement of the lymphatic glands about the trachea; the mucous membrane was pulpy. The lung near the glands was stuffed with miliary tubercle, concentrically arranged around the glands; so the glands were the foci of the disease. Remembering the inoculation experiments, one would say there were three changes—1, chronic catarrh of the trachea; 2, caseation of the glands; 3, tubercle of the lung. Now no one would say the catarrh was specific, but that the tubercle was; yet caseous degeneration through inoculation can produce the same kind of disease, so that from non-specific to specific a series, where does specificity arise? Looking from inflammation to tubercle one saw in tubercle only a branch of inflammation, somewhat similar to what Dr. Sanderson had proved by his paper before the Society last year. Why should tubercle be called specific more than pyæmia? There was nothing specific in tubercle; if inflammation was set up in a person he might be said to be on the high road to tubercle. Since we can define its cause it is not specific. If tubercle is a development of inflammation, how near are we to Niemeyer's views. He certainly says something like this. But he (Dr. Moxon) would oppose it through thick and thin. Niemeyer, taking up Buhl's theory—the possibility of auto-infection by caseous matter,—at once says it occurs through this particular disease. Hence the weakness of the views. He believed it followed caseation, but would not say from catarrhal pneumonia. He had never seen a case of incipient phthisis without tubercle, and the earlier the stage the more the tubercle. As to the origin of phthisis, he had little to say; he had his theories. He would like to say a few words about the so-called fibroid phthisis. He did not know if any of its asserters were present, but he must express his conviction that it was a very unfortunate idea of theirs as far as pathology was at all concerned in it. It was clear and plain, from the very defining descriptions themselves, that fibroid phthisis was nothing more than old phthisis, in which a good deal of fibre was necessarily present. Those who erect it into a distinct kind entirely disregard the fact that tubercle is short-lived. No tubercle lived three months, so that in five or six years—and it is naïvely given as a property of fibroid phthisis that it lasts such a time—generations after generations of tubercles must have come and gone, doing their natural work in destroying—first their matrix and then themselves, and leaving behind them the scar of fibre in which the nature of our bodies always enshrouded the traces of such mischief. But the author of fibroid phthisis, seeing the fibrous accumulation around these ancient remains, can think of nothing but their present state, and like a translator who has only one tense, renders the preterpluperfect, perfect, past, and present all alike by his one present tense, just as if one called an old united fracture an osteoid broken leg, or spoke of scars on the skin as fibroid lupus.

Dr. CAYLEY said that while no description of the changes which are met with in phthisis could be more complete and accurate than that which had been given by Dr. Wilson Fox, he thought that the doctrines founded on this description were open to some exception. It was well known that the so-called adenoid tissue, which Dr. Fox appeared to regard as the most characteristic element of tubercle and to be the result of an irritative overgrowth of pre-existing lymphatic elements, might be produced in parts of the body by almost any kind of irritation. It occurred, for instance, in the margin of a hard chancre, in a soft node, in early stages of cirrhosis of the liver, and in the lung it might be produced in the interstitial tissues and the walls of the air-cells by a great variety of causes, as the presence of a foreign body, the inhalation of irritating dust as in grinders' phthisis, chronic pneumonia, or pleurisy. If, therefore, adenoid tissue be distinctive of tubercle, tubercle cannot be distinguished. Many of the more recent writers on this subject give a very different interpretation of the structure of a grey granulation—Rindfleisch, Wahlberg (in his recent paper on tubercle of the larynx), Schüppel, &c., who all describe tubercle as made up in great part of large cells. Professor Schüppel, as is well known, regards the multinucleated giant cell as the most essential element of tubercle, and describes how it gives off processes which form the reticulum, in the meshes of which the other cells lie; and he considers the adenoid tissue which surrounds and is mixed up with this structure to be the product of simple irritation. Dr. Cayley exhibited specimens showing this structure. Dr. Fox, however, regarded these larger cells as of secondary importance, because they were frequently absent from tubercle of the lung. This might be explained by the fact that in so-called tuberculosis of the lungs true tubercles are often not present at all. Dr. Cayley thought that the general scope of Dr. Fox's argument was this:—Finding in the lungs of children affected with acute tuberculosis, in addition to the grey granulation, a great variety of other changes, which he has so fully described, he assumed that this must also be tubercular, because they are found associated with tubercle, and then finding in the lungs of adults affected with chronic phthisis these other changes to be predominant, while the grey granulation is often absent, he still regarded them as tubercular—an argument by no means necessarily valid. Dr. Cayley therefore thought that, though the question was not yet ripe for a final decision, we were still entitled to maintain, at any rate provisionally, and as affording at present a better explanation of the facts, that the term tubercle ought to be restricted to the grey granulation, which does not merely consist of a mass of adenoid tissue; that in many cases of chronic phthisis no tubercles are present at all, and in many other cases, when present, they are secondary to the pre-existing inflammatory processes, and are very likely produced in a manner in which they may readily be produced in the lower animals, by caseous infection.

Dr. BEALE.—In the main I agree with the account given by Dr. Wilson Fox, but I am sorry to say that I differ from him in one or two important particulars. With regard to the nature of the tubercle-corpuscle, I cannot think with Dr. Fox that it has anything to do with the lymph-corpuscle, or with the cells of adenoid tissue. Neither do I think that it is even proved that the walls of the air-cells of the lung are covered with an abundant plexus of lymphatic vessels, as I understood Dr. Fox to admit. I have never myself seen any appearance which would justify such a conclusion, although there can be no doubt that lymphatics are exceedingly abundant under the pleura. Neither do I understand how any process that may be denominated irritation can convert one of these lymphatic corpuscles into a tubercle corpuscle. It seems to me that the term irritation is rather too freely used. I am afraid there are very few here who if called upon could give a definition of what they mean by the process of irritation, and if we were to discuss the view of Virchow, I am sure every one would come to the conclusion that Virchow's doctrine of irritation could not be substantiated in any way whatever, for it almost leads to the ridiculous supposition that minute particles of protoplasm, or bioplasm, or whatever it may be called, have the power of sympathising with other particles. The term irritation may almost be looked upon as the Abracadabra of pathology. I hope that before long it may be ranged with such terms as vacuolation, fibrillation, differentiation, and many others, the use of which during the last few years has been abandoned, or at least less met with in pathological literature than was formerly the case. From an observer's point of view the question—What is tubercle?—is undoubtedly a difficult one to answer. It seems to me that tubercle, like much other matter in the body that has received a definite name, consists of living matter. There is no doubt that tubercle, like pus and cancer, possesses the power of growth and multiplication. But there is a great difference in the living matter of tubercle, and that of a

white blood-corpuscle, of a cancer cell, or of any other cell in the body. These different forms of living matter could not be distinguished under the microscope, or by any means yet known. We must give up the hope of ever finding any characteristics which shall enable us definitely to say that a particular object is a tubercle-corpuscle, cancer-cell, or any definite form of living matter. We are not therefore compelled to assume that these are identical. They all have great differences which remarkably, although they may not be open to observation or to be demonstrated by chemical tests. The life of all these particles of living matter is very different. The pus-corpuscle grows and multiplies at a rate very different from that at which the tubercle-corpuscle grows and multiplies, or a cancer-cell grows and multiplies. The pus-corpuscle lives faster than a tubercle, and the tubercle-corpuscle lives faster than the cancer-cell. The tubercle-corpuscle also differs with regard to the materials furnished by its death. The substances resulting from the death of the cancer-cell are different from those which result from the death of a pus or tubercle corpuscle. So that although these different kinds of matter are all living and all multiplying, and agree in many common characteristics, they have important differences. I think we should no more be expected to distinguish the living matter of tubercle from the living matter of pus or cancer than the cells composing the embryo of a chick from those composing the embryo of a dog, which, though alike under the microscope, have properties that are anything but identical. It seems to me that although it may be impossible for anyone to say that this or that is a tubercle-corpuscle, we must assent to the doctrine that a tubercle-corpuscle is a special thing; and I agree with Dr. Fox, that underlying many of these changes are tubercle-corpuscles, although in many of these cases the characters of the tubercle-corpuscles themselves may differ. In an ordinary tubercle we have of course many different structures—products of inflammation, of changes in adjacent tissues, and so on. It would be as impossible to distinguish any one of these component masses of different forms of living matter as to distinguish the corpuscles of the embryo of one animal from those of that of another if they were mixed together. Under different circumstances, no doubt, the tubercle-corpuscle will give rise to a differently formed material, fibrous, soft, glistening, or a matter which may at last degenerate into that which we know as caseous substance.

Dr. CHARLTON BASTIAN said: The question which has been so ably discussed by Dr. Wilson Fox is one of so important a nature, and at the same time one concerning which so much difference of opinion has always existed, that it would seem most useful for the different speakers in the debate to set forth as concisely as possible the views which they themselves entertain upon the general question, without entering too much into matters of mere detail. I shall therefore follow the example of Dr. Wilson Fox, and endeavour, as briefly as the complexity of the subject will allow, to set forth the nature of my views concerning the relation of "tubercle" to pulmonary phthisis, with no more reference to the opinions of others than is necessary for my own exposition. And if these views are found to differ, in some respects, very much from those which Dr. Fox has expressed, I feel sure that he will, nevertheless, be one of the first to recognise the desirability of looking at the question from all sides. I may, however, state at the commencement, that any discordancies which may appear between my own opinions and those of others will not be so much differences with regard to facts, as differences concerning the interpretation and mutual relation of facts about the reality of which we are all more or less agreed. It seems to me, in the first place, quite impossible adequately to consider the question of the nature of tubercle, and its connexion with pulmonary phthisis, wholly without reference to the relations of such a product to chronic inflammatory changes, to scrofulous changes, to the new growths known as lymphomata, and to those met with in leucocythæmia, and other morbid conditions. It appears to me also highly inexpedient to consider the lung affection alone, and wholly apart from the light which may be thrown upon the question before us by a brief consideration of parallel morbid changes in other organs. It is the treating of the question in this isolated manner which, as it seems to me, alone makes it possible to consider as legitimate, or expedient, views which may tend to introduce much needless confusion into pathological science. Taking up the involved question as to the nature of tubercle at that stage in its history to which it had been brought by the labours of Addison, Reinhardt, Virchow, and others who had preceded them, we find that it presents itself somewhat in this fashion. The notion that caseation, or cheesy degeneration, constituted the essence of tubercle, was thrown aside as one which a larger and more minute experience could in no way sanction. The characteristics relied upon by Laennec and others had been shown to be almost wholly valueless as characteristics of any special and peculiar something which it had become the fashion to name tubercle. The masses and nodules which were shown to be in the main new products of chronic inflammation in different tissues and organs. And inasmuch as such products, both in their incipient and in their advanced stages, were specially abundant in the lungs of persons dying from pulmonary phthisis, this affection became one of which tubercle could no longer be considered as the same could be applied? Only one product, which Laennec had been in the habit of regarding as an early condition of a deposit destined subsequently to undergo the caseation supposed to be characteristic of tubercle. It is true that Laennec's central idea had been shown to be erroneous; it may be true also that the body upon which the name of tubercle was now to fall had originally been described by Bayle as a non-tubercular substance.* This product was the "grey granulation," a body now known to have a constant and invariable structure, in the main resembling that of lymphatic gland tissue. It was, and is, the anatomical mark of an obscure general affection, having no necessary connexion with phthisis in the strict sense of the term—that is, as an ulcerative lung disease; it was a product prone to occur in more or less abundance within the cranium, within the chest, and within the abdomen of persons suffering from what we now term acute tuberculosis. But if this obscure and protean febrile affection, in which grey granulations are found disseminated through the organs, has no necessary relation to phthisis, on what pretence can it be called by its present name, and what possible reason could be assigned for giving to the grey granulation the mere name of tubercle, when the essential meaning of the term—that of a caseating product productive of phthisis had previously been so different? To which questions it seems only possible to return the following replies:—In the first place, we must assume a deeply-rooted reluctance wholly to cast aside the word "tubercle," a word which, up to this period, had borne such an important connexion in men's minds with phthisis—although phthisis had now been fully shown to be a non-tubercular affection in the past sense of that term. And, secondly, because there was a very flimsy justification for such a course. Although this obscure febrile affection, acute tuberculosis, is one which may occur quite independently of any traces of phthisis, still it has a special aptitude also to occur as an intercurrent affection in the course of chronic phthisis. Grey granulations are therefore to be met with at times in the lungs of those suffering from phthisis. For this reason, and because they were bodies possessing the knotty or granular characters which were originally implied by the term tubercle—because, in addition, they everywhere possessed a constant structure, Virchow and others were content to let the name "tubercle" rest with them, although it must have been fully recognised at the time that such bodies had no necessary connexion whatsoever with pulmonary phthisis as a chronic destructive lung disease. A few years ago this point of view was very widely accepted by pathologists in many parts of Europe. In the time of Laennec, pulmonary phthisis, in its common forms, meant tubercular disease of the lung, and tubercle was regarded as a "specific" product. Forty years later, the common varieties of pulmonary phthisis were regarded as due almost solely to various forms of chronic inflamma-

* On re-reading Bayle's four cases of uncomplicated "Granular Phthisis," I have become convinced that M. Thaon is right, and that these could not have been cases of what is now understood as acute tuberculosis.

tory changes in the lung; tubercle was regarded as a mere occasional and quasi-accidental complication: both phthisis and tubercle were robbed of their so-called "specific" attributes. This was the kind of view into which I finally drifted about the year 1866, recognising, however, fully, that the word "tubercle" had attained a thoroughly artificial meaning, wholly different from its original signification, and that the pertinacity with the name was of questionable expediency. Now, however, we see a powerful reaction setting in, and a marked tendency to restore to some of the old "infiltrations" the name of tubercle, so as at the same time to make phthisis again an essentially tubercular affection. What wranglings and never-ending disputations the very prospect opens up! What, it may be asked, is the meaning of the new point of view? We must endeavour to answer this question first, before attempting to come to an opinion upon the desirability of reverting to what one might almost venture to call an old and worn-out doctrine. The reasons which have been most influential, are, I think, not difficult to find. Most valuable investigations have of late years been made, both here and abroad, upon the so-called "artificial production of tubercle," and many of those who had carried them on had adopted the views of Virchow concerning the limitations to be attached to the word tubercle. Now the rodent animals, and guinea-pigs especially, are, in more ways than one, very peculiar creatures—so peculiar, it would seem to me, that it is not altogether safe to pass judgment offhand concerning the similarity of certain processes which may be set up in them, to those which are known to occur in the human subject. We now know that this artificial so-called tubercular affection may be initiated in guinea-pigs by the mere introduction of an ordinary irritant into and beneath the skin of the animal. Whether tubercular or not, therefore, the affection established is one which, in the old sense of the term, can have nothing very "specific" about it. The growths set up by local irritation gradually spread to lymphatic glands, and subsequently internal organs, such as lungs, liver, spleen, &c., also become affected. Such growths were at last believed by Villemin to be tubercular simply because they had followed the inoculation of "tubercle" beneath the skin of the animal, and because such a result was deemed harmonious with the supposed specific nature of the inoculating substance. But when it was clearly shown that ordinary chronic inflammatory products—even that the introduction of a mere seton beneath the skin—might give rise to similar morbid conditions and products in guinea-pigs, this reason became no longer of any avail. Something was, however, to be said, in favour of the lesions being tubercular, owing to the nature of the growths themselves. They had, in the main, the characteristic lymphoid or adenoid structure which the generalisation of acute tuberculosis possesses. But was this similarity sufficient to make it absolutely necessary to consider that the condition so frequently established in the Rodentia was tuberculosis, and that the lesions were tubercular? This is a most important question for pathological science, and one which, as it seems to me, never very carefully considered by those who were themselves engaged in these most interesting and important investigations. I cannot too strongly draw attention to this stage of the argument. Here was a generalised affection set up in guinea-pigs, marked by new growths, in the lungs and other organs, which presented the microscopical characters of lymphatic tissue. Was the histological constitution of the growths alone sufficient to justify us in calling these products tubercular? This question, thus nakedly put, would, I believe, have been unequivocally answered in the negative by many pathologists. None of the modern pathologists who had accepted the views of Virchow have ever pretended that the grey granulation had any characteristics specific and peculiar to itself. It was always recognised to be of the lymphoid type, and it was always admitted that growths in no way distinguishable from it histologically were to be met with in the organs of persons suffering from leucocythæmia and other allied affections. Growths of the lymphatic gland type—but to which no one thought of attaching the name tubercle—were in these affections more or less disseminated through different organs and parts of the body. Obviously, therefore, mere histological structure alone could give us no right to look upon the new growths in guinea-pigs as tubercular. As I have before endeavoured to point out, it was the grey granulation of acute tuberculosis which pathologists had arbitrarily agreed, in default of other products, to regard as the only true tubercle. If, therefore, this body was admitted to have no specific structure, if other products histologically similar were not named tubercle, then it becomes clear that the notion of tubercle was arbitrarily centred, not so much in the histological character of any individual product, as in its structure combined with its mode of occurrence in individual organs and throughout the body—it had become centered, in fact, in the grey granulation as the sole specific and invariable mark of that general constitutional affection known as acute tuberculosis, rather than in any mere form and histological characters of the grey granulation itself. Thus, the only point to be considered came to be, whether the general affection set up in the rodent animals was or was not identical with the febrile affection in man which, in the strict acceptation of the term, goes by the name of acute tuberculosis. I know not whether the question was ever argued out in this manner by others. It was obvious, however, that the majority of pathologists were quite willing to call these growths in the rodent animals tubercle, and the general condition itself tuberculosis. To this view I was never able to give in my adhesion. My difficulty arose from the fact that I was unable to see a real identity, such as appears to some, between acute tuberculosis as it occurs in man and the affection which could be so easily established in the Rodentia. The mode of origin; the comparatively slow and gradual spread of the guinea-pig affection from organ to organ; the almost invariable absence of growths in the brain or meninges; the mode in which lungs, liver, and spleen were affected—the wide-spread infiltrations and the greatly increased bulk of the latter organs—were all in striking contrast with acute tuberculosis as it occurs in the human subject. For in this latter affection we find an obscure origin, and the more or less sudden outbreak of an acute febrile malady which almost invariably leaves some marks upon the meninges, and in which the strictly essential anatomical lesions are minute granulations, either separate or aggregated, scattered through organs of almost normal size. With such differences staring us in the face, and with the patent, though by no means insignificant fact, that the means which sufficed for inciting the affection in question in the Rodentia were wholly incapable of setting up acute tuberculosis in man, it has always seemed to me to be very difficult to come to the conclusion that the two affections were similar, and therefore equally difficult to come to the conclusion that the products found in the rodent animals have any real right to be considered as tubercle. My doubts at the outset in these investigations and regarding their importance are, however, not in the least diminished merely because I do not feel justified in applying a particular name to the morbid conditions or products themselves. These doubts, however, which I still very strongly entertain, concerning the propriety and legitimacy of the present generally received views as to the name which should be given to the affection in the rodent animals, have not been shared by others. Foreign investigators, as well as Dr. Wilson Fox, Dr. Burdon Sanderson, and others in this country, have not hesitated to look upon the condition as tuberculosis, and upon the products as tubercular. Nay, more, Dr. Sanderson stated nearly four years ago, in a communication which appeared in the *Edinburgh Medical Journal* for 1869, that the occurrence of "infiltration," in which large patches of lymphoid new growth appeared in the liver or other organs of the guinea-pig, should suffice to open our eyes with regard to the different forms in which tubercle might manifest itself in the human subject. This, I might venture to say so, was the insertion of the thin edge of the wedge which Dr. Fox has now, with no much vigour, endeavoured to drive home. Already there was the dawning notion that infiltrations and chronic inflammations were again to have their day as tubercular products. I mentally shuddered at the chaos into which—I say it with all respect—it seemed to me we should again be introduced, and I was by no means comforted by the fact that new doctrines of "infection" were to be introduced with the view of explaining multiple morbid processes in general. In Dr. Sanderson's opinion, tubercles are adenoid bodies (or lymphoid patches) enlarged; and, for the establishment of phthisis three things are necessary: (1) a constitutional

predisposition, (2) a local irritation leading to an increased growth of pre-existing lymphoid structures, and (3) a process of infection, by means of which the morbid growths extend to adjacent or related parts. Concerning this process of infection, Dr. Sanderson says: "The word designates the fact that, wherever a chronic induration *due to overcrowded corpuscularities* exists in any organ, it is apt to give rise to similar processes elsewhere." Dr. Sanderson would apply these views even to the mode of extension of "the so-called infiltrated forms of induration" met with in ordinary cases of phthisis. Here, then, the tendency was strongly manifested to consider that all infiltrating fibroid indurations which were marked by "overcrowded corpuscularities," might spread by a process of infection, and the logical outcome of the views stated was that such infiltrating indurations were tubercular in nature. The transition to such a belief is all the more easy because, as I have before insisted (Pathological Transactions, 1868, p. 54)—in anticipation of what has followed—rapidly advancing fibroid over-growths in their early stages are as notable for their number of corpuscles as the most typical lymphoid tissue. The two differ, in fact, merely in the degree of perfection of their intercorpuscular stroma, although almost all transitions are to be met between the two. That difficulties may sometimes exist in distinguishing between such products is assuredly also to be the opinion of Dr. Wilson Fox, since he says, "The reticulum under a microscope with high powers is to be found in almost all forms of tuberculosis except in the most recent granulations, and there nuclei and small cells crowd upon one another, forming a dense mass, and no reticulum can be seen." The views which Dr. Wilson Fox has now so ably expounded will thus be found essentially similar to those which were more or less explicitly stated by Dr. Sanderson; and I cannot doubt but he also has been largely influenced by considerations similar to those which have found favour with Dr. Sanderson. It is true Dr. Fox claims to have an additional warrant for his views by proving that, in the lungs of children dying from acute tuberculosis, infiltrating corpuscular lesions are to be met with in addition to grey granulations in different stages. Such a fact, however, as it seems to me, can have no real title to induce us to modify our views, when we recollect that the restricted signification of the term tubercle, adopted by Virchow and others, is confessedly arbitrary, and necessitated by the complete overthrow of the old doctrines as to the nature of tubercle. Almost all forms of phthisis had previously been regarded as tubercular, under the belief that a "specific" and peculiar product was almost invariably present. Afterwards it had been shown that this supposed product proved to be no specific had nothing peculiar about it, and was the result of common inflammation in various forms, and that none of such products were worthy of the name tubercle; although, as a mere concession, pathologists were willing to allow this name to be retained by a product (the grey granulation) which was an accompaniment of many phthisis. It is literally true, therefore, that in the minds of those who had accepted his position, "tubercle had come to be a mere accidental complication of phthisis, and in no respect its chief anatomical distinction." And as it seems to me the fact that an arbitrary signification of the term had been consented to as a mere compromise, and simply with the view of retaining the old name for something—even when its original meaning had been entirely taken from it—is lost sight of by Dr. Wilson Fox, when he now comes forward and urges us to accept a new signification of the term merely because, in acute tuberculosis as it occurs in children, pathological products other than the grey granulation are apt to be found, although such products are confessedly not necessary elements of the disease. It seems, moreover, to me almost wholly beside the question to say, as an additional reason for the adoption of the new views, that "proof is wholly wanting that tubercle in the lung can appear in no other form than the isolated grey granulation." No proof was even attempted to be given: we must again urge the fact, that the restriction in the use of the term was from the first confessedly though advisedly arbitrary. The products which Dr. Wilson Fox and Dr. Burdon-Sanderson would now have us include under the name tubercle were therefore amongst those which, amidst the shipwreck of the old term, were deliberately cast aside. They were known and recognised as products of an irritative or chronic inflammatory overgrowth, under the names of interstitial pneumonia and fibroid indurations. Such products, not only in the lung, but also in other organs, are, as is well known, very largely, and often almost exclusively, made up of minute corpuscular elements similar to those occurring in lymphoid tissues. And if in some cases these overgrowths, or portions of them, as they occur in the lungs or other organs in man, have a still more exact resemblance to lymphoid tissue, the reason of this has now been made perfectly clear and simple to us, owing to the important discovery by Dr. Burdon-Sanderson that minute masses of lymphoid tissue are normally present in the peribronchial tissue of the lung, and upon the walls of the vessels in other organs. This being the case, what could we expect but that under the influence of such disturbing or irritative influences as lead to the overgrowth of the connective-tissue elements in any region of the lung, these intimately intermixed patches of lymphoid tissue should also undergo an irritative increase or hyperplasia. But, in the face of all that has been said, is this a justification for calling such bodies tubercles? Certainly not; no more than the possession of a similar histological structure was a sufficient warrant for immediately calling the morbid products which are met with in the guinea-pig's lung tubercle. If we are now to suppose that tubercle can be described broadly as "an adenoid body enlarged," or as a "lymphatic overgrowth produced by irritation," what are we to say of the lymphatic overgrowths which occur in the liver in leucocythæmia? Are they also tubercles? Again, what are we to say of a mere irritated and enlarged lymphatic gland? Is this a tubercle? Or is the body no longer tubercle when it attains a certain size? Everywhere, as it seems to me, such views as have now been advocated land us in nothing but confusion. If the word tubercle is be retained at all, we cannot start beyond the narrow circle of the limited and confessedly arbitrary signification given to it by Virchow and others, without plunging ourselves into a mere whirl of inconsistencies and contradictions. Let it not be supposed, however, that I am in favour of retaining the word—even with the limited signification given to it by Virchow. No; it seems to me, for many reasons, by far more expedient to renounce its use altogether. To this opinion I have come slowly and deliberately, after an experience of several years in teaching the present doctrines. Year by year I have been more and more impressed with the altogether gratuitous and unnecessary difficulties besetting the path of any teacher who endeavours to explain to students what is and what is not tubercle, and why anything product is or is not to entitled to such a name. The principal difficulties seem to me to be of this nature. First, there is the fact that in ordinary chronic phthisis some of the lesions are most prone to appear in the form of granulations either simple or aggregated, although such granulations have nothing whatever to do with acute tuberculosis in the strict sense of that term, and therefore have no right to be called tubercle. Such granulations may be softer than the ordinary grey granulation, owing to their containing a larger proportion of epithelial elements or of their derivatives; others may be harder and more pigmented; whilst others still may, both in naked-eye characters and in microscopical appearance, be almost indistinguishable from the grey granulations of acute tuberculosis. And yet there is oftentimes not the slightest suspicion that acute tuberculosis has existed—far from it; growths of this kind are occasionally to be met with in such a mere local, though chronic, disease as cirrhosis of the lung. In one of the most typical cases of this affection, where the opposite lung was healthy, and where no granulations were to be found in other organs, the part in which the fibroid consolidation was still advancing was thickly studded with minute granulations, having microscopical characters closely resembling those found in acute tuberculosis. Again, take the affection commonly known as "tubercular peritonitis," but which I prefer to call granular peritonitis. Here the parietal and visceral peritoneum is more or less densely overgrown with granulations, presenting a truly lymphoid structure. Yet such an affection has, as I believe, no necessary connexion whatsoever with acute tuberculosis. If we would be consistent, therefore, we must say that the products are not tubercular. Occasionally, such growths in the peritoneum and omentum are more lawless still: the reticulum is not developed; we have

nothing but a prodigious overgrowth of minute corpuscular elements, so exuberant, however, that the growth may win for itself the more dignified appellation of "cancer." Again, the correlation between lymphoid overgrowths and mere ordinary irritative overgrowths of connective-tissue elements is most close. In the five or six cases in which I have made an examination of persons dying from well-marked granular peritonitis, I have found the liver similarly and very characteristically altered. The organ has been more or less enlarged, pale in colour, and minutely mottled both on its external surface and on sections. What appeared to be minute, pale, and yellowish granulations, were thickly sown through a basis-substance, much of which had a more or less pellucid appearance. On microscopical examination, the pale and yellowish areas were found to correspond to islets of liver-cells more or less distended with fat, or simply granular and bile-stained, these areas being imbedded in an enormous new growth of tissue which had replaced the proper liver-substance. The new growth consisted, in part, of distinct fibre-tissue; in part, of a mere nuclear overgrowth, amongst which there was no definite reticulum; and in part, though in much smaller quantity, of more distinct patches of characteristic lymphoid tissue. On the other hand, kidneys, spleen, lungs, brain, and meninges have been entirely free from any characteristic or constant changes. There has been no evidence, therefore, of the existence of acute tuberculosis. What I have already said may suffice to indicate a few of the difficulties besetting the path of the teacher, who, seeing the destructive snares which lie in wait for him if he venture beyond the narrow circle of a confessedly arbitrary definition of tubercle, tries what he can do to keep within these narrow and monotonous limits. Attempt to step beyond these bounds, and see what follows. In answer to the question, what is tubercle? let one who is disposed to be adventurous, reply, in the terms of Dr. Fox and Dr. Sanderson, that it is a new growth, lymphoid in nature, and resulting from a hyperplasia of a pre-existing nidus. The preservation of the word "tubercle" is beset with difficulties. No one knows what another means when the term is used, nor can he explain his meaning without entering into tedious statements. We have all this worry about a word we can perfectly well do without. The facts themselves will not be weakened by giving up the use of the word, but will be rendered more significant. A few words will indicate the views which would be accepted if the word tubercle was sunk altogether. The affection we have spoken of might be thus characterised :—1. The disease —acute tuberculosis—might be spoken of as granulia, a name given it by M. Empis. All the facts known concerning the disease would remain the same. 2. Tubercular peritonitis might be spoken of as granular peritonitis: the condition has no necessary connexion with the acute general disease granulia. All the known facts concerning its natural history would remain the same. 3. Intermediately between these two affections we should place the lymphatic affection in the rodent animals, the so-called artificial tubercle, the characteristics of which have been well described. 4. Two allied affections as distinct from pulmonary phthisis, and which are characterised by disseminated new growths having a lymphoid character—viz., leucocythæmia and the affection named adenic by Trousseau. 5. Lastly, we have the great majority of cases of pulmonary phthisis—a malady which has nothing more "specific" than belongs to chronic Bright's disease in the kidney. It is the various proportions in which these different kinds of lesions exist in different cases, combined with their different stages of evolution or decay, which accounts for the varying appearances presented by one case of phthisis as compared with another. In the main, these tissue changes, as almost every one admits, belong to three different categories. They are as follows:—(*a*) *Epithelial or more or less purulent impactions* within the bronchi and air-cells, occupying areas of very different sizes, and presenting different colours of grey or yellow, according to the amount of fatty degeneration or caseation which they have undergone. Changes of this kind are more or less intimately intermixed with those which follow. (*b*) *Fibroid overgrowths* resulting from a hyperplasia of connective-tissue elements in different parts of the portion of lungs affected; these being characterised by tissues presenting every grade of structure or variety between mere nuclear overgrowths of an embryonal character and the densest fibro-cartilage. (*c*) *Lymphoid overgrowths*, more or less inextricably interblended with tissue-changes of the kind last mentioned, due to an irritative hyperplasia of the normal lymphoid patches which are to be met with around the bronchial tubes and their minute ramifications. The changes described under *b* and *c* together constitute the well-known *indurating infiltrations*. The two kinds of changes occur in intimate union; and the more characteristic lymphoid hyperplasias shade away insensibly into the ordinary embryonal or nuclear overgrowth of the connective-tissue elements. The amount of actually developed fibre-tissue in this intermixture of *b* and *c* increases with age or in proportion to the slowness of its evolution. As Dr. Fox has so forcibly pointed out, the tendency to undergo caseation or cheesy metamorphosis is by no means confined to the epithelial impactions; it extends also to these indurating infiltrations, and is generally well marked in direct proportion to the rapidity of their evolution and the abundance of their corpuscular elements. The more acute the cases of phthisis, the more apt are we to meet with a predominance of mere epithelial or purulent impactions; whilst the more chronic the cases, the more abundantly do we find the indurating infiltrations and granulations above referred to. It is, however, as Dr. Fox has remarked, almost impossible not to get some amount of the other change where either of them exists to any well-marked extent. Taking into account the ulcerations, pigmentations, and other changes which these morbid tissues and products are apt to undergo, all the anatomical characters of pulmonary phthisis are explicable enough, without the necessity of our ever having recourse to a word the very mention of which suffices to summon to the mind a confusing cloud of unproved assumptions and conflicting theories. And if the lessons themselves of pulmonary phthisis can be fully explained without resort to or occasion to use the word "tubercle," this is also certainly true concerning the general constitutional condition associated with the malady, and concerning anything we may know or say as to its hereditary nature. Those who have occasion to use the phrase, "tubercular diathesis," will find themselves none the less wise if, in future, they speak or think of a "phthisical diathesis." And, surely, nobody in these days, when so much more is known concerning heredity, will think it necessary to keep up old notions concerning the "specific" nature of a very ordinary disease, simply because there is evidence to show that a tendency to such a disease is frequently transmitted from parent to offspring. A man may inherit from his ancestor a well- or an ill-developed brain, and similarly he may inherit a well- or an ill-developed lung. If he have an ill-developed brain, we should say that he inherited a predisposition to brain-disease, though the particular form which might appear would be altogether uncertain. Nobody doubts, however, that quasi-pathological accidents may determine in another individual, who inherits no such predisposition, similar forms of disease. The case is precisely the same with regard to lung-diseases. A man may inherit from his ancestors lungs which contain within themselves the elements of weakness—organs the tissues of which are so constituted, with relation to the whole organism, that the very slightest determining causes suffice to initiate a set of changes which terminate in one or other of the forms of pulmonary phthisis. And, similarly, just as brain-disease may be acquired in the life of an individual who inherits no predisposition, so, under the strong pressure of general and local causes, may any of the forms of phthisis manifest themselves in individuals who inherit no family predisposition to such a disease. Where is the difficulty? What occasion have we to resort to the use of the word "tubercle"? How does so much depend upon the use we make of it? I must confess myself unable to understand what Dr. Wilson Fox means when he says—"The etiology of phthisis, the therapeutics of phthisis, and the prognosis of phthisis, all hang upon this point—how far tubercle is concerned in the morbid anatomy of phthisis." To me it seems quite the reverse. I regard this question as one of a mere verbal nature, and as capable of being separated entirely from all problems as to the etiology, therapeutics, and prognosis of phthisis. Were it not for the very important nature of these questions under discussion —were it not for the fact that professional opinion in this country might be led into grooves which would subsequently, as I conceive, introduce a lamentable confusion into the science of medicine and pathology—I should not have ventured to speak at such length. What I have said, however, may be taken as the expression of views which I have arrived at slowly and deliberately, after the most earnest consideration of all the facts. I would say then, emphatically, if we accept the new signification which has of late been proposed for the word "tubercle," we involve ourselves in an endless series of disputes and differences of opinion as to the real nature and limits of such a growth as compared with many chronic inflammations, with syphilitic indurations, scrofulous products, leucocythæmic growths, the lymphomata of adénie, and other morbid products. Whilst, on

the other hand, if we throw away this indefinite and almost meaningless word "tubercle," we shall at the same time get rid of an entangled brushwood of conflicting opinions, and of a series of pitfalls which simply hinder our progress, and prevent that almost complete unanimity concerning the mere facts themselves which would otherwise prevail.

Dr. PAYNE said he wished to ask Dr. FOX if he would state in his reply whether this view of his general argument was correct, that cases of acute tuberculosis formed the pivot of his argument, and that the seventeen changes found in the lungs of children were due to acute tuberculosis, and that he regarded changes in chronic phthisis as due to suppuration. If this was the point of the argument, it was important to know in what sense acute tuberculosis is understood, and the kind of disease in the children whose lungs were examined. Then we had to consider the chronology of the products and the appearances met with. He must admit that the question was by no means simple, and even in the best marked cases it was difficult to decide the dates with accuracy. On examining the lungs of patients dying with phthisis, we find grey granulations or tubercles (Virchow), or what might be called by some other name (only it must have a name). We found tubercle in this sense, and inflammatory change. There were two views as to the sequence—either the tubercle was first, or the other change was first. To state briefly the results of such an inquiry, there would be cases where the tubercle was the first, and the inflammatory change secondary; or the inflammatory change might be the first, and the tubercle secondary. He thought this because there was no preparation, and that he had brought these to-night, which showed an ordinary tubercle surrounding a vein, near it were air-cells with inflammatory changes; also other cells with catarrhal changes and hæmorrhage; also a bronchus, showing the results of inflammatory change. The tubercle itself showed no sign of degeneration, no sign of age; so here the inflammatory changes of the bronchus were anterior in time to the tubercle. In many cases like this we might say the inflammatory change from local affection was anterior to the production of tubercle, and the changes in the air-cells subsequent to the tubercle. From this evidence, that when the lung was full of tubercles, and in one part larger, yellower, and more opaque, there were the cells, and these the larger were not true tubercles, but enlarged by inflammatory changes; hence, no doubt, tubercle was first here, and inflammatory change secondary. The two classes of cases were clear; if we asked how tubercle causes inflammation and inflammation tubercle, we had a large scope for argument. We must employ experiments, &c. That tubercle was produced by the influence of inflammation he thought had been established by pathological experiments—that inflammatory matter induces this change. There was no difficulty in supposing it acted as an influence, as in pyæmia, at times directly or through distant channels; when a cavity in the lung would result, and this was absorbed and lung affected, and tubercle formed; or inflammatory matter from a distance conveyed to the lung, and miliary tubercles were found scattered through the lung. When tubercle was in a lung it acted (as Laennec said) as a mere mechanical agent, or from containing a septic agent; either was possible. When both tubercle and inflammatory changes were present, not the one produced by the other, but by some common cause, say some septic substance, in one part of the lung-tissue you meet with inflammatory change of a catarrhal kind, in another part tubercle. If it was said that early phthisis depended on inflammatory changes, then he thought we were open to the charge of speaking on insufficiency of knowledge; it was not certain that catarrhal diseases formed the beginning of phthisis; it was not associated with ordinary catarrhal diseases, but both were accompanied by hyperæmia, &c. The inflammation dependent on syphilis could not be distinguished from those simply produced; so it was by no means proved that there was not some septic substance in which produced tubercle. No one word to say as to the internal structure of tubercle: he could not agree with the statement that it was always adenoid tissue; sometimes it was like it, but not always. Tubercle was not often symmetrical, and its appearance depended much on the section.

Dr. DOUGLAS POWELL observed that the main question in debate was as to the restrictions to be applied to the terms tubercle and tuberculosis respectively. The facts brought forward by Dr. FOX, and illustrated by his specimens, drawings, and diagrams, were so faithfully and truly represented that they would be accepted and confirmed by the experience and observation of all who had most studied the subject. But these facts would naturally be regarded by different observers from different points of view, and different inferences would be drawn from them. Dr. Powell accepted Dr. Fox's definition of tubercle as most typically applying to the grey granulation, and as not essentially implicating the inflammatory changes with which the granulation was so often associated, and he would restrict the term "acute tuberculosis" to that acute disease all the local phenomena of which were occasioned by the definite anatomical element, tubercle. The striking pathological characteristics of acute (pulmonary) tuberculosis were that, usually a part of a general disease, its anatomical element, the grey granulation, was developed almost simultaneously throughout the lungs and any other organ that might be affected; there was found post mortem very little difference in date between the tubercles in different organs; that these granulations in the lung were in such typical cases unaccompanied by any pneumonia. On this point he, with great respect, slightly differed from Dr. Fox. He had often seen in the acute tuberculosis of adults the tubercle unaccompanied by any pneumonia, every portion of the lung floating freely in water, although there might be found on minute examination some epithelial shedding, such as is common to all active or passive congestions of the organ. Again, in the acute tuberculosis there was no breaking up of the lung tissue; there were no lung elements in the sputa; the patients did not die of lung obstruction as in the case of acute phthisis, but of the general disease and obstruction to respiration. On the contrary, he agreed with Dr. Fox that one of the striking characteristics of tubercle was its tendency to fibroid development. On these grounds he thought *acute tuberculosis*, as thus restricted, could not be admitted under the definition of phthisis at all, and that tubercle should not be regarded as the essential specific element in phthisis. Dr. Powell fully admitted the local development of tubercle, both in the granulation and diffused form, as frequently present in the lesions of acute and chronic phthisis; but this local tubercle was always attended with inflammatory changes and breaking up of lung, and he could not regard the tubercle as the element primary or specific to these changes, nor speak of such cases as varieties of tuberculosis. Dr. Powell concluded by observing that he thought much of the intricacy of phthisical lesions would be explained without the aid of any specific precedent deposit tubercle, if we remembered the peculiar construction of the lung as an intricate unfolding of a surface continuous with a mucous surface, and very analogous to it, but richer in lymphatics and bloodvessels. By a mere passive expiration we could not recover without danger or difficulty the products of nasal or bronchial catarrh, but the products of an equally simple affection of the alveoli could not be so easily expelled; they accumulated, decayed, irritated the alveolar wall and set up those proliferative and inflammatory changes which constituted local tubercle—just as the retained secretion of a sebaceous follicle gave rise to the sebaceous pustule; the thickened alveolar walls in their turn degenerated, softened, or suppurated, and in these changes, complex only with the complexness of the surface in which they occurred, we saw the rough but accurate outlines common to the many morbid pictures presented to us by the lungs of those dead of phthisis. Taking further into account the constant cease-less movements of the lungs, and the free access of air to the diseased parts, and the mystery was to him that such lesions were not even more intractable and destructive.

The discussion was adjourned to the next meeting, when, the President stated, the discussion must be concluded, and if needful for that purpose the meeting would be prolonged beyond the usual time.

BEQUESTS, &c., TO MEDICAL CHARITIES.—The Directors of the Bank of England have voted £1000 towards the London Hospital Extension Fund. The Great Northern Hospital has received a fifth £1000 from "S. W. Y." Mr. William Merry, of Highlands, Wilts, bequeathed £3000 to the Royal Berks Hospital, payable on the death of his widow.

HÆMATOZOA.

To the Editor of THE LANCET.

SIR,—Allow me a short space to explain a portion of my paper on the canine filaria, which appears from the second paragraph of Dr. Cobbold's letter in your last issue to be misunderstood—viz., "the hæmatozoon in the dog being referred by me to the so-called Spiroptera sanguinis."

My intention was to show that the anatomical details of the worms sent by Dr. Lamprey negatived the idea of their being "spiropteræ," and justified me in classing them as "filariæ."

I also mentioned that the canine nematodes hitherto named spiropteræ did not appear to have been accurately determined as such, while their "crowded features" suggested the linking of them with Dr. Lamprey's specimens—*i. e.*, filariæ; and, considering the concordance in many details between the Netley Museum worms and those described by Gruby and Delafond, I noted the propriety of retaining their nomenclature at least as far as the genus was concerned.

Dr. Cobbold, however, has shown in his letter that the canine hæmatozoon has already received the name abroad of Filaria immitis, and the felicity of such a designation to embrace the main characters of the nematode cannot be questioned.

Correcting this error in Dr. Cobbold's complimentary letter,

I am, Sir, &c.,

FRANCIS H. WELCH, F.R.C.S.

Royal Victoria Hospital, Netley, March 29th, 1873.

BIRMINGHAM.

(From our own Correspondent.)

THE municipal authorities and the magistrates of the borough have come to a dead lock with regard to the appointment of a surgeon to the gaol. The late surgeon received £200 per annum, and that was quite little enough, considering the onerous and important duties he was called upon to perform; but when Dr. Hill resigned, the so-called economists in the Town Council carried a resolution reducing the salary to £100. The magistrates, with whom the election rests, declined to appoint anyone at such a pittful a remuneration, and requested a conference with the Town Council, when they pointed out how inadequate the proposed stipend was, and how much more was paid for similar services in even smaller gaols. Still, argument was of no effect; numbers prevailed, and again the Council has resolved that they will only give £100 a year for a gaol surgeon. It is to be hoped that the magistrates will not recede from the position they have taken, and that they will decline to act as the servants of the corporation.

Dr. Robinson, the vaccination officer, has had a successful year:—8052 births were registered during the twelve months, and of these 6702 had been successfully vaccinated; 714 died previous to vaccination, and only 52 cases could not be accounted for. Small-pox yet lingers in the district, and a few deaths have recently occurred from it. The death-rate is 24·4.

"Hospital Saturday" is still bearing fruits: the sum of £4060 which you noticed a fortnight ago, as the probable result of the collections in the manufactories and workshops, has already swelled to £4700, and the example set at Birmingham has already been followed at West Bromwich, and will shortly be introduced at Walsall in aid of the Cottage Hospital. The older source of income, the "Hospital Sunday," has again done good service at Wolverhampton, by the addition of £915 to the funds of the South Staffordshire Hospital.

The people of Dudley are complaining bitterly of the insufficient and costly nature of the water-supply of their town. As fever has so recently visited them, it is incumbent on the corporation to see that the inhabitants are supplied with an ample and pure supply of water, and also that they are not compelled to pay an extortionate price for it by the officers of an irresponsible water company.

At the recent annual meeting of the Midland Counties Middle-class Idiot Asylum, Dr. J. C. Bucknill, F.R.S., delivered an address on the usefulness of and necessity for such institutions, and made an earnest appeal for increased aid for this charity.

Supra-pubic lithotomy in children is rare, but a successful case of that operation is now in the Queen's Hospital: the patient is a little girl aged 3½, and the stone, which was of lithic acid, measured ¾ of an inch in diameter. Mr. Jordan operated ten days ago, and though the urine has since passed through the external wound, no peritonitis has taken place. Mr. Jolly lately performed resection of the ankle for caries in a young man aged 27, at the General Hospital, and the patient has done well.

At the last meeting of the Midland Medical Society, Mr. Gilbert Smith presented a specimen of fracture of the spine in the cervical region, and a hydrocephalic brain. Mr. West read notes of an ovariotomy in a lady, aged 32, which had terminated successfully. The case was complicated with pregnancy, the patient having been prematurely delivered of twins just two months prior to the operation. Dr. Savage exhibited a uterine fibroid, removed by lateral incision, and Dr. Pritchard Davies read a paper "On Meningeal Neuralgia."

Birmingham, March 31st, 1873.

Obituary.

J. S. WILKINSON, M.R.C.S.

THE late Mr. J. Sebastian Wilkinson received his professional education at St. Thomas's and Guy's Hospitals. He was an esteemed pupil of the late Sir Astley Cooper, and served the office of dresser to the late Mr. Chandler; later he assisted Mr. E. Granger as Demonstrator of Anatomy. He became a member of the Royal College of Surgeons in 1820; afterwards entered the Royal Navy, and was attached to the Royal Naval Hospital at Plymouth, under Sir Stephen Love Hammick, which appointment he resigned and returned to London, opening a private class in Dean-street, Borough, to teach anatomy, physiology, and surgery, in which he was distinguished for a very apt mode of conveying a ready insight and clear knowledge, and was very successful. The class was largely attended, and amongst his pupils was the late Dr. Hodgkin, and other distinguished men. Whilst holding these classes, the late Mr. R. Granger, who succeeded his brother at the Webb-street School of Anatomy, offered him the demonstratorship, which he accepted, and held for a short period, when the late Mr. Pilcher made terms with Mr. Granger for a partnership and superseded him. This led to much disappointment among the students, who met and offered to open a new school if Mr. Wilkinson would consent; this he declined on account of sincere friendship for the Grangers. He contributed several communications to THE LANCET, and was particularly distinguished to the last for his remarkable memory of anatomy, and power and facility of demonstration. Of late he had settled in the City, succeeding to the practice of an old pupil and friend.

HENRY EAMES, A.B., M.D., T.C.D.

IT is with regret that we record the death of this gentleman, which took place on the 24th March, at his residence at Upper Fitzwilliam-street, Dublin, from that scourge of our profession, typhus fever, at the early age of 31. The disease was of the most malignant form, and proved fatal after a short illness. The deceased was a Lecturer on Medicine in the Ledwich School of Medicine, Physician to Mercer's Hospital, and Honorary Secretary to the Medical Society of the College of Physicians. Possessed of an ample fortune, and gifted with abilities of a high order, he would unquestionably have obtained a foremost position in the profession in the city he resided in, had his life been spared. He was of a kind and genial disposition, and his loss is felt deeply by his medical brethren. Dr. Eames leaves a widow and three children to deplore his untimely end.

Medical News.

APOTHECARIES' HALL.—The following gentlemen passed their examination in the Science and Practice of Medicine, and received certificates to practise, on March 27th:—

Hassall, William Charles, Lexton, Somerset.
Lindsay, William Vickress, Pulham-place, Paddington.
McDonnell, Michael Sweeny, Storrington, Sussex.

As Assistants in Compounding and Dispensing Medicines:—

Currah, George Ingersoll, Falmouth.
Earle, John, King's College Hospital.
Severs, Samuel Thomas, Ripon.
Stevens, Joseph, Stourbridge.

The following gentlemen also on the same day passed their Primary Professional Examination:—

Frederick Hawes Elliott, University College Hospital; Charles Louis Wells, Guy's Hospital; John Payne Massingham, Queen's Hospital, Birmingham.

Tuberculosis & Mal Caseux

Dr. HERMANN WEBER introduced the question of the Dependence of Tuberculosis on the existence of Caseous Deposits in the Body. He communicated the history of the case of a man who had died from tubercular meningitis six months after having had typhoid fever. The post-mortem examination showed the cicatrices from the typhoid affection of the intestines, enlarged caseous mesenteric glands, and the phenomena of tubercular meningitis, and of miliary tuberculosis of the lungs and pleura. Dr. H. Weber then discussed the connexion between the caseous mesenteric glands and the tuberculosis as cause and effect. After having given a short sketch of the views of Dittrich, Buhl, Virchow, Niemeyer, and C. E. Hofmann, he mentioned that in all the cases of tuberculosis of the serous membranes which had come under his own observation during the last five years—viz., eleven cases of tubercular meningitis, and four of tubercular peritonitis—caseous deposits were found in the body; and he added that the dependence of tuberculosis on the previous existence of caseous affections in the body had so impressed itself on him, that in the last three cases of tubercular meningitis which had occurred to him, he had to a great degree been guided in his diagnosis by the history of previous affections likely to leave behind caseous deposits, as of catarrhal pneumonia, pleuritis, measles. In five out of eleven cases of tubercular meningitis, the caseous deposits had existed in the lungs, in two in the pleura, in one in the mesenteric glands after typhoid fever, in one in the cervical glands after scarlet fever, in one in the same from impetigo capitis, and in one in a scrofulous knee-joint; in all the four cases of tubercular peritonitis, the caseous masses had existed in or near the abdominal cavity—viz., in the mesenteric glands, the kidneys, the lumbar muscles, and the ovaries. In some of the cases the tubercles were most abundant and of greater size and age in the immediate neighbourhood of the caseous focus; but in others the adjacent tissues were almost or quite free, whilst remote parts, as the meninges of the brain, were the seat of tubercles, showing that the disease need not spread by contiguity, but may at once be carried to distant organs. The author alluded to the important relation between this question and the experiments on artificial tuberculosis in animals, especially those by Villemin, Sanderson, Wilson Fox, and Waldenburg, and he requested the members of the Society to examine in how far the following proposition be correct—"That miliary tuberculosis is produced by the introduction (generally through absorption) into the circulation of minute corpuscular elements from caseous and allied pathological products, and by the migration of these particles to numerous points of different organs, there giving rise to the formation of nodules and further changes."

Dr. WILSON FOX had already alluded to effete matter as a cause of tubercle, and he confirmed the views of the Germans, particularly Hoffmann, but in Clinical Medicine he thought the necessity of having caseous matter might be pushed too far. He rather looked on the caseous matter in the glands as an expression of the tubercular diathesis, of a past attempt on the part of the subject to become tuberculous. Such do not easily become caseous, but show the tendency to tuberculosis. A secondary outbreak might prove fatal. This showed that tubercle was a curable disease, that the patient might have several attacks, only yielding to the last of them. He had some doubt as to the caseous gland as a source. In experimental inquiry the disease was never at a standstill; from the site where it was caused by local irritation, where there was almost invariably lymphatic change, the same as that internally, extending to the internal organs affected, there was a continuous chain, and both spread by continuity. There was not this connecting chain between the caseous gland and the tubercular deposit in Clinical Medicine. He thought there was still something behind all this. Only certain animals were susceptible of tubercular inoculation, and that might be by local injury. This was not so with man.

Dr. BURDON SANDERSON said tuberculosis could be produced in some animals by feeding them on tubercle. The researches of Chauveau, not yet published, proved this. He selected heifers in good health: tubercle from the lungs of men and animals was pounded in water and given to these one, two, three, or four times. After from one to three months their trachea and bronchi became the seat of granulations under the epithelium close to a mucous gland, extending along its duct and round about it, but not in it. In the lung the granulations were subepithelial, others were outside the bronchi. Besides these there were nodules like infiltrated tubercle, and having the same structure. In the intestines were sub-epithelial granules, and even collections in the villi. The liver was also tuberculous. These animals were liable to a disease similar to that of man, and the tubercles acted in exactly the same manner in both. The granules were formed from the normal adenoid tissue surrounding the vessels, etc.

Dr. MURCHISON said, with regard to Dr. Weber's reference to his specimen shown last year, there was an ulcer of old standing in the duodenum, and the patient was cut off by tuberculosis. There was no tubercular predisposition, but there was great enlargement of the glands near the duodenum and at the root of the lung.

up than usual. There were some cysts in the centre, and a mass of calcareous matter under the periosteum. The tissue was mostly connective, with some spindle cells.—Also another, for which Mr. Nunn had amputated through the thigh. This patient died of pyæmia. The tumour was a spindle-celled sarcoma. There was no clear connexion between these and accident, although the latter often called attention to them. They used to be called medullary and osteo-sarcoma, and to be counted malignant. His own experience showed that amputation in the continuity of the bone was perfectly safe in such cases. This would not be so were they cancerous.—Referred.

ASSOCIATION OF MEDICAL OFFICERS OF HEALTH.

SATURDAY, FEBRUARY 19.

MR. LIDDLE, President, in the Chair.

DR. STALLARD was elected a member of the Association.

Dr. WOODFORD brought up a report of the visits made by himself and Mr. Liddle to Messrs. Fraser's works to test the working of their disinfecting apparatus. After sundry experiments and modifications, the apparatus proved successful, inasmuch as a thermometer placed inside one of the beds subjected to the apparatus registered a temperature of 273° without the articles being scorched or injured. In other respects also the apparatus seemed to work well. A conversation arose as to whether it would be possible to prevail upon the Legislature to remedy certain defects in the Building Act; and it was eventually determined that the General Purposes Committee should meet to consider the subject.

Dr. E. BALLARD next read a paper on Suggestions made by Mr. James Lewis for National Sickness Returns, in which he stated that what was proposed by Mr. Lewis had been carried out by him for several years in his own district. His form also would be found on examination to tally with that of Mr. Lewis. Dr. Richardson proposed to go further, and give particulars as to age, sex, occupation, &c.; but he (Dr. Ballard) thought the simpler plan ought to be preferred. He agreed with Mr. Lewis that the returns, to be made valuable, should be issued weekly. With regard to the returns being forwarded to a central office, he wished for further information as to what office was proposed, whether an existing one or one to be created. His own opinion was that the office to which the returns should be sent ought to be the office that is most intimately concerned with the sickness of the country—whatever that may be. He thought, however, that

having died from disease or the [illegible]

NEW INVENTIONS.

THE NAUTILUS LIFE-PRESERVER SWIMMING-BELT.

WE had an opportunity of seeing this admirable invention in use on Thursday at the Endell-street Swimming-baths. This trial had no doubt the advantage of bringing home more directly to the understanding the merits of the invention, but its usefulness has already been sufficiently attested by competent authorities. Its materials are india-rubber and canvas, held in proper position by a steel spring. The air by which it is distended, and to which it owes its buoyancy, is readily admitted, but does not readily escape, any more than water can readily enter when the belt is injured. The fastening is simple and complete. The apparatus is manufactured and sold by Maw, Son, and Thompson, Aldersgate-street, London.

NEW BOOKS, WITH SHORT CRITIQUES.

*** We are indebted to the courtesy of several of our contemporaries during the past year for interesting enclosures and issues of their periodical publications. Many of these on perusal have been found of great interest, but the pressure of home materials has prevented our doing them anything like justice. Among recent acquaintance we may notice the *Pabellon Medico* as conducted with much spirit, and some numbers of the *Gaceta Medica de Granada*, just received. Spain has now about half a score of Medical publications which follow pretty closely in the track of science. Of those of our contemporaries whose appearance we miss during the current year we may mention with regret the *Escholiaste Medico*, the best known Medical periodical of Portugal, which has come to an end after a career of twenty-six years' duration under the auspices and support of Government, and under very talented direction. We can say scarce less of the *Hygiene Militar* in Madrid, also in connexion with the army, which, after a shorter term of existence, came to an end in Spain a year previous, though maintained throughout with much ability. Both these Governments deserve credit for advanced undertakings, in which we lament to have seen less perseverance in their original views.

Effets du pneumothorax et de l'épanchement consécutif chez les phthisiques, par le Dr A. Czernicki (*Gazette hebdomadaire*, 19 juillet).

Dans la discussion sur la thoracentèse, à l'Académie de médecine, M. Pidoux rappela cette opinion de Laënnec que dans les *phthisies irrégulières*, tant qu'il y a épanchement, la diathèse reste stationnaire, mais qu'elle devient violente après sa diparition. M. Hérard, de son côté, appuya cette opinion en déclarant que dans quelques cas la compression de l'épanchement peut enrayer le développement tuberculeux, M. Czernicki rapporte deux observations qui donnent, dit-il, une complète confirmation à cette proposition. Deux tuberculeux, arrivés à la troisième période de leur mal, furent, *in extremis*, frappés de pneumothorax suivi d'épanchement. Cet accident, si formidable et si redouté en général, enraya manifestement la diathèse et prolongea d'une façon inespérée la vie des deux malades.

Les deux observations sont données avec nombreux détails et sont prises avec grand soin. M. Czernicki pense que l'épanchement comprime les parois des cavernes et entraîne une anémie locale défavorable à la suppuration pulmonaire et à l'hypersécrétion bronchique; alors l'expectoration purulente se supprime; la fièvre, les sueurs, la diarrhée, la dyspepsie, disparaissent, car leur générateur, l'ulcération pulmonaire, se cicatrise, comme l'autopsie le démontre. M. Czernicki donne donc le conseil de ne pas évacuer par la thoracentèse l'épanchement consécutif à un pneumothorax, chez les tuberculeux, dans certains cas du moins.

F. R.

X..., 38 ans, entre le 17 avril 187?, à la Maison de santé ; c'est un homme d'une constitution appauvrie ayant contracté quatre blennorrhagies aigües (la dernière, il y a sept ans). Quelques semaines avant son entrée à l'hôpital, X... fut pris de rétention d'urine avec ténesme, et en même temps il se forma une petite tumeur au périnée, en avant de l'anus. Celle-ci suppura, fut incisée, mais de là ultérieurement les téguments furent pris à une certaine profondeur, en arrière du bulbe. M. Demarquay diagnostiqua un abcès ayant pour point de départ les glandes de Cooper. Les parties décollées furent largement incisées, mais la cicatrisation se fit mal, et l'urine suintait parfois par la plaie.

Le 1er juin, la peau de la verge devint rouge, tendue et douloureuse à la partie inférieure, et le 5 juin, le pénis présentait dans sa totalité un volume énorme ; il semblait dans l'état d'érection. Au toucher, on percevait une sensation de dureté plutôt que d'empâtement ; la pression était médiocrement douloureuse. — M. Demarquay diagnostiqua une *penitis*.

Le surlendemain le malade fut pris d'accidents généraux graves qui amenèrent la mort le 10 juin — Voici le résultat de l'autopsie :

Corps caverneux. — Du côté gauche, les altérations portent principalement sur la partie moyenne ; il y a là un véritable foyer purulent formé par la destruction complète des cloisons — Du côté droit, les lésions sont à peu près les mêmes, mais le foyer est moins étendu.

— Hanad. anevrysme de l'aorte (crotte? concavité.
recueilli chez Bucquoy.

— dans un des poumons, le gauche le murmure vésiculaire disparaît et souffle caverneux

atrophie — La branche gauche de l'artère pulmonaire était repoussée en avant; — le poumon gauche était caséeux avec caverne isolée — mais pneumonie généralisée et complète — art. pulmonaire très rétrécie. — dans cor

— Le cœur était atrophié. (chose remarquable)
Il n'a pas pu trouver de plexus cardiaque

artère, celle-ci beaucoup moins volumineuse que celle-là, et reconnaissable d'ailleurs aux divisions dichotomiques qu'elle présente.

Voici maintenant en quoi toutes ces particularités se trouvent modifiées dans le cas d'*induration grise progressive*.

La papille n'a pas éprouvé de changement, soit dans sa forme, soit dans ses dimensions; ses contours sont toujours très accentués. Les vaisseaux restent ce qu'ils étaient auparavant; seulement, contrairement à ce qui a lieu dans l'état normal, on ne peut plus les suivre, pénétrant à une certaine distance dans l'épaisseur de la papille, sur laquelle ils paraissent être simplement appliqués. Rien d'ailleurs qui s'éloigne profondément de l'état normal; mais voici le caractère décisif. Par suite du changement de texture qu'a subi le nerf optique, et en conséquence surtout de la disposition du cylindre de myéline, la papille a cessé d'être transparente; elle réfléchit au contraire fortement la lumière, et ne laisse plus voir dans sa profondeur les vaisseaux propres. Il s'en suit qu'elle ne présente plus la teinte rosée normale, et qu'elle offre, au contraire, une coloration blanche, crayeuse, comme nacrée.

Tel est, messieurs, le caractère dont il faut bien se pénétrer; car, à lui seul, quand il est manifestement accusé, il suffit pour spécifier l'amaurose tabétique et pour éclairer la situation d'une façon décisive. Il convient néanmoins de ne point négliger les troubles fonctionnels qui, eux aussi, ont bien leur importance. Ils peuvent, en effet, contribuer puissamment à établir le diagnostic, dans les cas où les symptômes ophthalmoscopiques sont peu accentués, en donnant plus de poids à l'impression ressentie par l'observateur. D'ailleurs, parmi ces troubles fonctionnels il en est quelques-uns qui, même àdéfaut des signes ophthalmoscopiques, font jusqu'à un certain point préjuger la nature du mal.

Je signalerai en premier lieu la limitation concentrique et unilatérale du champ visuel, trouble fonctionnel qui ne se retrouve pas dans la *névrite optique*; en second lieu, la contraction plus ou moins prononcée des pupilles, contraste frappant avec ce qui a lieu dans la névrite optique, où les pupilles se montrent au contraire dilatées.

Nous devons citer un symptôme qui, suivant quelques auteurs (Galezowski, Benedikt), est en quelque sorte spécifique; il s'agit d'une forme particulière d'achromatopsie caractérisée ainsi qu'il suit : 1° perte de la notion des teintes secondaires (1 et 5 de l'échelle de M. Galezowski); 2° perte de la notion du *rouge* et du *vert*; la notion du *jaune* et du *vert* persistant, au contraire, à un haut degré et pendant longtemps.

Ces signes peuvent se montrer déjà très fortement accusés, alors que la perte de l'acuité visuelle est incomplète et permet encore de lire les gros caractères.

J'ajouterai que le début de ces accidents par un œil et la localisation prolongée de la lésion, dans ce même œil, sont tout à fait l'inverse de ce qu'on observe dans la névrite optique. De plus, dans le tabes, l'évolution des troubles visuels est, dans l'immense majorité des cas, lente, graduelle, progressive, tandis que, dans la névrite optique, leur début s'opère assez souvent d'une façon à peu près subite.

Les autres troubles fonctionnels qu'il nous reste à signaler sont plutôt de nature à obscurcir le diagnostic; mais, en raison de cela même, ils méritent, eux aussi, d'être relevés. Telles sont des douleurs de tête continues ou à peu près et qui siégent principalement au front et à la nuque. A ces douleurs permanentes s'associent, dans bien des cas, des fulgurations revenant par accès et occupant le trajet des branches de la cinquième paire. Dans les paroxysmes, les malades éprouvent des sensations qu'ils comparent à celles que produirait l'arrachement du globe oculaire.

Si l'on fait abstraction des douleurs de tête, qui sont un symptôme assez banal, les phénomènes qui viennent d'être signalés constituent, dans leur ensemble, un symptôme à peu près caractéristique. Ils permettraient de distinguer aisément l'amaurose tabétique de celle, par exemple, qui accompagne la sclérose en plaques.

L'embolie de l'artère centrale de la rétine donne lieu, à la longue, à des apparences ophthalmoscopiques qui rappellent celles de la papille tabétique. Il y a toutefois des caractères distinctifs tranchés et que nous trouvons exposés dans les traités spéciaux. Le début brusque dans le cas d'embolie et la concomitance habituelle d'une hémiplégie et d'une cardiopathie ne laisseraient d'ailleurs pas longtemps subsister le doute.

Je ne ferai que mentionner en passant la lésion du nerf optique qui s'observe quelquefois dans la glycosurie et la rétino-choroïdite syphilitique, comme pouvant également reproduire, jusqu'à un certain point, l'aspect de l'atrophie papillaire tabétique. Enfin, dans la paralysie générale, on observe quelquefois une lésion de la papille qui ne diffère en rien d'essentiel de celles qui se montrent dans l'ataxie; mais nous avons eu le soin de vous faire remarquer déjà que les lésions spinales tabétiques se rencontrent quelquefois liées à la paralysie générale, et cette circonstance permet peut-être d'expliquer l'occurrence fréquente de l'atrophie papillaire progressive dans la méningite chronique diffuse.

Je me borne à signaler brièvement ces diverses affections, me proposant d'attirer toute votre attention sur les symptômes objectifs que produit l'altération du nerf optique désignée sous le nom de *névrite optique* ou de *névro-rétinite*, car là se trouve en réalité le nœud de la situation.

(*A suivre.*)

PATHOLOGIE INTERNE

De la Tuberculose. — Infiltration granuleuse du poumon

par L. THAON

D'après les travaux de Reinhardt et de Virchow, on s'accorde à ne voir dans le poumon des phthisiques que deux sortes de lésions : 1° de petites tumeurs, constituées par des nodules isolés, ou groupés ensemble ; 2° des inflammations, sous forme d'infiltrations (*gélatineuse, grise, jaune*) limitées à une partie d'un lobe, ou étendues à un poumon tout entier. Ce serait là toute la tuberculose des organes pulmonaires. Nous sommes en état d'affirmer que les infiltrations du poumon ne sont pas seulement des inflammations dégénératives; elles peuvent être aussi des granulations confluentes. C'est à l'hôpital des Enfants et à l'hôpital Laribosière qu'ont été faites nos recherches; elles portent sur 250autopsies de phthisiques. L'étude microscopique des lésions a été pratiquée par nous, dans le laboratoire d'histologie du Collége de France.

L'infiltration granuleuse du poumon peut envahir le dixième, le quart, le tiers d'un lobe, un lobe entier, ou enfin tout un poumon. Nous l'avons vu occuper une étendue notable, 8 fois 0/0 chez l'adulte, et 2 fois seulement 0/0 chez l'enfant.

Tantôt elle se montre dans la phthisie chronique, et elle prend place dans les lobes inférieurs; elle est alors secondaire; tantôt elle se montre brusquement dans les lobes supérieurs, d'où elle descend rapidement jusqu'à la base. C'est dire que l'infiltration granuleuse peut n'être que le dernier terme d'une phthisie de longue durée, ou bien constituer à elle seule toute la *maladie et le danger de cette maladie*.

Ces faits ont été présentés à la So-

ciété de biologie dans la séance du 23 novembre 1872.

Un poumon, envahi par l'infiltration granuleuse, représente un bloc lourd, qui tombe au fond de l'eau ; néanmoins ce bloc n'est pas aussi tendu qu'il le serait, s'il était frappé de pneumonie fibrineuse. La surface extérieure, à moins de pleurésie tuberculeuse, est normale, sauf quelques nodules blanchâtres non saillants, que l'on aperçoit par transparence, à travers la plèvre viscérale. — A la coupe, il ne s'écoule pas de liquide; la surface de section varie d'aspect avec l'ancienneté des lésions. Jeunes, l'infiltration est tout à fait gélatineuse, transparente, légèrement oireuse ou rosée ; plus tard on voit sortir depetits points circonscrits, arrondis, blanchâtres; ces petits points se groupent; ils deviennent opaques et enfin jaunâtres. Le nombre des groupes augmente; ils se pressent les uns contre les autres, la confluence devient extrême. — Enfin, on aperçoit des masses grises qui passent à l'état jaunâtre; mais encore à cet état, les surfaces jaunes conservent leur aspect à gros grains, et n'ont pas la surface lisse de la pneumonie caséeuse habituelle. — Lorsque la caséification est effectuée, des cavernes se creusent dans la masse dégénérée.

Ces différentes lésions peuvent se trouver réunies chez le même sujet, lorsque la marche de la maladie est un peu lente; mais on rencontre à l'autopsie des poumons qui sont encore gélatineux du haut en bas, avec des groupes assez rares de points jaunes.

Les vrais caractères différentiels entre la pneumonie caséeuse et l'infiltration granuleuse sont fournis par l'analyse histologique. Après avoir fait durcir le poumon dans l'acide picrique, la gomme, l'alcool, on peut pratiquer des coupes très minces et très larges, que l'on colore par le picro-carminate d'ammoniaque, et que l'on monte dans la glycérine (1): on voit ainsi les détails suivants. Partout où existe l'infiltration gélatineuse, on trouve les alvéoles remplis de petites cellules (anciens cytoblastions) vivement colorées, étroitement serrées les unes contre les autres; on cherche vainement à les charger avec le pinceau. — Sur les parois des alvéoles courent des capillaires, remplis de sang. Un petit nombre d'alvéoles sont tout à fait normaux; ceux-ci étaient affaissés pendant la vie, de là la tension du poumon moins grande que dans la pneumonie. D'autres alvéoles, également en petit nombre, sont remplis d'éléments épithéliaux gonflés et d'une matière colloïde, sur laquelle nous nous expliquerons plus tard.

Déjà, à cette période, on peut dire que la granulation est organisée dans les alvéoles; elle est née par bourgeonnement, aux dépens de l'épithélium pulmonaire. C'est d'ailleurs, et nous avons pu le vérifier bien des fois, le développement commun à toutes les tumeurs du poumon : lymphadénome, carcinome, sarcome(1). Il n'a fallu rien moins que des idées préconçues sur l'origine conjonctive du tubercule pour que tout le monde ait déclaré que la granulation dans le poumon naissait exclusivement à la périphérie des vaisseaux, des bronches ; il a fallu des préparations bien mauvaises pour ne pas remarquer son développement intra-alvéolaire. Au surplus, le fait a peut-être été vu, mais interprété au profit de la pneumonie caséeuse, en raison même du siége dans l'alvéole : c'est faire trop bon marché des autres caractères de la néoplasie.

Ainsi la granulation existe déjà dans le tissu gélatineux : des vaisseaux, les uns sont oblitérés par endartérite, les autres par thrombose. Il se fait des coagulations et des amas de globules blancs : tout le vaisseau coupé en travers donne des figures élégantes, représentées par un centre granuleux jaunâtre et une belle collerette de cellules rondes, vivement colorées par le picro-carminate d'ammoniaque (2). La circulation est suspendue peu à peu partout, les éléments se tassent, ne se colorent plus. On a affaire à un foyer qui contient des amas granuleux, des parois alvéolaires en voie de destruction, des sphères réfringentes graisseuses.

Les granulations et la pneumonie caséeuse aboutissent donc à l'ulcération; ce qui les caractérise, ce qui les sépare, ce sont les premiers degrés de l'évolution.

Quelle est maintenant la marche clinique de cette forme particulière d'infiltration? — Deux cas se présentent. — Ou bien la lésion est secondaire; elle survient à la dernière période de la phthisie à marche chronique : elle passe alors généralement inaperçue. Ou bien elle se montre d'emblée chez des individus bien portants; elle est primitive. Elle a, dans ces dernières conditions, une marche très nette; elle présente des symptômes facilement appréciables, mais nullement caractérisiques.

Le début est brusque, s'accompagne de frissons. On trouve à l'auscultation du souffle, des râles sous-crépitants, en un point du poumon, le plus souvent au sommet; à la percussion, on a de la matité. La matité et le souffle descendent, et tout l'organe peut être envahi, pendant que l'autre poumon se prend à son tour. — Les malades présentent un aspect typhoïde; la température est très élevée le matin et le soir : 39°5, 40°, 41°; la mort survient au bout d'un ou deux septénaires.

La maladie peut traîner un mois, six semaines; des cavernes peuvent se faire; la fièvre est alors rémittente, avec exacerbations le soir.

Voici quelques observations :

Bagot, 49 ans, sujet alcoolique, entre le 18 nov. 1872, salle saint-Henri, service de M. Guyot.

Toux depuis quelques mois. (Renseignements très vagues). Le 13 nov., accidents fébriles graves; tuméfaction douloureuse de la jambe gauche se rapportant à un ulcère enflammé. — Depuis le 15 nov., il ne se lève plus : toux, dyspnée intense. — Délire le 17 nov. — Entre le 18 : fièvre intense, langue très sèche, subdélirium; — respiration supérieure, toux pénible, dyspnée excessive. Aux deux sommets, râles sous-crépitants humides; souffle, gargouillement, matité dans le tiers supérieur de la fosse sous-épineuse; râles sous-crépitants disséminés dans toute la hauteur à droite et à gauche. — Phlegmon de la jambe gauche; traînées lymphatiques superficielles; gonflement des ganglions crureux. R. 44 P. 136 T. 39°8.

19 nov. matin. T. 38°6. — Soir : délire, faciès très altéré, teint subictérique.

Mort dans la nuit. — Autopsie le 21 nov.

Larynx : Œdème de la portion sus-glottique, petits foyers miliaires disséminés dans le larynx, la trachée, les grosses bronches.

Poumons : Les deux tiers supérieurs du poumon gauche sont envahis par une masse à fond brunâtre; sur ce fond tranchent de petits points, très confluents, tout à fait transparents; ces points deviennent blancs en certains endroits; et, enfin, tout à fait jaunes; ils constituent alors des îlots jaunes à gros grains. Les mêmes lésions existent à gauche, mais moins avancées et moins

endues. — L'étude histologique montre les alvéoles envahis par les granulations : à peine quelques-uns d'entre eux sont libres ou envahis par de la pneumonie épithéliale. — (Communiquée par M. Homolle.)

Obs. II. — Legrand Victor, pâtissier, entre le 1er août 1872, service de M. Woillez. Bien portant, âgé de 20 ans. Il est malade [illegible]

(1) Ce qui rend l'étude de la tuberculose pulmonaire très confuse, c'est la grande variété des lésions dans un organe aussi compixe ; mais aussi on doit un grand nombre des erreurs répandues dans la science au mode insuffisant de préparation. — Il ne faut plus parler de la méthode de dessèchement, trop longtemps employée; l'alcool et l'acide chromique sont excellents, mais le premier ne fixe pas assez les éléments, le second les rend vésiculeux, donne du réticulum partout. Nous avons adopté la technique du laboratoire de M. Ranvier : acide picrique, gomme, alcool et le picro-carminate si précieux pour ses affinités.

(1) *Traité d'histologie pathologique*, t. I. Tubercule, par Cornil et Ranvier. — Ces deux histologistes ont vu le tubercule se développer aux dépens des cellules épithéliales du corps thyroïde.

(2) Les Allemands prennent ces vaisseaux oblitérés pour des éléments anatomiques — Voir le *Mouvement médical* du 23 nov. 1872.

depuis quinze jours : début brusque avec toux, sans frisson, sans point de côté, quelques crachats muco-purulents; pas d'épistaxis ni de céphalalgie, pas de diarrhée. Est resté au lit avec soif vive et fièvre.

1er *août, soir.* — P. 120, T. 40°1 Face cyanosée, dyspnée très prononcée, toux. — Langue humide, blanche. — En avant, à droite, submatité et râles sous-crépitants; à gauche quelques râles au sommet; en arrière, à droite, matité au sommet, avec souffle, râles sous-crepitants dans tout le poumon; à gauche quelques râles au sommet, voix retentissante.

2 août. P; 12 T. 40°2 — 20 ventouses sèches, kermès, vésicatoire. — Soir : P. 112 T. 41°

3 août. P. 108; T. 40°8, moins de dyspnée. En arrière, à droite, souffle avec gros râles crépitants au sommet, râles sous-crépitants dans la moitié inférieure; en avant, râles sous-crépitants avec souffle et timbre caverneux. — Soir : P. 110; T. 40°8.

	Matin.	*Soir.*
4 août.	40°2	41°2.
5 —	40°2	40°5.
6 —	40°3	40°9.
7 —	39°7	40°9.
9 —	40°8.	
10 —	40°8	41°4. Langue sèche, respiration soufflante et caverneuse à droite; le souffle descend presque jusqu'à la base; état de prostration et de cyanose.
11 août.	40°4	41°
12 —	40°	41°2. Pas de râles à gauche.
13 —	40°4	Mort dans le calme, au milieu de la nuit. Autopsie, le 15, soir.

Le poumon droit de haut en bas, forme un bloc, contenant quelques points caverneuleux au sommet des granulations jaunes disséminées, et un fond gélatineux. Le poumon gauche est emphysémateux, œdémateux. Il contient un noyau de la grosseur d'une noix, de même aspect que le poumon droit et plaie au sommet. — L'étude histologique a montré les lésions caractéristiques de l'infiltration granuleuse.

Nous avons recueilli un certain nombre d'autres observations, où les lésions étaient arrivées jusqu'à la formation de cavernes; enfin, la plupart de ces lésions ont été trouvées à l'autopsie d'individus morts de phthisie chronique ordinaire.

ut à fait à sa naissance, elle a été entre-
ise par MM. Lartet, Mayer de Bonn, Bro-
, etc. Elle s'est bornée à enregistrer, jusqu'à
ésent, quelques traces de traumatisme
os, et une altération encore mal déter-
inée, particulière à l'*arsus*.

M. Rabuteau a cherché à s'expliquer
ourquoi les lunettes bleues sont tolérées
ar les gens qui craignent la lumière ordi-
aire ou la lumière jaune. Pour lui la rai-
on est toute physique : les ondes de pro-
agation, appartenant à la couleur violette,
nt une longueur bien moindre que celle
e la couleur rouge ; partant, le violet est
oins intense que le rouge et impression-
era moins désagréablement les rétines en
at d'hypéresthésie.

M. Malassez, à l'aide du capillaire artifi-
iel qu'il a présenté à la Société dans une
éance précédente, a cherché à se rendre
ompte des variations physiologiques des
lobules rouges dans les différentes espèces
nimales.

Le nombre des globules rouges est loin
'être le même, selon les espèces animales.
l est très considérable chez la chèvre, qui
18 millions de globules rouges dans un
illimètre cube de sang ; — chez le chameau
t le lama, il est d'environ 10 millions : —
hez le cheval, il est de 6 millions, — chez
âne, de 5 millions ; — chez les oiseaux, le
ombre est moins grand, il dépasse rare-
ent 3 millions, excepté chez le pigeon ; —
ez les poissons, on remarque un fait cu-
eux : les poissons osseux ont beaucoup
lus de globules que les poissons cartila-
ineux, et on pourrait par ce seul carac-
ère, différencier des individus appartenant
ces deux espèces. Chez les poissons carti-
agineux, le nombre des globules rouges
lescend jusqu'à cent cinquante mille.

Chez l'homme, il y a écart physiologique
ntre 3 millions et demi et cinq millions.

M. Malassez s'est demandé si l'abaisse-
ment dans le chiffre des globules était
compensé par les dimensions plus grandes
de ces éléments. — En effet, on constate
que le volume des globules est en raison
inverse de leur nombre, mais ce rapport
ne suffit pas à rétablir l'équilibre, et on
peut considérer comme établi, que chaque
espèce animale est caractérisée par une
proportion différente du nombre des glo-
bules.

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ANATOMIE ET PHYSIOLOGIE

PATHOLOGIQUES

Des Lésions vasculaires dans la tuberculose

par **L. THAON**, interne des hôpitaux de Paris

Ces lésions se retrouvent :

A dans les tuniques du vaisseau ;

B dans la lumière du vaisseau ;

C dans une certaine zone autour des foyers tuberculeux.

A. — Que les granulations se développent de préférence autour des vaisseaux, cela ne fait de doute pour personne. Dans les poumons, le foie, les reins, la langue, le cerveau, partout on peut vérifier cette loi : pour la pièce et l'épiploon le fait saute aux yeux.

Ces phénomènes se passent à peu près de la façon suivante. L'*adventice* des vaisseaux est infiltrée de petits éléments arrondis qui naissent par génération endogène aux dépens des cellules conjonctives de cette tunique. La première enveloppe se transforme ainsi peu à peu en tissu de granulation. — La *tunique moyenne* à son tour peut subir deux sorts un peu différents ; ou bien les fibres musculaires lisses qui la composent disparaissent par atrophie : dans le cas le plus fréquent ; ou bien elles prennent part au développement de la tumeur, elles se segmentent, chaque segment donnant lieu à une cellule embryonnaire. M. Durante, qui le premier a bien décrit la segmentation des fibres-cellules dans l'inflammation des artères, produite expérimentalement, nous a montré des préparations qui rappellent cette même segmentation dans la tuberculose. Pour vérifier ce fait, on choisira de préférence les artères de la pie-mère, dont les fibres lisses sont bien distinctes : on pourra d'ailleurs, en hiver et peu de temps après la mort, leur appliquer les procédés d'imprégnation au chlorure d'or.

Que devient la *tunique interne* ? Plusieurs cas peuvent se présenter.

1° Cette tunique peut disparaître avec la tunique moyenne sans opposer de résistance. Ce fait se remarque assez souvent pour le poumon : les artères sont emprisonnées dans un manchon de granulations, elles sont aplaties, effacées, les tuniques interne et moyenne ne recevant plus de suc nutritif ni de l'intérieur ni de l'extérieur, s'atrophient. Ce fait est commun à toutes les tumeurs du poumon et même à la pneumonie chronique.

2° Le plus souvent la tunique interne s'enflamme de très bonne heure : il y a endartérite, aux dépens de l'endothélium et des cellules plates de Langhans. Cette végétation marche concentriquement, ou s'avance par bourgeons, enveloppe le caillot ou le pénètre, si une coagulation du sang a eu lieu déjà : l'oblitération de l'artère est ainsi effectuée. C'est encore un phénomène qui n'a rien de spécial à la tuberculose : dans le poumon, il survient à propos de toutes les tumeurs et de la plupart des inflammations. Et cependant, on ne peut pas nier qu'il ne soit plus constant sur la tuberculose, dans la pneumonie caséeuse que partout ailleurs ; qu'il ne s'ajoute aux autres causes pour amener la caséification des produits ; que l'on ne doive l'envisager comme quelque chose de providentiel, puisqu'il met un bon tiers des phthisiques à l'abri des hémorrhagies. Dans ces espèces de cordages qui traversent une caverne tuberculeuse, ou qui sont appliqués sur sa paroi, à l'instar des colonnes charnues du cœur, on retrouve les vaisseaux oblitérés par endartérite.

3° Enfin dans les artères de plus petit calibre, dont la tunique interne est réduite à peu près à la couche endothéliale, l'endartérite produit peu ou point de végétations ; mais l'oblitération est encore assurée : elle l'est par la coagulation du sang.

B. — La coagulation du sang, qui est fréquente dans les tissus irrités, est constante dans la tuberculose. On peut suivre pas à pas ce phénomène de la coagulation, jusqu'à ce que le vaisseau, pris dans une granulation, soit rempli de fibrine et de globules blancs accumulés à la périphérie du bouchon fibrineux.

On peut même dire qu'à ce moment la granulation est complète, qu'elle est à sa période d'état, qu'elle a tous ses caractères. Coupée en travers et colorée par le picro-carminate d'ammoniaque, elle se présente avec une zone périphérique, composée de cellules assez grandes à plusieurs noyaux; d'un centre où les éléments sont de plus en plus petite, et arrivent à se fondre; de vaisseaux oblitérés, représentés par un fond granuleux, un peu jaunâtre, entouré d'une belle collerette de cellules, vivement colorées en rouge, ce sont les leucocytes restés accumulés et adhérents à la paroi. A une période plus avancée, la granulation a perdu son aspect; elle est dégénérée; on ne peut plus lui reconnaître de caractères particuliers.

Ces faits paraissent fort simples, ils ont été vus par tous ceux qui n'étaient pas imbus d'idées préconçues: MM. Ranvier (1) et Cornil (2) les ont décrits; ils ont même donné le moyen de les réaliser expérimentalement.

« Il y a accumulation de globules blancs, le long des parois vasculaires, toutes les fois que le cours du sang se ralentit. Cette proposition incidente résulte d'expériences que nous avons faites sur la membrane interdigitale de la grenouille. Lorsque, en effet, on y pratique une incision, les vaisseaux capillaires oblitérés au niveau de l'incision forment des culs-de-sac, où la circulation se ralentit. Ces espèces de diverticules où le sang séjourne, se remplissent de globules blancs, qui y demeurent, parce que l'impulsion du sang n'est plus assez grande pour lutter contre les propriétés adhésives de ces globules. — Comme, d'un autre côté, les globules blancs ne sont pas dans la tuberculose plus nombreux qu'à l'état normal, nous serons conduits à admettre que le ralentissement de la circulation témoigné par l'accumulation des globules le long des parois des vaisseaux a précédé la formation de la fibrine et l'arrêt de la circulation dans les tubercules. »

Cependant ces vaisseaux, remplis d'un détritus granuleux, entouré d'une collerette de globules blancs, ont été pris pour des éléments anatomiques, pour des *cellules gigantesques*, des *Riesenzellen* (3), par E. Wagner(4), et elles ont été figurées comme telles, dans l'atlas d'un de ses préparateurs Thierfelder (1). Or, comme ces cellules gigantesques se rencontrent également, d'après Förster, dans les ganglions lymphatiques, il a semblé naturel à M. E. Wagner de s'en faire un argument de plus pour établir que le tubercule est formé de *tissu lymphatique*.

Nous nous proposons de combattre plus tard cette manière de voir sur le tubercule : pour le moment nous voulons signaler seulement cette erreur qui consiste à prendre un vaisseau oblitéré pour une cellule gigantesque, remplie de plusieurs noyaux. Le protoplasme de la cellule correspondrait à la fibrine granuleuse, les noyaux multiples aux leucocytes de la zone périphérique. Mais toujours ces cellules rondes sont disposées en collerette à la périphérie; si elles paraissent empiéter sur le centre, c'est que l'on aperçoit également celles qui sont situées sur un plan plus profond; avec un objectif à grand angle d'ouverture on arrive à corriger cette erreur d'optique. Ce qui a pu tromper encore M. E. Wagner et les autres, c'est qu'à cette période on ne voit plus généralement ce paroi à ces vaisseaux oblitérés, la granulation s'est substituée aux tuniques normales; elle est là, entourant de toutes parts la prétendue cellule gigantesque. D'ailleurs, il ne manque pas de vaisseaux, et si l'on trouve les parois encore distinctes, alors que la coagulation de la fibrine et l'accumulation de globules blancs sont déjà effectués : ce sont les vrais points de repère.

Le fait constant dans la tuberculose est donc l'oblitération des vaisseaux, que celle-ci survienne par endartérite ou par coagulation du sang. On trouve à cet égard une différence complète entre la syphilis et la tuberculose, deux maladies qui ont plus d'un point de ressemblance. Dans la syphilis, au milieu des gommes dégénérées, des foyers caséeux, la circulation s'effectue encore; les vaisseaux sont perméables; dans la tuberculose, le cours du sang est suspendu, alors que la granulation est encore près du début. Que faut-il en conclure? c'est que la dégénérescence des produits n'est pas subordonnée fatalement à l'arrêt de la circulation; les néoplasies dans la syphilis et la tuberculose sont vouées à la mort, parce que ce processus est dans leur nature.

C. — Le champ de la circulation se trouve singulièrement rétréci dans un organe, le poumon, par exemple, lorsque la tuberculose est étendue. Les capillaires restés sans fonction sont détruits avec les parois des alvéoles; le sang qu'ils contenaient se décompose; la matière colorante abandonne les globules; elle passe à l'état de granulations libres; celle-ci pénètrent dans les lymphatiques et arrivent, portées par les cellules migratrices, jusque dans les ganglions bronchiques, généralement anthracosés dans la tuberculose.

D'autres fois, dans les capillaires non encore détruits, il se fait des accumulations de leucocytes, par suite du retard de la circulation : ces amas de leucocytes finissent par former une zone blanchâtre autour du vaisseau en voie d'oblitération. Nous en avons vu dernièrement un très bel exemple dans le foie d'un tuberculeux. Cet organe était parsemé à sa surface d'une foule de petits îlots arrondis, blanchâtres; à la coupe, il était parcouru de lignes blanchâtres, rameuses, se détachant manifestement d'un vaisseau environné lui-même d'une virole de même coloration. Or, ces lignes et ces îlots qui tranchaient vivement sur le fond rouge du foie n'étaient autre chose que des leucocytes accumulés dans les capillaires du lobule hépatique et formant une zone autour des vaisseaux envahis par des granulations. C'est encore un phénomène dû au ralentissement de la circulation; les globules blancs s'entassent là comme dans le poumon d'une grenouille, que l'on fait sortir à travers une boutonnière pratiquée au niveau de la cavité abdominale de l'animal.

Le sang, arrêté par les vaisseaux oblitérés, cherche sa voie ailleurs : dans le poumon, il quitte la petite circulation générale (M. Guillot, Schroeder van der Kolk). On voit, en effet, autour des foyers de pneumonie caséeuse, autour des granulations, se former du tissu fibreux. Dans ce tissu se creusent des vaisseaux de nouvelle formation, tapissés d'une couche de cellules embryonnaires : ces vaisseaux sont en très grand nombre, ils s'abouchent par les adhérences pleurales avec la circulation générale.

Mais tissu fibreux et vaisseaux n'ont qu'une existence temporaire; ils subissent les phénomènes de la rétraction; ils s'atrophient comme une cicatrice. A leur place, on ne trouve plus tard qu'une travée grisâtre, infiltrée de sels calcaires.

Nous pourrions conclure de tout ce que nous avons dit que :

1° L'oblitération des vaisseaux est un fait constant dans la tuberculose;

2° Que cette oblitération se fait par coagulation sanguine, ou par endartérite végétante;

3° Que de nouveaux vaisseaux se forment souvent au voisinage.

(1) *Arch. de physiologie*, 1868. Ranvier : *Tubercules des os.*

(2) *Manuel d'histologie pathologique*, par Cornil et Ranvier. T. 1, pag. 203.

(3) *Archives de Virchow*, tom. 42. — Langhan.

(4) Wagner (E.). *Ueber tuberkelähnliche lymphadenom.* (Ues cytogene oder reticulirte Tuberkel. (*Archiv. der Heilkunde*, Heft. 6 Taf. VI et VII.

(1) *Atlas de Thierfelder*, 1872.

De la granulation dite de Bayle et de la phthisie granuleuse chronique.

Mémoire lu à la Société de Médecine, 25 janvier 1875

Par L. THAON (1).

On attribue communément à Bayle la découverte de la granulation tuberculeuse; on sait qu'il a créé une variété spéciale de phthisie, appelée phthisie granuleuse.

La granulation de Bayle serait donc la granulation vulgaire, celle que Virchow a si bien définie histologiquement, et la phthisie granuleuse équivaudrait à ce que M. Empis a dénommé granulie, c'est-à-dire à une maladie à marche très-rapide, à une phthisie aiguë. C'est une erreur qui s'est perpétuée depuis Laënnec jusqu'à nos jours; la granulation de Bayle n'est pas la granulation ordinaire; sa phthisie granuleuse est une phthisie chronique et n'a rien à voir avec la granulie.

La granulation de Bayle était miliaire, transparente, luisante de nature et de consistance cartilagineuse, ne devenant jamais opaque, et ne se fondant jamais. La granulation ordinaire, celle de Laënnec, est une petite nodosité dure, saillante, d'abord transparente, mais devenant bientôt opaque et jaunâtre à son centre.

La Phthisie granuleuse de Bayle, comme on pourra en juger d'après les observations 4, 14, 15, 22, 31 de son livre est une affection de longue durée qui a plusieurs périodes, comme toute autre phthisie. Dans la granulie, au contraire, les malades sont pris rapidement, succombent en peu de temps après avoir montré des phénomènes de suffocation considérable, ou des phénomènes typhoïdes; à leur autopsie on trouve le poumon farci de petites granulations, déjà jaunes au sommet, encore rosées vers la base; les autres organes, ainsi que les séreuses contiennent généralement ces granulations à leurs anciens états.

Entrons maintenant dans quelques détails anatomiques et

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histologiques sur la *granulation de Bayle* et sur sa *Phthisie granuleuse.*

Sur le plus grand nombre d'adultes qui succombent à la phthisie tuberculeuse chronique, on trouve dans les poumons les lésions suivantes: au sommet une ou plusieurs cavernes de dimensions variables; autour d'elles une masse plus ou moins étendue de pneumonie chronique, reconnaissable à son aspect transparent, à sa coloration plus ou moins ardoisée, à sa consistance scléreuse: le reste du poumon est envahi par une série de gros noyaux, déjà appréciables extérieurement par le toucher et qui, sur la coupe, se montrent comme des *grappes* fort élégantes; chaque grappe est composée d'un nombre variable de grains qui ne sont autre chose que les granulations de Bayle Ces grains sont d'une consistance de grêle, un volume de la graine de chénevis, très-saillants, d'une transparence notable ou bien jaunâtre au centre. A mesure que l'on descend vers la base, les grappes sont moins nombreuses et moins ramifiées, et on arrive à ne trouver que des grains solitaires. Le tissu pulmonaire compris entre les grains est habituellement sain à une première période, mais bientôt il est envahi par un tissu grisâtre, sclérosé, par de la pneumonie interstitielle qui comble tous les vides, et l'ensemble des granulations et du tissu inflammatoire forme un noyau saillant plus élevé de 0mm 5 à 1mm au-dessus du reste du poumon. Ces noyaux sont superficiels ou profonds: superficiels, ils affectent la forme de cones à base tournée vers la plèvre. Ces cones peuvent devenir confluents, se fondre ensemble, et l'on rencontre des poumons envahis de haut en bas par ces lésions; on ne sait vraiment où pouvait se faire la fonction respiratoire, et on s'explique difficilement la survie si longue des malades. Toutes ces lésions sont essentiellement chroniques, aussi sont-elles bien moins fréquentes chez l'enfant que chez l'adulte.

Voyons ce que nous apprend l'étude histologique de semblables poumons, durcis dans l'ac. picrique, la gomme, l'alcool, et après coloration au picro-carminate d'ammoniaque. Les granulations tout à fait transparentes se montrent à nous, comme composées de tissu fibreux parfait, organisé en couches concentriques, séparées par des cellules de tissu conjonctif un peu gonflées et contenant quelques granulations pigmentaires. Les granulations qui offrent deux zones, l'une transparente et l'autre jaunâtre, renferment des éléments dégénérés dans les points caséeux et du tissu fibreux dans la zone transparente. La zone jaunâtre diminue de plus en plus, et on voit le tissu fibreux empiéter sur elle; le détritus caséeux est pris par les espaces conjonctifs du tissu fibreux qui rayonnent vers le centre; à un moment donné la granulation est fibreuse dans son entier, elle est transparente dans toute son étendue. De sorte que non-seulement on peut dire que la granulation de Bayle ne perd pas sa transparence avec l'âge, mais on peut soutenir que cette transparence ne fait que s'accuser à mesure que la granulation s'organise en tissu fibreux dans toute sa

lation de Bayle qui ne tient pas à la présence du tissu fibreux je veux parler de l'état cireux de la granulation. A un moment donné, les éléments dégénérés, placés au centre, s'agglutinent, se fondent avant de passer à l'état jaunâtre, et la petite masse prend alors une transparence qui se perd avec celle du tissu fibreux.

On peut se demander quel est le mode de développement des granulations fibreuses de Bayle. Il nous a été facile de constater ce développement sur une foule de préparations: nous avons vu des granulations ordinaires, occupant un, deux ou plusieurs alvéoles, s'entourer à un moment donné d'un tissu sarcomateux à cellules fusiformes, né de l'épithélium des alvéoles voisins; à ce tissu s'ajoute une matière interfibrillaire, et la préparation simule alors grossièrement du tissu réticulé, surtout si elle a été précédée de macération dans l'acide chromique. Bientôt ce tissu sarcomateux s'organise, devient fibreux et alors on a, en marchant de la périphérie au centre: des alvéoles un peu affaissés, une coque de tissu fibreux et un amas d'éléments dégénérés; le tissu fibreux s'avance toujours vers le centre et, à la dernière période on n'a plus affaire qu'à un fibrome. Nous avons donc eu une granulation ordinaire qui par irritation a développé une zone fibreuse d'enkystement; quelquefois cette irritation se poursuit et il se fait de la pneumonie interstitielle qui réunit les petits foyers primitifs. En somme, la lésion diffère peu de ces nodules, signalés par différents auteurs, et en particulier par Zenker, chez les ouvriers qui manient l'oxyde de fer, et sont soumis à l'inhalation de cette poussière minérale. Le poumon dans ces circonstances est infiltré aussi par des nodosités de la grosseur d'une tête d'épingle jusqu'à celle d'un pois, arrondies, dures, d'un gris transparent. L'agent irritant, dans ce cas, est une poussière minérale; dans la phthisie chronique c'est une granulation tuberculeuse, un petit foyer caséeux, miliaire.

Ceci nous amène à nous demander quelle loi suivent ces petits foyers dans leur répartition au sein du poumon? La réponse nous est donnée par la disposition si nette des granulations de Bayle; cette disposition donne lieu aux grappes les plus élégantes, ces grappes suivent exactement la distribution d'un rameau bronchique et sont d'autant plus étendues, que le rameau bronchique lui-même est plus considérable; chaque granulation occupe un infundibulum entier, ou une partie de l'infundibulum, et il n'est pas rare de voir la petite bronche qui aboutit à la grappe, remplie de matière caséeuse. Plus tard lorsque la pneumonie interstitielle survient, elle se limite très-exactement au territoire desservi par la bronche, et c'est ainsi que l'on a des cônes très-saillants, très-exactement délimités, à base tournée vers la surface pleurale. On peut supposer pour expliquer la succession de ces lésions, que les altérations tuberculeuses qui ont débuté au sommet, sont les centres d'infection; il en part à chaque instant des particules, entraînées par la respiration, qui arrivent dans une petite bronche et là s'arrêtent dans les alvéoles qui terminent un canalicule respirateur, y développent une granulation et consécutivement une zone fibreuse qui suffirait pour guérir ce petit foyer local, si d'autres foyers ne se développaient au voisinage. Le champ respiratoire à la longue finit par être occupé en entier par les granulations de Bayle, par les grappes, par la pneumonie interstitielle. C'est la même série de phénomènes qui se passe chez les gens qui sont soumis aux inhalations de poussières; on a encore des granulations fibreuses et de la pneumonie interstitielle, et ce sont les bronches aussi qui sont la voie de transport des irritants.

Ceci nous montre encore que ce qui fait le danger de la tuberculose, ce n'est pas l'existence d'une tumeur essentiellement maligne, appelée granulation tuberculeuse et qui agirait à la manière du cancer. Au contraire, la granulation est susceptible de s'enkyster, comme une parcelle de fer ou de silex: ce qui rend la tuberculose une maladie terrible, c'est l'apparition de ces granulations dans un grand nombre de points à la fois et cela dans des conditions que nous ne pouvons nous expliquer (*phthisie aiguë*); ou bien la formation lente mais continue de ces granulations, jusqu'à l'envahissement entier d'un organe (*phthisie chronique*): à peine une de ces granulations vient de s'enkyster, vite il s'en fait une autre.

La granulation de Bayle a été entrevue par Virchow: cet auteur l'appelle *tubercule fibreux*; et plus loin, il la confond avec la *péri-bronchite* chronique. Rindfleisch en fait de la *lymphangine noueuse*, et il donne une figure qui s'applique très-bien à la lymphangite tuberculeuse du mésentère, mais qui n'a rien à voir avec les lymphatiques du poumon. L'observation d'un fait remarquable de lymphangite primitive du poumon, recueillie par M. Raynaud à sa clinique de l'hôpital St-Antoine nous avait rendus attentifs aux lésions des lymphatiques pulmonaires dans la phthisie; mais nous n'avons jamais rien vu qui pût leur être rapporté. Que dirons nous encore de l'opinion de E. Wagner qui considère ces tubercules fibreux comme composés de tissu lymphatique, nous avons réfuté plusieurs fois cette manière de voir et montré qu'elle n'avait pas de raison d'être.

Nous proposons de réserver le nom de *Granulation de Bayle* à ces granulations qui sont l'apanage de la phthisie chronique et qui par leur structure, s'éloignent de la granulation ordinaire.

inférieure du cerveau, vers la partie moyenne de la [illegible] Bichat, naissent deux artères qui se portent en avant sur les côtés de la glande pinéale : artères de la *toile choroïdienne et des plexus choroïdes du 3me ventricule.*

L'artère des plexus choroïdes des ventricules latéraux, née de la carotide, se place en dehors de la bandelette optique, fournit, chemin faisant, quelques ramuscules à la bandelette, au pédoncule qu'elle contourne, aux circonvolutions voisines, et vient à la partie la plus externe de la fente de Bichat, dans la corne sphénoïdale pénétrer le plexus choroïde. On peut la suivre dans toute la longueur de celui-ci jusqu'au voisinage du trou de Monro, où elle se termine dans la tête du corps strié. Elle fournit : *a*) des rameaux inférieurs à la face de la couche optique située au-dessous d'elle ; *b*) des rameaux externes à la tête et à la queue du corps strié. Dans un cas, où ils étaient très-nombreux, on les voyait s'épanouir en rayonnant sous l'épendyme qui recouvre la tête du corps strié ; j'ai constaté deux fois l'anastomose de ces branches avec celles de la sylvienne qui perforent de bas en haut le corps strié ; mais ces anastomoses étaient peu importantes. Un petit rameau occupe généralement le sillon optico-strié au-dessus de la lame cornée. *c*) Des rameaux supérieurs aux piliers postérieurs du trigone. Ces mêmes branches pénètrent dans le corps calleux. Toutes ces artères sont très-multipliées et de petit volume (un quart de millimètre environ). *d*) Très-souvent cette artère fournit les rameaux des corps genouillés.

L'artère de la toile choroïdienne des ventricules latéraux vient de la cérébrale postérieure. Elle se porte en avant et un peu en dedans. Son volume est en raison inverse de celui de [illegible]

inflammatoires décrites sous le nom de végétations en chou-fleur et en crête de coq ; la difficulté du diagnostic est même si grande alors, que certains auteurs pensent qu'il est impossible de dire si ces végétations sont inflammatoires ou cancéreuses.

On rencontre encore du côté du col des ulcérations dépendant de la syphilis, ce sont les chancres et les plaques muqueuses. Le *chancre mou*, qui ressemble assez à l'ulcération simple, est caractérisé par des bords taillés à pic, à fond grisâtre. Le *chancre induré*, bien que rare, a cependant été constaté par M. Ricord, dans un cas où le col faisait saillie en dehors de la vulve

M. Bernutz a signalé encore une troisième variété de chancre qu'il a décrite sous le nom de chancre *diphthéritique*, et dont la surface saignante, un peu boursouflée, est recouverte d'une sorte de membrane.

Le siége des chancres est assez différent de celui de l'ulcération simple ; il ne siége pas ordinairement au pourtour de l'orifice du col, mais sur un point variable de la surface externe du museau de tanche ; Marjolin a fait remarquer qu'il siégeait le plus souvent vers la partie postérieure du col au point de réflexion du vagin Bien que le chancre siége le plus communément sur un point plus ou moins éloigné de l'orifice du col, il se rencontre quelquefois au pourtour de cet orifice. Je me demande si cette variété ne dépendrait pas de ce qu'il y aurait ulcération préexistante et inoculation par cette ulcération.

Les ulcérations secondaires que l'on rencontre du côté du col, sont les plaques muqueuses qui se présentent sous l'aspect d'une légère saillie d'un blanc nacré et brillant.

Nous arrivons maintenant au traitement des ulcérations, et nous devons nous demander quel traitement particulier on doit diriger contre elles. Ce que je puis dire ici, c'est que l'ulcération n'étant qu'un des éléments de l'inflammation, c'est surtout à la métrite chronique que devront s'adresser les divers agents thérapeutiques. Néanmoins, il est avantageux de pratiquer quelques cautérisations de la surface ulcérée qui déterminent en même temps des modifications utiles du parenchyme. C'est ainsi qu'on devra avoir recours, dans les cas légers, à des cautérisations avec une solution de nitrate d'argent au quart, avec de la teinture d'iode, ou avec du perchlorure de fer, surtout quand les surfaces ulcérées sont un peu saignantes. Quand l'ulcération devient végétante, on se trouvera bien de caustiques plus énergiques, tels que l'acide chromique, le nitrate [illegible] négliger de

acide de mercure ; mais, alors, il ne faudra pas négliger de faire, avant d'enlever le spéculum, une injection d'eau dans le but d'enlever l'excès du liquide et de l'empêcher de produire la cautérisation des parties voisines. Quant à la cautérisation au fer rouge, elle sera surtout utile quand le col de l'utérus est volumineux.

On ne devra pas négliger non plus l'emploi des injections légèrement astringentes, dans le but de déterger la surface ulcérée, et d'enlever les produits de la suppuration. C'est alors que les poudres absorbantes, telles que le bismuth, ou l'amidon mélangés à l'alun pourront être utiles.

par la voie des ventricules.

M. VULPIAN attribue l'insensibilité complète du membre supérieur chez le malade de M. Lépine, à la lésion du pédoncule cérébral, plutôt qu'à la couche optique.

M. LIOUVILLE, croit que l'éjaculation du sperme, constatée par M. Lépine, est un fait très-fréquent chez les malades qui succombent brusquement à une affection des centres nerveux elle se remarque aussi dans les attaques d'épilepsie, et M. Liouville l'attribuerait volontiers à l'état d'asphyxie dans lequel sont tous ces malades.

M. LÉPINE répond que son malade ne présentait pas de phénomènes d'asphyxie, et l'éjaculation a eu lieu bien avant la mort, puisque elle a été constatée dès l'entrée du malade.

M. BERT a continué ses recherches sur *l'action de l'oxygène* à une forte pression sur les plantes et sur les animaux ; il expose à la société les nouveaux résultats qu'il a obtenus. Il a remarqué que la *Germination* était impossible à une pression de 8 atmosphères, à 2 atmosphères elle est languissante ; à 4 atmosphères, il n'apparait que la radicule, la gemmule ne se montre pas. Voilà le phénomène tel qu'il se passe pour les graines qui renferment un albumen farineux considérable; pour les graines qui en possèdent très peu, telles que les graines de moutarde, le phénomène est vrai également, mais il est moins accusé. — Non-seulement la germination est empêchée, mais les graines qui ont été soumises à cette épreuve perdent à jamais le pouvoir de germer, même placées dans les conditions les plus favorables. La composition de la graine est profondément modifiée, et on peut s'en assurer d'après l'odeur alcoolique qui s'échappe des réservoirs, alors que l'on fait la décompression ; il est probable que la fécule au lieu de se transformer en produits qui donnent lieu au développement d'acide carbonique, favorable à la plante, se transforme en alcool : des expériences plus précises devront éclaircir ce dernier point. — Quelle est la cause de ces troubles et de cet arrêt dans la germination ? Cette cause n'est pas dans la compression de l'air, mais dans l'action toxique de l'oxygène, contenu en excès dans les atmosphères artificielles; on pourra s'en assurer en employant une compression aussi forte, mais avec des gaz indifférents. Il est donc inexact de dire que l'oxygène est très-favorable à la germination ; il ne l'est que dans des proportions déterminées.

M. BERT arrive à un second fait : il a cherché par divers moyens à empêcher la mort ou les accidents terribles qui surviennent chez les animaux que l'on décomprime brusquement; on sait que des animaux décomprimés de 8 atmosphères succombent infailliblement, ceux qui sont décomprimés à 4, 5, 6 atmosphères, sont frappés de paraplégie. Il en est de même chez l'homme, les accidents de décompression sont fréquents et ils le seraient bien davantage, si l'on ne tenait à les laisser ignorer, dans le but de ne pas effrayer les ouvriers. Un premier moyen consisterait à récomprimer les animaux ; un autre consisterait à éliminer les bulles d'azote qui se sont développées dans les vaisseaux et qui obstruent la circulation. Ces bulles n'ont aucune tendance à s'échapper, parce que l'air du poumon contient aussi un excès d'azote, qui empêche l'échange : dès lors il fallait recourir à l'insufflation d'air, très-chargé d'oxygène. En agissant très-rapidement, M. Bert est parvenu en effet à sauver les animaux d'une mort certaine, à l'aide de ce moyen ; mais il y a des accidents qui persistent toujours, et particulièrement la paraplégie; néanmoins on a paré au premier danger et on peut espérer triompher même de la paraplégie.

MM. CHARCOT, CARVILLE, COTARD, ONIMUS se demandent quelle peut être la cause de la paralysie dans ces conditions, et ils cherchent à se rendre compte pourquoi les fonctions du

Шевелевъ.

ст. Лешинскій собств.

p. 13. Pour l'anatomo-pathologiste pas, il est difficile de ne pas convenir à deux maladies différentes. – – p. 13.

Il y a là dit-on 2 malad. différentes, l'une spécifique, d'abord héréditaire, l'autre n'a rien de tout cela. p. 18.

Pour Virchow – La granulie tuberculeuse est une néoplasie – – –

La granulat. est d'origine conjonctive, la tub. caséeuse est de nature épithéliale.

— Le tubercule n'est pas toujours nodulaire — tubercule infiltré, alvéolaire.

p. 21. J'accepte que le microscope seul est insuffisant à distinguer toujours le tubercule des produits d'inflammat. simple – mais quand on ne veut pas se contenter d'un examen histologique et qu'on réunit les notions cliniques, et celles d'une autopsie soigneusement faite+, à celles que fournit le microscope, il est toujours facile de le décider.

+ et il cherche le tubercule dans les viscères.

p. 24. Leur origine ne serait pas aussi nettement qu'on le dit le tubercule de la pneumonie caséeuse. – le tubercule naissant lui aussi de l'endothélium

p. 32. Le tubercule siège tout aussi bien que les [illegible] caséeux dans l'alvéole.

38. Virchow sépare tout le tubercule de l'inflammation.

– or la granulat. tuberculeuse est le résultat d'une irritation nutritive et même formative, ce qui le ramène à la définit. de l'inflamm. d'après Virchow lui-même.

Cependant Virchow dit 55 pas. (Path. des tumeurs T. III. p. 71) que les granul. tub. se rapprochent beaucoup du pus dont elle possède les petits noyaux et les petites cellules.

Pour Virchow d'ailleurs toutes les productions cellulo-[illegible] qui ne sont pas nodulaires tombent dans l'inflammation simple – Il en est qui il s'agit [de] l'alvéolite, de tubercule, mais c'est à tort.

— p. 42. quel que soit le mode de début d'une inflammat. pulmonaire, si celui qui la porte la rend caséeuse, il est bien près de devenir tuberculeux, et de fait il l'est réellement.

– Il y a il est vrai une caséification accidentelle.

. Les infl. tuberculeuses se caséifient régulièrement.

– ainsi la caséification est ordinairement presque une [illegible] tuberculeux.

Splénisation

L'altération anatomo-pathologique qui caractérise la bronchopneumonie est ce que l'on a décrit sous le nom de Splénisation.

Et peut-être Sarcone l'a-t-il employé en disant à propos d'un cas sans doute de bronchopneumonie, que le tissu du poumon avait ressemblé à un morceau de rate, mais plus résistant et plus solide.

Roceas. p. 28.

Walshe.

analyse de la nouvelle édition. dans

"Dublin Journal

3e Série. N° 10. octobre 1872.

Caseous Pneumonia.

1. Acute. caseous. infiltr.

2. Chronic. cas. infiltrat.

Tuberculo en general

Cell. glandes dans le
conduits glandulaires.

— Les Cellules géantes de la Lactante en général peuvent prendre origine dans les vaisseaux lymphatiques : par procédé des ~~sp~~ endothéliums

— Dans les recherches des conduits glandulaires. Jaulé. 1877. Thèse

— Kolz pense qu'il en est de même dans la veine. Path. p. 406

Cornil et Ranvier p. 209

Modifications nutritives des tubercules.

– Les tuber. ne peuvent pas se résorber comme le font les gommes syphil.

– ~~Tiss~~ Transformat. caséeuse est constante dans tout tubercule âgé – c'est un caractère essentiel.

une petite nodosité caséeuse au centre ou disséminé au tubercule.

— La dégén. cas. a été attribuée à l'oblitération des vaisseaux. et en effet. l'oblitération est constante

— mais dans les gommes où les vaiss. sont perméables il y a une dégéner. de même nature.

Cause de la Caséification

On explique les Lardons à la région du Rachis par la non vascularisation du Pleonasme — mais la chute n'est pas expliquée par aussi simple. Dans cependant le cartilages, il faut imaginer que la partie centrale d'un Tubercule n'est guère plus loin du voisinage que ne l'est l'ovule des parois vasculaires du follicule —

Il doit donc y avoir dans le follicule d'autres conditions — Les éléments du tubercule portent en eux le germe de la mort.

Fricolard. C.S.J.

Collège Vorläufer.

— mais le diagnostic est difficile, inclinant davantage

" comme la pneumonie caséeuse [phtisie] est beaucoup plus fréquente que la phtisie tuberculeuse — dans ce moment la majorité des cas c'est la pneumonie caséeuse qui forme la phtisie primitive

tandis que ~~ce qui est~~ à la [pneumonie phtisie] est locale [ce que nous savons aujourd'hui par 3 tuberculoses] on peut la dire phtisie simple, ou

~~caséeuse~~ caséeuse

— mais dit qu'il y a quelques signes de généralisation à cas la phtisie tuberculeuse en venu compliquer la phtisie simple —

En tout cas la phtisie tuberculeuse primitive du poumon est une rareté

Ce qui a lieu pour le poumon a lieu pour les autres organes. —

— 3°/. Consid. la planche de Carswell. — tubercules dans les ciments — planches de Carswell — c'est une curieuse impression — Carswell 3 étapes — ce qui a servi pour la tubercule. —

— fig. de Calberg. —

— 4°. Enfin Cliniquement — non seulement en ... générale et ... organes ... —

mais tout spécial à pneumonie doit servir le point de départ de ... caséeuse.

des poitrinaires
Tabes mesaraïca — Tabes des poitrinaires

Modifications — Ces caractères de la tuberculose — au même titre que la dyspepsie symptomatique du commencement

2° — Ces notions peuvent être étendues au poumon. — Il y a des inflammations caséeuses du poumon ; — ces lésions caséeuses qui en résultent s'étendent à tout un lobe ; ou bien disséminées sous forme de nodules disséminés

Pneum. caséeuse. Les contours des nodules sont moins arrêtés, ils sont diffus — non contours nets comme le pur tubercule —

— Il n'y a pas eu d'essaimage de petits nodules dont la réunion aurait pu produire la grosse masse.

— Il y a des phén. inflammatoires autour du nodule tuberculeux

" Il est certain du reste, que ces masses caséeuses se sont développées au lieu et place où s'étaient accumulés des exsudats inflammatoires, de plus dedans, épaissis dans les alvéoles qu'il encombre

— " La masse purulente épaissie ressemble tellement déjà à la matière tuberculeuse jaune ou blanche qu'on ne peut macroscopiquement les distinguer "

Et même au microscope ~~dans les cas récents~~ la distinction est impossible.

— Mais dans les cas récents on peut reconnaître les globules de pus non lésés, qui composent les noyaux —

— Et peu à peu on sut hésiter à déclarer la tubercule =
{ 1° Tuberculum — Pneumophthisis
{ 2° Phthisis tuberculosa
{ 2° Caséeuse. Pneumophthisis pneumonique
Pneumonia — caséeuse.

— Car les processus caséeux sont bien plus fréquents — bientôt on ne parle plus des tubercules.

— Le diagnostic en est difficile cependant.

Waldenbourg. – 8. 161.

Käsige nicht aus Miliartuberkeln hervorgangene produkte.

Pneumonie caséeuse. –

– occupons nous de ces produits caséeux – qui ne sont pas de provenance caséeuse.

~~Rien de mi~~ Il n'y a pas de fait mieux démontré en pathologie, aujourd'hui, que celui-ci

1°. ~~Il est clair que~~ (cette) ~~tubercule miliaire~~, lésions d'organes (autres) ~~caséeux~~ – ne se confondent pas dès le 1er examen anatomique avec granulations grises: ce n'est pas dès le 1er coup qu'on discerne leur caractère "tuberculeux" – ne devant à pas les 2. les. anatomiques des temps et nouvellement offrant comme d'aujourd'hui peuvent de
– 2. D'un autre côté

Il est ~~tuberculeux~~ (travail) dans l'aspect caséeux autrefois considéré par Laennec ~~comme~~ (faute d'oubli) propre au Tubercule (Mat. tuberculeuse, Mat. scrofuleuse c'est le tubercule) ne lui appartient pas en propre. – La matière caséeuse, tuberculiforme, peut ~~[illegible]~~ produire aux dépens d'autres éléments que ceux qui constituent le néoplasme tuberculeux

– Andral avait déjà signalé que le pus "tuberculeux" ordinaire par inspissation peut devenir "tuberculeux" c.a.d. caséeux. (inflammation est ainsi donnée pour le poumon et d'autres organes)

– Reinhardt que le pus peut devenir caséeux

– Virchow. fit remarquer que bien d'autres produits, sarcomes, &c, peuvent subir la Metam. caséeuse –
noyaux caséeux,

chez divers animaux lapins, dit M. Waldenburg, tous les abcès ~~[illegible]~~ caséeux.

Il y a des tumeurs caséeuses par excellence, dit. Virchow – mais les ganglions lymphatiques du scrofuleux, sont caséeux. non tuberculeux. (Schuppel)

Cadification.

Non cadificatum de

Intestino.

Intestina fibrosa

Intestina tenuia

Le Tuberculose Générale

par Langhans.

Ueber Riesenzellen und wandständige Kernreihen in Tuberkeln und die febröse Form der Tuberculose -

Virch. arch. t. 42, 1868

Tandis que dans le Lésion, la dure mère, la pie mère, la choroïde et la synoviale, c'est surtout le tubercule
cellulaire qui est observé, le tubercule appelé fibreux
par Virchow, se rencontre surtout dans les
parenchymes : Poumon, foie, rate, Rein,
Testicule, capsule surrénale, cerveau. Bien que dans
les organes aussi on trouve le tubercule cellulaire.
Dans le Poumon, le foie, on trouve le tubercule
cellulaire très fréquemment.

D'ap. Virchow. (Geschwülste. II. 639) le tubercule
fibreux se rencontre souvent dans des parties dures,
fibreuses, souvent dans le tissu conjonctif nouvellement
formé.

Cependant dans les membranes pleurales fibreuses, j'ai
trouvé seulement le tubercule cellulaire, absolument comme sur la
plèvre normale.

- Le tubercule fibreux macroscopiquement se distingue
déjà par sa dureté, sa grande transparence, cela
le distingue du tubercule cellulaire qui est plus opaque,
plus mou.

Il est composé de tissu conjonctif dense, avec des
cellules ± nombreuses, subissant souvent la métamorphose
graisseuse.

Dans les gros nodules on ne trouve plus de cellules
rondes, mais seulement un centre granulo graisseux,
ça et là strié, et une Bordure (Schaale) purement
conjonctive.

Cette forme est moins connue que les autres - elle
contient toujours des Riesenzellen.

Un gros tubercule de cette catégorie

Contient en général 3 zones. 1° centrale où la
le tissu est métamorphosé. 2° moyenne très riche en
cellules. 3° externe ou peu de cellules.

Les 2 dernières zones servent d'enveloppe à la partie
centrale dégénérée — elles sont minces comparées à la
zone centrale.

La masse centrale est trouble, nettement séparée des
zones périphér. plus transparentes. Elle n'a pas la
consistance sèche, friable des masses caséeuses habituelles.
Elle est plus tenace et plus dure.

Cela dépend de ce qu'il y a en elle une certaine quantité
de tissu conjonctif fibreux qui s'y est développé.

Ce tissu conjonctif contient des cellules plus ou certaines
points que sur d'autres. Les cellules en question sont
tuméfiées, granuleuses opaques, infiltrées de granul.
graisseuses.

L'opacité du tissu dépend d'une quantité de
granul. graisseuses, qui se détachent par l'acide de l'acide
acétique; granulations qui sont uniformément répandues
au centre, et à la périphér. au contraire sont disposées
en groupes et en séries.

Le tissu fondamental par l'acide acétique est
rendu très clair, homogène et se gonfle, et se plisse
quand le tubercule est libre, comme tout autre tissu
conjonctif.

Cette métamorphose graisseuse atteint principalement
les masses ~~[illegible]~~ de cellules, tandis que les
tractus qu'elles renferment peuvent encore contenir des
cellules bien formées.

La transition entre cette partie centrale
et la zone riche en cellules, est constituée par un
tissu qui a une substance fondamentale dense
, contient des cellules normales, nombreuses, souvent
étoilées et anastomosées.

quand ces cellules reçoivent des granulat.

graisseuse, se développe l'aveuglement particulier de la graisse dans les couches internes voisines.

La zone moyenne se sépare nettement de ces parties que le composé des mêmes éléments ~~qui en, comme un~~ que le tubercule cellulaire à l'acmé de son développement ~~[illegible]~~ c'est-à-dire qu'à la périphérie il y a une couche fibreuse à fibres parallèles, des petites cellules lymphatiques, ~~et~~ vers ~~séparant de~~ la partie centrale ~~composée~~ des grosses cellules et de Riesenzellen.

Les Riesenzellen ont absolument les mêmes caractères que dans le tubercule cellulaire. Elles sont disposées parallèlement à la périphérie de la partie centrale (en partie) à côté en partie conjonctive, à peu près à égale distance les unes des autres.

Je pense que ces corps ailleurs on peut croire qu'elles sont de provenance épithéliale (et non vasculaire) dans l'épithélium des bronches, des alvéoles pulmonaires, cellules hépatiques. Mais on ne trouve pas un pareil point de départ pour le cerveau.

On trouve de fait, surtout dans le tubercule du foie dans la zone externe, des groupes de cellules hépatiques bien conservées, des canaux biliaires avec épith. bien conservé. Qq. les cellules épith. sont détruites et le canal ne se reconnaît plus qu'à sa lumière — si la lumière disparaît on a des masses ou cordons avec noyaux parallèles semblables ~~aux~~ ceux des cellules géantes, et un petit intermédiaire, appelant çà et là des prolongements dans des directions diverses.

Mais je ne vois aucune connexion entre ces corps et les véritables cellules géantes.

- Celles ci ont pour siège la limite interne de la zône moyenne ou riche en cellules. 4.

La zône externe n'existe pas dans tous les tubercules tellement que la zône moyenne, riche en cellules se trouve directement en contact avec le tissu normal. Ainsi dans le poumon la couche de cellul. embryon. est en contact direct avec les parois alveolaires.

- Cette zône externe consiste en un tissu fondamental transparent, fibroïde, dont les fibres sont parallèles à la surface du tubercule.

Comme les cellules qui composent cette couche ne sont pas toujours facilement visibles, elle peut pas être abondante, cette zône apparait comme une capsule autour du tubercule.

Mais ce n'est pas une capsule enrichissant le développement, c'est la matière même du tissu tuberculeux. Les cellules sont rondes, allongées, en fuseau, etoilées, s'anastomosant entre elles. Tantot homogènes, brillantes, tantot grenues, granulées.

Cette zône se trouve même dans les plus petits tubercules qui n'ont pas encore de centre fibro-caséeux et dont le centre est formé par conséquent du tissu riche en cellules et de cellules géantes - tandis que dans les tubercules volumineux ces parties occupent la zône moyenne.

Le tissu conjonctif de cette zône n'est pas le tissu normal des parenchymes, poumon et foie, apparaissant par suite de l'atrophie des éléments spécifiques de la

région. Nota. c'est un tissu nouvellement formé – c'est le produit d'une inflammation, si l'on veut. tout comme les fausses membranes dans lesquelles on trouve les tubercules des plèvres.

Aussi quand un tubercule se développe dans le foie, dans le rein, loin des vaisseaux et des espaces, il a cependant une enveloppe fibreuse.

On trouve d'ailleurs dans les foies tuberculeux au voisinage de tubercules bien développés, qq. uns des lésions, de petites places rondes dans lesquelles le tissu glandulaire a complét. disparu – là dans une masse légèrement fibreuse ou homogène, on trouve de petites cellules rondes, lymphoïdes, également disséminées. qq. plus condensées au centre, qq. plus condensées à la périphérie.

Ces amas ne sont pas encore des tubercules – mais leur présence dans les foies tuberculeux semblent indiquer que nous avons là le 1er stade du tubercule.

– Les vaisseaux. dans les tubercules fibreux ont une disposit. spéciale – qq. on les trouve très bien injectés dans les prép. à l'acide chromique.

Ils traversent les 2 zones externes, dans une direction parallèle à la périphérie des tubercules, ils sont capillaires, ils ont pas plus de 0,05 mm diam. – sont ils néoformés ?

Sur la limite vers la zone centrale ils cessent de croître tout à coup.

– on a raison quand on attribue à

formation du tissu fibreux (et non à l'accumulation de cellules) la disparition des vaisseaux dans les parties centrales et la perte qui en résulte. 6.

Il n'y a pas ~~de~~ cette relation étroite constante habituelle entre les gros vaisseaux et le tubercule fibreux, qu'on constate dans le tubercule cellulaire — ainsi dans l'acinus du foie il n'y a pas de gros vaisseaux et le tubercule fibreux s'y développe.

— Le tubercule fibreux, diffère assez du cellulaire pour qu'on puisse se demander si lui aussi est un tubercule.

Virchow y voit un tubercule arrêté dans son développement.

Je n'y vois pour qu'une différence : les zones sont les mêmes. mais au lieu de dégénérer, il se forme déjà dans la zone riche en cellules un tissu conjonctif, pauvre en cellules, et c'est ce tissu qui subit d'abord la dégénération.

Le tubercule fibreux n'est donc pas un 1er stade - C'est plutôt une forme plus développée, plus avancée —

Les 2 formes de tubercule se ressemblent dans les 1ers stades, antérieures à la dégénération (tub. cellul.) ou à la formation de tissu conjonctif (tub. fibreux)

Nous les tubercules cellulaires qui se voient dans un organe, à côté des tubercules fibreux, pouvons être considérés comme représentant le 1er développement de ceux-ci.

L'analogie physiologique de ces

formes est surtout mis en lumière par le fait que sur un même cadavre.

1°, dans le poumon, la rate on trouve le tubercule fibreux

2° sur les séreuses le tubercule cellulaire.

Peut on dire maintenant que la forme cellulaire appartient surtout à la tuberculose miliaire aiguë, la forme fibreuse aux cas chroniques. Mes recherches ne sont pas suffisantes p. trancher la question; jusqu'à présent elles ne sont pas favorables à cette manière de voir.

Calcificazione, non
Calcifici di
Tubercoli.

La Cachexie des
glandes lymphatiques.

La Caséification des glandes scrofuleuses et
ou caséiforme. tuberculeuses ..

Die Verkäsung welche an hyperplastisch. Drüsen
so gewöhnlich auftritt, kann ich nicht als Zum
Bilde der Scrofulose gehörig betrachten, sie
gehört vielmehr schon der Tuberculose an
welche zur Drüsenhyperplasie hinzutritt.

Schuppel L 138

Schuppel p. 122.
Inflammations scrofuleuses.

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p. 201

— Il y a une tuberculose primitive des gangl. lymphatiq. — cas des ganglions lymphatiq. peuvent se rencontrer atteints de tuberculose chez des sujets qui ne présentaient ni ailleurs aucune trace d'inflamm. caséeuse ni de tuberculose ailleurs.

— Cela est considéré comme rare — mais cela a raison mais si l'on admet avec Schuppel que tous les gangl. lymph. caséeux, dits scrofuleux, sont en réalité d'origine tuberculeuse.

— On remarque les tubercules se développant dans la plupart de cas chez des sujets qui présentent des foyers caséeux. On les trouve en effet dans la plupart des cas de tuberculose des masses caséeuses dans ces lymphatiq. des bronches, à la suite du mésentère.

Suivant les uns tout foyer caséeux est propre à développer la tuberculose, Hoffmann d'autres Klebs, admettent un poison spécifique. Mais alors le poison se répandrait par tout foyer caséeux quelconque. Il faut remarquer la matière caséeuse des glandes lymphatiques soit de nature tuberculeuse.

(p. 131) Je n'ai pas vu Schuppel s'opposer à cette hypothèse parce que suivant moi la matière ~~tuberculeuse~~ caséeuse scrofuleuse, est en réalité une matière caséeuse tuberculeuse. Donc les cas de tuberculose des organes qui succèdent à l'adénite caséeuse ou scrofuleuse, sont réellement d'origine tuberculeuse.

Mais la question ~~se~~ trouve seulement déplacée car on se demande pourquoi les glandes sont primitivement atteintes.

— Il faut admettre ici pour cette tuberculose des ganglions, ce qu'on admet pour la scrofulose ganglionnaire. Les glandes scrofuleuses se présentent à la suite de foyers irritatifs. Survenus dans la peau ou muqueuses des parties correspondant à ces ganglions.

Souvent inflammat. [illegible] de dépôts articulaires — [illegible] les [illegible] d'ostéomyélite [illegible] — dans les cas 1, 10, 14 et 18 [illegible] d'arthrite purulente.

Quand il y a tuberculose des gl. bronchiques, c'est qu'il y a presque [illegible] irritatifs dans les poumons et les bronches; pour les glandes mésentériques dans les intestins. (Klebs cite des auteurs, dans ses Ratigen, Durlande der Gekrösdrüsen durch die Aufnahme des Tuberkelgiftes von Seiten des Darmes verursacht würden.)

— Mais ce n'est pas l'inflamm. simple comme infl. simple seulement. Il faut un autre élément — cet élément c'est la prédisposition individuelle — qui fait que chez un individu donné l'inflamm. produit la disposition tuberculeuse — cette disposition s'appelle diathèse tuberculeuse — Dans la disposition native, [illegible]
[illegible] tuberculeuse

Mais la question ~~est~~ tombe seulement déplacée, car on se demande pourquoi ces glandes sont primitivement atteintes.

— Il faut admettre ici pour cette localisation des ganglions, ce qu'on admet pour la scrofulose ganglionnaire. Les glandes lymphatiques se prennent à la suite de processus irritatifs survenus dans la peau ou muqueuse des parties correspondant à ces ganglions.

Souvent l'inflammation résulte de défaut ... – ... dans les cas 1, 10, 14 et 18 ... d'arthrite paralente.

Quand il y a tuberculose des gl. bronchiques, ce qui est presque constant dans les poumons et les bronches, par les glandes mésentériques dans l'intestin. (Klebs sie dadurch, dass die Kätigen Zustande der Gekrösdrüsen durch die Aufnahme des Tuberkelgiftes vom Leiten des Darmes verursacht würden.)

— Mais ce n'est pas l'inflammation simple comme inflammation simple seulement. Il faut un autre élément – cet élément c'est la prédisposition individuelle – qui fait qu'elle chez un individu donné l'inflammation produit la disposition de la ... – cette disposition s'appelle : diathèse tuberculeuse – ... la disposition native ... liquide.

La diathèse n'est pas dans le sang – elle est dans les tissus – et tous les tissus ne sont pas également vulnérables (qui sont vulnérables) – ... qu'il y a des tissus prédisposés ; d'ailleurs où la tubercule est très rare –

Pourquoi les lymphatiques – Les ganglions lymphatiques sont particulièrement vulnérables parce qu'il s'y forme incessamment des cellules. Il y a toujours là du tissu en voie de formation.

— Sur les rapports anatomiques, reprise aux ... d'inflammation.

ainsi 1° Tuberculose primaire des ganglions lymphatiques - compren... de l'état diathésique -
2° cause accidentelle - inflammation du tissu.
Et dans la glande c'est le follicule et non les tissus.

—— La tuberculose primaire glandulaire peut rester longtemps stationnaire et latente (quand il s'agit de glandes ...)
— Cette tuberculose peut guérir —
— Mais le caseum tuberculeux peut devenir le point de départ de nouvelles formations tuberculeuses par l'infection de voisinage.
c'est par des liquides que se fait cette infection, et non par la voie de cellules migratrices, puisque les cellules dans le caseum sont mortes.
p. 178 [— Il y a analogie de la tuberculose avec les tumeurs malignes. non seulement par l'infection de voisinage, mais aussi par la propagation dans les organes éloignés (la généralisation) c'est à dire les métastases.
Quand une tuberculose locale de quelques glandes lymphatiques, d'une tuberculose miliaire des poumons, ..., foie et des reins, que le point de départ soit dans une glande lymphatique ou dans le testicule, c'est le pendant de la généralisation d'une tumeur maligne.
c'est par l'intermédiaire du sang que cette propagation a lieu.
Il y a donc une dyscrasie tuberculeuse.
— Le travail récent de Ponfick (Berlin. Klin. Woch. 12. ...
1877. nouvelle la voie hypothétique.
depuis Buhl et Ponfick on suppose que la généralisation se fait par la voie d'un virus provenant d'un foyer caséeux quelconque et provient par la voie des liquides de l'organisme.
des faits cliniques, anatomiques, et expérimentaux plaident en faveur de cette théorie, mais la constatation de la substance infectieuse soit dans le sang, soit dans les foyers secondaires.
quant à la voie on peut penser aux lymphatiques ou ... 1re ligne. on sait déjà que les lymphatiques dans leur adventice ont souvent des tubercules, lorsque ces tubercules proviennent d'un foyer caséeux.
c'est pourquoi j'ai examiné spécialement le canal thoracique et trouvé ce canal infecté lorsque la tuberculose est restée locale dans le ... anatomie clinique, "dass es aber in der Mehrzahl der Fälle von generalisirender Miliartuberkulose in eigenthümlicher Weise, mitbetheiligt wird, indem sich eine meist vielfältige Eruption kleiner tuberkelähnlicher Knötchen an der intima entwickelt.
... vermögen uns als Beweis zu dienen ... beschaffene, ein ...

— Le travail ...
1879. Mémoire sur la voie lymphatique.
depuis Buhl dit Ponfick, on suppose que la généralisation se
fait par la voie d'une Veine provenant d'un foyer caséeux quelconque
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in eigenthümlicher Weise, mit betheiligt wird, indem sich
eine meist vielfältige Eruption kleiner tuberkelähnlicher
Knötchen in seiner intima entwickelt.
Die Herde vermögen aus als Beweis zu dienen
dafür, dass eine höchst abnorm beschaffene, ein
Specifisch, Virus enthaltende Lymphe den Gang
passirt haben müsste.

— Quant au siège de prédilection des tubercules secondaires et quand on ne peut expliquer, de nouveau la diathèse locale. Il faut en effet que les divers organes aient une susceptibilité spéciale.

— On comprend le précepte de Virchow d'enlever les glandes lymphatiques tuberculeuses, et tous les produits tuberculeux accessibles — testicules, os, poumons. (Voir Virchow. Geschw. II. 727

En dehors de cela il reste. 1° à combattre la prédisposition, 2° les causes [illegible] les excitants nuisibles.

L'éloignement des subst. excitantes est chose importante : Denn keine maligne Geschwulstsbildung ist an sich so auffällig mit entzündlichen Vorgängen, namentlich mit wirklichen Entzündungen verbunden, als der Tuberkel.

Jede namentlich chronische Reizung des prädisponirten kann die Tuberkeleruption nach sich ziehen. Welches

Jede bestehende Reizung, jeden Katarrh ist sorgfältig zu überwachen.

Tuberculose et Scrofule.

Schüppel

Schüppel f. 1893.

Die meisten Fälle von ~~Drüsen~~ Tuberkulose der verschiedensten Organe auf eine primäre Drüsentuberkulose zurückzuführen sind — aber (wie wir wissen) dass der Lymphdrüsentuberkulose in Wahrheit Tuberkulose ist.

Primäre Drüsentuberkulose.

Die primäre Drüsentuberkulose ist eine primäre Tuberculose, nicht als irritativer Process, dessen irritamentum vielmehr die regelmässige Beute von einem Atrium aus begleitet wird. (Virchow's Archiv. II. S. 673.)

Importance des nodules dans la forme ~~tuberculeuse~~ caséeuse.

Friedlander fait remarquer que les parties les plus jeunes de la néoformation du lupus est composé presque complètement ou principalement de ces nodules. Tandis que les parties les plus anciennes, là où il y a eu des violences ou cautérisations, se compose surtout de tissu de granulations avec des nodules parsemés.

Friedlander. p. 30.
Virchow's archiv. t. 60.

Mode de propagation des
Lepisma Entercularia. —

Infection tuberculeuse de Vortinage

. Kleb cite le cas d'une femme
atteinte d'tuberculose pulmonaire,
ayant eu ulcérat. tuberculeuses de
l'estomac ; elle a présenté une
tuberculose du péritoine, développée
au voisinage de ces ulcères et dont
la progression pouvait être suivie
sur le péritoine d'après la répartition
des nodules. —

Kleb, p. 257.

— cinq cas gastro-intestinale tuberculeux, parmi
lesquels à l'autopsie péritonite tuberculeuse.
— la péritonite tuberculeuse a toujours
son point de départ, dans les
ulcérations intestinales.

Laennec avait déjà remarqué que des <u>éruptions</u> Lemoins se font autour des lésions tuberculeuses en voie de ramollissement. et même ces éruptions peuvent se faire dans une cavité d'organe

— Il faut arriver avec Chauveau et Koster que c'est la seule matière caséeuse, et non pas toute matière tuberculeuse qui reproduit la tuberculose.

cas de Orth et de Lichtheim chacun 8. de tuberculose miliaire, avec foyer caséeux primitif.

Imp. Lymphatique
Interculeuse.

Insertion.

Cruveilhier

- Anat. pathol. Liv. II.
pl. I.

Chez les phthisiques arrivés
à l'ulcérat. intestinale, pourra
on trouv. tuberculeux dans
les vaisseaux chylifères
qui, des points malades
de l'intestin, se rendaient
à des ganglions mésentériques
tuberculeux - Soit que
la matière tuberculeuse fut
portée dans les vaisseaux
par voie d'absorption, soit
que bien plus probablement
elle y eut pénétré en nature
et en envahi les parois

Rindfleisch p. 324

– on trouve dans la tunique intestinale, au voisinage d'une ulcération tuberculeuse

« Le long des vaisseaux aux capillaires surtout les artères, une certaine quantité de tub. miliaires dans l'adventice.

Je ne suis convaincu que ce sont les vaiss. lymphatiq. qui de tuberculiser les gaines vasculaires ne paraissent pas évidemment le siège de l'éruption tuberculeuse que parce que ils contiennent les lymphatiques efférents de l'intestin.

Et cette direction des vaisseaux et des vaiss. lymphatiques est importante parce qu'elle indique la direction dans laquelle s'épanouit l'ulcération –

Vaisseaux lymphatiques, pour ainsi dire injectés de tissu tuberculeux — se dessinent sous la séreuse s. forme de vaisseaux noueux moniliformes et blanchâtres, se rendant de la zone péritonéale correspond. des ulcérat. de l'intestin aux gangl. mésentériques.

— Toujours ils sont remplis dans la Tab. intestinale. ils sont le noyau dispersés intestinaux — Des granulat. de granul. tuberculeuse —

— La tuberculisation des ganglions mésentériques est qq. primitive — Rilliet et Barthez.

— Cependant Louis affirme qu'il n'a jamais rencontré de ganglions mésentériques tuberculeux, sans qu'il existât d'ulcérat. intestinales.

Quand le tubercule a franchi les gangl. lymphatiques, les tubercules secondaires ne sont plus attachés aux vaisseaux lymphatiques mais bien aux vaisseaux artérielles petits et aux capillaires.

Reinflecti f. 94.

Tubercules disséminés.

Ronfleur p. 87, Manuel 2e édition

Sous le nom de tuberculose il y a la plus grande analogie entre la tuberculose pulmonaire et l'invasion des tumeurs malignes.

Il y a 3 étapes 1° infection locale, 2° infect. des glandes lymphatiques et 3° infection générale.

La résorption se fait par les vaisseaux lymphatiques — la généralisation par le sang.

L'affection des glandes lymphatiques est pendant ce temps une barrière contre la diffusion du poison morbide.

Souvent la barrière est par franchie.

Mais quand elle est franchie on trouve, alors que les tubercules miliaires se développent dans les petits vaisseaux et capillaires, ce qui démontre que l'infection est dans le sang —

REVUE DES JOURNAUX DE MEDECINE

JOURNAUX ALLEMANDS.

Du mode de développement et de propagation de la tuberculose miliaire aigue; par le docteur Ponfick (de Gœttingen).

On sait que Buhl a émis une théorie d'après laquelle la tuberculose miliaire aiguë généralisée reconnaîtrait pour cause un virus spécifique, lequel, élaboré par un foyer morbide situé en un point quelconque de l'organisme, se répandrait de là dans les voies circulatoires pour engendrer des foyers multiples disséminés. Mais jusqu'ici on n'a pu isoler cette substance infectieuse, pas plus dans le sang et dans les organes de la circulation, que dans les foyers d'éruption miliaire.

Dans ces derniers temps, Ponfick a fait des recherches nombreuses sur l'état du canal thoracique chez les tuberculeux. Il est arrivé à ce résultat, que le canal thoracique se montre intact chez tous les individus qui succombent à une tuberculose localisée. Au contraire, chez la plupart des individus qui sont emportés par une tuberculose miliaire aiguë généralisée, la tunique interne du canal thoracique est le siége d'une éruption de nodosités qui rappellent l'aspect des tubercules. Ces petits foyers semblent démontrer que le canal thoracique a été traversé par une lymphe douée de propriétés irritantes spécifiques. Ils constituent l'unique preuve que nous possédions jusqu'à ce jour, de l'adultération du sang par une matière inconnue, quant à sa nature, mais dont l'existence ne saurait plus, selon l'auteur, être mise en doute. (Berlin Klin. Wochensch., n° 46, 1877.)

Lupus. Friedlander Virch. t. 60. 1874

Structure du nodule du Lupus –

Virchow en fait une tumeur de granulation – sur le type des granulations des plaies – dans le tissu conjonctif – non dans le rete

Rindfleisch en fait un adénome des gl. sébacées, une affect. spécifique

3 cas récents de lupus exfoliatif – ulcéreux ou granuleux –

8 cas – diverses parties du corps face, membres

+2 – excision

Ces résultats les mêmes dans les 3 formes – 1° exfoliative, 2° ulcéreuse – 3° granuleuse (végétante)

1° Epiderme conservé

2° derme – il est changé en tissu de granulation – on y voit des cellules rondes et lymphoïdes

ceci dit par les auteurs – mais spécial, nouveau nodules de 0,15 à 0,11 de diamètre – très rapprochés – le tissu intermédiaire a qq. caractères de jeune alors comme on trouve des nodules difficile au centre des nodules –

– Ceci surtout dans la forme exfoliative les nodules innombrables avec les parties rouges se rapportent les jaunes –

Les nodules difficiles à séparer du tissu granulat. ambiant

mais cell. Epithelioïdes — elles ont 0,012
Cellules granuleuses 0,004

— cell. géantes à même protopl. que épithelioïdes

avec ou sans prolongements

ne manquent jamais — Vivantes

— pas de Vaisseaux dans le nodule

On trouve qq., dans les formes ulcéreuses qq nodules ayant un centre granuleux, et de noyaux libres — c'est une esquisse de caséification — mais jamais cela ci franchement

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Les glandes muqueuses, sébacées, [illegible] ne jouent aucun rôle.

La structure acineuse de Rindfleisch ne [illegible] que les nodules

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Les Lupus sont sains, cependant qq. cas d'[illegible] mais pas de phthisie —

Dans 2 cas glandes sous maxillaires consécutiv. très volumineuses — Là nodules avec Riesenzellen —

Donc analogie du Lupus avec glandes scrofuleuses

Tuberculose Locale

Tuberculose aiguë chez les jeunes enfants.
— 4 mois, Barlow.

— La tuberculose rare chez l'enfant au dessous d'un an.

Duckworth — enfant de 8 mois
Path. Trans. 1875. cas rare
Sorrell 7 mois. Path. Trans. 1874.

— Cependant Steiner, de Prague a rencontré
des tubercules chez des enf. à la mamelle.
de 2 mois.

Ueber Localisirte Tuberculose der Leber.

Von Orth.

Virchow's Archiv 1876 66. Bd.

In organes ou la Tuberculose primaire localisée, d'histoire, Cerveau, organes genitaux, sont peu disposés aux formes disséminées secondaires — tandis que la rate et le foie qui sont presque toujours atteints dans la Tuberculose generale sont peu atteints par le tubercule solitaire — ainsi dans le foie les nodules subpleuraux sont plus fréquents que dans les autres organes

— Dans le foie il y a 2 formes de Tuberculisation — 1° la Tuberculose miliaire du parenchyme, et 2° la

Tuberculose des voies biliaires —

a La première forme se distingue par la petitesse des nodules qui peut aller à tel point qu'il faut le microscope pour les reconnaître — Chez les enfants ceux-ci vont jusqu'à une cerise. ceux gros comme un haricot sont très rares.

b La forme la plus rare est la Tuberculose des gros conduits biliaires. alors les tubercules sont volumineux. Ils se distinguent par une couleur jaune verdâtre laquelle se montre au centre des foyers ramollis et dépend du mélange de la bile.

— De plus gros comparables aux tub solitaires du cerveau, se voient très rarement dans le foie — 2 cas de ce genre sont rapportés par Orth. Dans ces cas aucune participation des voies biliaires.

paroi externe longeait le muscle obturateur interne, la face interne de l'ischion, le pyramidal, le sacrum et allait jusqu'en arrière du rectum. Les parois étaient formées d'un tissu lardacé doublant la séreuse dans une certaine étendue et qui était constitué par la coque du ganglion. En haut, sur le psoas, on trouvait plusieurs ganglions lymphatiques volumineux dont les uns étaient infiltrés de pus et les autres durs.

Cette inflammation des ganglions péri-utérins est toujours le résultat d'une cause locale, différant en cela de la lymphangite utérine des auteurs, qui est plus souvent l'effet d'une contagion ou de l'épidémicité. L'adéno-lymphite péri-utérine s'observe souvent après l'accouchement; elle résulte d'une déchirure des orifices utérins pendant le travail; elle survient d'autant plus facilement que les femmes y sont plus prédisposées par un tempérament lymphatique ou strumeux. Elle s'observe aussi, en dehors des conditions de la puerpéralité, dans des circonstances pathologiques diverses, telles qu'une blennorrhagie (c'est le cas de l'une des premières observations recueillies dans le service de M. A. Guérin), un chancre du col, une métrite aiguë, une application de sangsues sur le col, etc. Dans l'un des cas rapportés dans le travail de M. Mary, il s'agit d'une adéno-lymphite observée dans le service de M. le docteur d'Heilly, à l'hôpital temporaire, qui aurait reconnu pour cause les mouvements nécessités par la mise en œuvre d'une machine à coudre.

L'affection dont il s'agit offre plusieurs modes de début. Lorsqu'elle se montre en dehors de la puerpéralité, elle débute ordinairement par une douleur vague que la malade ressent dans les reins, par de la fatigue, de la courbature. Lorsqu'on l'observe à la suite de couches, la nouvelle accouchée, au bout de deux ou trois semaines, commence à souffrir dans l'aine; cette douleur, d'abord unilatérale, irradie à la partie antérieure de la cuisse.

Dans un deuxième mode, le mal commence par une fièvre violente, avec frissons, sueurs abondantes, prostration rapide des forces. Ce n'est qu'au bout de vingt-quatre ou quarante-huit-heures que la malade éprouve une douleur vive à la racine du membre inférieur, remontant dans le ventre.

La douleur, qu'elle soit initiale ou qu'elle ne se fasse sentir qu'après le frisson, siège particulièrement dans le pli de l'aine. C'est là un signe presque pathognomonique ou qui peut, du moins, mettre sur la voie.

Les symptômes physiques sont la sensation que donne la palpation d'une tumeur ou plutôt d'un empâtement situé immédiatement au-dessus du ligament de Fallope, se continuant vers le canal crural, adhérent à la symphyse pubienne, immobile et douloureux à la pression; et celle que fournit le toucher vaginal de la même tumeur, qui peut être ainsi nettement circonscrite entre les deux mains, d'une douleur vive et de la température élevée du vagin. L'exploration de l'utérus fait souvent reconnaître la cause même de l'adéno-lymphite, soit la déchirure du col, soit des granulations.

Les symptômes rationnels et sympathiques du côté des autres organes, sont l'altération de la face, la constipation, la suppression des règles.

La marche de cette affection est subordonnée à la durée de la lésion voisine qui lui a donné naissance, d'où de grandes différences dans sa durée. Cependant la résolution est fréquente. M. Mary, en se fondant sur les cas qu'il a observés, l'estime aux 7 à 8/10es des faits. Il y a lieu d'y compter lorsque l'affection qui en a été le point de départ, une blennorrhagie par exemple, s'est elle-même éteinte ou a rétrogradé. La tumeur perd sa sensibilité, puis diminue graduellement de volume; arrivée à la période phlegmoneuse, l'adénite est encore susceptible de résolution. Dans le pronostic de l'affection il faut tenir compte de l'anémie consécutive.

Les éléments principaux du diagnostic sont, d'après ce qui a été dit plus haut, la douleur au-dessus du pli de l'aine, la tuméfaction adhérente au pubis et la direction de cette tumeur vers le trou ovale, enfin l'absence de mobilité et la résolution en général assez rapide sous l'influence du traitement dont il nous reste à parler.

L'adéno-lymphite péri-utérine comporte avant tout, comme moyen de traitement, l'usage des révulsifs. Les vésicatoires arrêtent et font rétrograder l'inflammation lymphatique. Dans les cas les moins heureux, dit M. Mary, ils circonscrivent au moins le foyer morbide et le concentrent autour des ganglions. Si, au moment où on se décide à y recourir, la résolution n'est plus possible, ils ont du moins l'avantage d'activer, d'accélérer la suppuration et d'en abréger ainsi la durée.

Lorsqu'un seul vésicatoire ne suffit pas, il ne faut pas hésiter à en réitérer l'application.

Les laxatifs sont également utiles, ainsi que les cataplasmes intra-vaginaux. Enfin, dans le but de diminuer la congestion pelvienne, on devra prescrire la position horizontale et le séjour au lit pendant toute la période d'acuité de la maladie.

Tels sont les moyens qui ont été le plus utilement mis en usage dans les faits observés par M. Mary.

Traitement des vomissements incoercibles des femmes enceintes.

Il y a quelques années, M. le docteur Lubelski, médecin de l'hospice de l'Enfant-Jésus, à Varsovie, fit savoir qu'il avait appliqué avec un succès aussi rapide qu'inattendu, contre la chorée, la méthode alors nouvelle de l'éther pulvérisé. Ces premiers essais furent répétés quelque temps après avec les mêmes avantages, paraît-il, dans les hôpitaux de Strasbourg, par M. Zimberlin, et dans les hôpitaux de Lyon, par M. Perroud et M. Meynet. M. le docteur Lubelski a eu l'idée, depuis, de traiter par le même moyen les vomissements incoercibles de la grossesse. Dès la première apparition des vomissements ou même des nausées, il prescrit l'usage d'une douche d'éther pulvérisé, à la région épigastrique et à la partie correspondante de la colonne vertébrale; il recommande de prolonger cette douche de 3 à 5 minutes et même plus longtemps si la femme s'en trouve bien, et de la renouveler toutes les trois heures; dans les cas rebelles, d'alterner les douches d'éther avec celles de chloroforme.

M. Dujardin-Beaumetz a communiqué à la Société de thérapeutique le fait suivant, dans lequel, en imitant la conduite de M. Lubelski, il a obtenu un résultat également satisfaisant.

Une jeune femme, au deuxième mois de sa grossesse, était atteinte de vomissements incoercibles, qui ne lui permettaient de conserver aucun aliment solide ou liquide. Après avoir essayé sans aucun résultat tous les moyens propices en pareil cas, valérianate de caféine, chloral, emplâtre d'opium, teinture d'iode, etc., il a eu recours à la méthode des pulvérisations d'éther.

Voici comment il a procédé: immédiatement avant le repas, on faisait, pendant 5 minutes, avec l'appareil Richardson, des pulvérisations d'éther vers le milieu de la région dorsale et au milieu de l'estomac. Les vomissements cessèrent presque aussitôt. Le traitement fut suspendu au bout de huit jours, les vomissements avaient disparu pour ne plus revenir

Dr Brochin.

HOPITAL MILITAIRE DE GIVET. — M. Challan.

Péritonite tuberculeuse survenue d'emblée chez un sujet robuste et d'une superbe santé habituelle.

Louis n'admettait pas la péritonite tuberculeuse en dehors de la tuberculose pulmonaire. Depuis Louis, plusieurs observateurs, Grisolle et Lebert entre autres, ont, au contraire, déclaré que l'évolution de la tuberculose péritonéale précède presque toujours la tuberculose pulmonaire. Le fait suivant me paraît une nouvelle démonstration de cette dernière assertion.

N..., soldat porte-sac d'ambulance, au 128e régiment d'infanterie, entre à l'hôpital de Givet le 23 novembre 1877. C'est un robuste garçon, dont les antécédents sanitaires ne laissent rien à désirer. Son père, sa mère, son frère et ses sœurs, tous cultivateurs, jouissent d'une excellente santé. Le 18 novembre, cet homme, assistant à un exercice militaire en campagne, dut faire une course assez longue pour accompagner le médecin-major du régiment, appelé en toute hâte auprès d'un officier frappé de congestion cérébrale. Arrivé sur les lieux, il fut chargé de maintenir des chevaux et demeura assez longtemps au repos. Il était couvert de sueur et se trouvait alors sur une élévation balayée par un vent froid et humide. Il but deux chopes de bière et ne tarda pas à éprouver un très-vif refroidissement. Le soir même il se plaignit de coliques; dans la nuit il eut un violent frisson. Très-dur de sa nature, il voulut continuer son service.

Le lendemain, en accompagnant le médecin-major à sa suite, il se plaignit de battement abdominal et prit un purgatif salin.

Le surlendemain et les jours suivants, les douleurs s'accentuèrent: elles arrachaient des plaintes incessantes et s'exaspéraient sous l'influence du moindre attouchement. Le ventre parut alors tendu et météorisé, il y eut quelques vomissements alimentaires mêlés d'une petite quantité de bile, la fièvre cependant ne parut pas très-intense. Le diagnostic était fait; le 23 N... fut envoyé à l'hôpital.

A son arrivée, M. le professeur Chauvel, alors médecin en chef, constate :

Ventre tendu, météorisé, très-douloureux à la pression; le moindre attouchement arrache des cris au patient. Légère matité dans la fosse iliaque droite. Face anxieuse. Pouls fréquent, petit. Température normale, 26°8. Ni toux ni expectoration. Intelligence parfaitement lucide. Insomnie sans agitation.

Il prescrit : vésicatoire abdominal; cinq pilules extrait d'opium, à 0,01, et de belladone à 0,01.

Pas de changement appréciable les jours suivants.

1er décembre. — Face grippée. Décubitus dorsal. Vomissements bilieux abondants. Matité absolue dans les hypochondres. Ascite évidente. Le moindre mouvement exaspère la douleur. Respiration normale dans toute l'étendue de la poitrine. Insomnie persistante. Pouls 80, petit. Température moyenne, 37°,2.

P. : Tilleul nitré. Potion gommeuse, morphine 0,05. Teinture de digitale 1 gramme. Teinture de scille 2 grammes. Eau de Seltz.

15 décembre. — Face pâle, amaigrie, langue normale. Ventre volumineux, irrégulier, plus développé à droite, dur, rénitent et moins douloureux à la palpation. Les intestins paraissent refoulés vers l'ombilic. Les hypochondres sont mats, l'ascite a cependant sensiblement diminué. Pouls petit. Température du soir 36°,4. Appétit assez bien conservé malgré des vomissements bilieux parfois abondants. Nuits relativement bonnes.

Frictions mercurielles belladonées. Dix pilules extrait d'opium 0,01.

Le 5 janvier, à mon arrivée à l'hôpital, je constate :

Amaigrissement très-prononcé. Face pâle, yeux excavés, interrogateurs. Langue normale. Ni céphalalgie ni manifestation cérébrale d'aucune sorte. Thorax émacié permettant de compter les côtes à vue. Respiration régulière, normale. A peine un peu d'affaiblissement du murmure vésiculaire et légère diminution de la sonorité sans localisation précise. Ni toux ni expectoration. Bruits du cœur faibles, souffle anémique plus prononcé dans les carotides. Ventre ballonné, le creux épigastrique est effacé. La percussion, qui est peu douloureuse, donne une sonorité obscure, sauf dans les deux fosses iliaques où la matité est absolue; cependant l'épanchement ascitique paraît presque complétement disparu; le décubitus latéral ne modifie pas la nature du son, on ne perçoit nulle part ni frottement ni fluctuation. La palpation permet de constater la présence de tumeurs dures, irrégulières, assez volumineuses et absolument indépendantes de la paroi abdominale qui glisse facilement sur elles. Le malade accuse une douleur sourde mais peu intense, bien qu'elle soit, dit-il, assez vivement réveillée par la palpation. Il se plaint surtout de la fatigue que lui occasionnent des vomissements d'une grande abondance, survenant assez régulièrement deux fois par jour, deux heures environ après les repas. Les vomissements, d'une odeur fade, sont très-acides; ils contiennent des débris alimentaires presque digérés et mêlés d'une grande quantité de bile. Les selles sont régulières, légèrement cendrées; ni diarrhée, ni constipation. Il n'y a pas trace d'œdème des extrémités. Les urines sont peu abondantes, à peine un litre par jour, et pâles. Elles sont acides, légèrement albumineuses et contiennent également un peu de sucre (examen de M. Troupeau, pharmacien aide-major). L'examen microscopique y révèle la présence de cellules épithéliales et de quelques globules purulents et graisseux.

Viande crue hachée 150 grammes, vin de Bagnols 200 grammes, café édulc., alcool. 5 pil. ext. d'opium 0,01. Fric. mercur. bellad.

Le 11, décubitus dorsal fixe. L'amaigrissement fait des progrès rapides. Débilitation générale, le malade a besoin d'un aide pour manger, son repas dure près d'une heure. Les douleurs abdominales ont disparu, la palpation les réveille, mais peu intenses. Les tumeurs paraissent moins nombreuses, mais le ventre tout entier donne une sensation de résistance très-accentuée. Les nuits sont généralement bonnes. N... ne se croit pas en danger, il sollicite un congé de convalescence qui lui est immédiatement promis. Il se plaint seulement de l'abondance et du retour régulier de ses vomissements, deux heures environ après les repas. Volontiers il recommence à manger après qu'il a vomi. L'abdomen est mat dans toute son étendue, sauf au niveau du colon transverse; son ballonnement contraste avec la maigreur squelettique du corps. Pas de fluctuation appréciable. Les urines sont peu abondantes, acides, chargées d'urates et de phosphates; elles contiennent toujours un peu de sucre et d'albumine. La température reste normale, 37 à 37, 4; le pouls est fréquent et misérable. L'intelligence est parfaitement nette, aucun trouble cérébral, aucune manifestation nerveuse pathologique. Respiration normale. Selles régulières, molles et cendrées. La viande crue n'est pas acceptée avec plaisir, elle est remplacée par l'huile de foie de morue que le malade préfère.

Le 18, pas de changement sauf l'amaigrissement qui fait des progrès incessants; toutes les éminences osseuses font saillie sous la peau. Mêmes vomissements réguliers; le malade néanmoins conserve quelque appétit. Malgré sa faiblesse, il déclare se trouver mieux, il espère une prompte convalescence. Le ventre est dans le même état, la respiration reste normale. Les douleurs ont disparu, les nuits sont bonnes. Température 36, 4 à 36, 8; pouls à peine perceptible. Légère constipation.

Mêmes prescriptions. Lavements de vin et de bouillon.

Le 23, faiblesse extrême. Intelligence cependant parfaitement nette; le malade, ne souffrant plus, se croit en convalescence et ne se rend pas compte de sa faiblesse. Depuis trois jours, des injections sous-cutanées de chloroforme, 2 à 4 grammes, pratiquées une heure avant les repas, ont retardé les vomissements et même les ont supprimés pendant un jour entier. Ces injections occasionnaient seulement une sensation de chaleur très-supportable et de courte durée, autour de l'endroit piqué, et une sorte de coma vigil assez agréable, paraît-il. — Même état de l'abdomen. On pourrait à peine admettre un peu d'obscurité de la respiration au sommet du poumon droit, en avant. Même état du pouls et de la température, 80 à 90,

36° 8 à 37° 2. Les urines sont toujours acides, elles contiennent encore du sucre, mais plus d'albumine, on y rencontre également quelques cellules graisseuses très-faciles à distinguer au microscope. Les selles sont régulières, cendrées et peu abondantes.

Le 24, marasme absolu, malgré l'intégrité des fonctions cérébrales et respiratoires. Véritable épuisement des forces vitales. A cinq heures du soir la température était encore de 36° 8, mais le pouls était difficilement perceptible, malgré la fréquence et l'énergie des pulsations du cœur. Ce contraste marqué entre la force des pulsations cardiaques et la faiblesse du pouls artériel, même dans les gros vaisseaux, a été signalé par mon père comme étant un signe probant de mort prochaine. Je l'ai nettement constaté chez N... huit heures avant la mort, survenue à minuit sans agonie, sans convulsions, sans souffrance accusée.

Autopsie trente-deux heures après la mort.

Maigreur squelettique. Quelques suffusions sanguines à la partie postéro-interne des membres, et dans la région dorsale.

A l'ouverture, la paroi abdominale se détache sans difficulté, à peine quelques légères adhérences entre elle et le péritoine. Il n'est pas possible, à première vue, de distinguer les différents viscères qui sont étroitement fixés au péritoine par des fausses membranes épaisses et très-résistantes. Les deux feuillets du péritoine sont remplis de granulations jaunâtres, ramollies, petites et déposées sur une couche plus ou moins épaisse, ce qui explique la sensation de bosselures constatées sur le vivant. La séreuse, partout adhérente à elle-même, se présente sous l'aspect d'une couenne lardacée, jaunâtre, lisse, résistante à la coupe et d'une *épaisseur de 1 à 3 centimètres*. La masse intestinale, mise à découvert, se présente sous forme d'une masse pelotonnée dont il est impossible d'isoler les anses, toutes reliées entre elles par des membranes recouvertes d'innombrables granulations miliaires, absolument identiques, quant à leur aspect, aux granulations de la phthisie caséeuse. A peine est-il possible d'isoler le colon transverse, tout le reste de l'intestin est rétracté, et d'un diamètre bien visiblement inférieur au diamètre habituel. Partout où il est possible de l'examiner, la muqueuse paraît saine; il n'y a pas traces d'ulcérations ni de granulations, non plus que de fistule apparente. Le colon transverse et le colon descendant paraissent sains, ils contiennent une certaine quantité de matières stercorales argileuses et grisâtres. La bile, sans doute, n'arrivait pas jusqu'à elles pour les colorer. Le foie paraît légèrement atrophié, il est de couleur brunâtre et d'une consistance normale. Il ne présente, du reste, pas trace d'altération organique. On rencontre seulement dans le lobe droit deux petits noyaux de matière caséeuse ramollie. Tout autour, le parenchyme est sain, il n'a pas du tout l'aspect du foie gras. La rate a conservé son volume, sa consistance et son aspect habituels. Le péritoine qui la recouvre est, de même que les ligaments suspenseurs du foie, infiltré de granulations caséeuses.

La substance corticale des reins présente sa coloration fauve normale, la substance tubuleuse est pâle, couleur de paille claire, on n'y rencontre ni granulations ni plaques laiteuses.

L'estomac est distendu, il contient encore des aliments liquides mêlés de bile. La muqueuse est pâle, peut-être un peu ramollie, mais sans autre altération sensible.

On constate quelques légères adhérences entre la plèvre costale et la plèvre pulmonaire, sans épanchement. La masse pulmonaire tout entière, plongée dans l'eau, surnage parfaitement. Du reste, les poumons sont crépitants dans toute leur épaisseur. Le parenchyme a son aspect rose grisâtre habituel. A la coupe, et malgré des incisions multipliées, on constate seulement dans le lobe supérieur gauche un noyau caséeux jaunâtre, de la grosseur d'un haricot et parfaitement isolé; tout autour le parenchyme pulmonaire est intact.

Le cœur est petit, les colonnes charnues des ventricules paraissent pâles, décolorés; mais les valvules et les orifices sont parfaitement intacts et libres.

Le cerveau et la moelle épinière n'ont pas été examinés.

Il ne m'appartient pas de me prononcer sur la nature tuberculeuse, granuleuse ou simplement inflammatoire des produits morbides que j'ai signalés. Mes connaissances histologiques ne me le permettent pas; et, par une fâcheuse inattention, les pièces que j'ai adressées à Paris pour être soumises à l'examen de l'un des plus habiles micrographes du Val-de-Grâce ont été plongées dans de l'alcool mélangé d'ammoniaque. Ce mélange a suffi, paraît-il, pour les rendre absolument méconnaissables.

Quelle que soit la nature du produit, est-il possible de mettre en doute, ici, l'influence d'un brusque refroidissement, le corps étant en sueur, comme cause déterminante de l'évolution de la maladie? Le sujet jouissait d'une parfaite santé; il n'avait fait aucune chute, n'avait été exposé à aucune violence susceptible de déterminer une rupture viscérale. Il était d'une sobriété exemplaire. Il n'avait jamais eu ni fièvre typhoïde, ni rhumatismes, ni dysentérie, ni coliques hépatiques, ni ictère, rien en un mot qui pût expliquer une perforation intestinale. Les premiers accidents s'étaient montrés quelques heures à peine après le refroidissement, et les douleurs, que le malade prenait pour des coliques, n'étaient pas de nature à faire supposer une perforation intestinale. Il serait cependant imprudent de nier la possibilité d'une perforation occasionnée par quelque corps étranger. Mais la démonstration n'en a pas été possible, et les antécédents du malade n'en fournissent aucune indication.

Évidemment il n'est pas possible d'admettre une tuberculose pulmonaire. Le noyau isolé que j'ai constaté dans le lobe gauche ne saurait infirmer cette manière de voir; il s'est développé consécutivement à l'évolution de la péritonite, si même il est permis d'admettre cette influence sur son évolution.

Le malade ne présentait, sur le corps, ni abcès glandulaire, ni trace d'affection chronique susceptible d'expliquer une inflammation par propagation. Impossible donc de considérer ce fait comme justiciable de la théorie de Niemeyer admettant la résorption de quelque foyer scrofuleux par les lymphatiques, comme cause de développement de granulations miliaires tuberculeuses.

La cause déterminante paraît donc vraiment l'action du froid, sur un individu en pleine transpiration.

Les symptômes observés ont été classiques. Les vomissements alimentaires et bilieux, d'une grande abondance, revenant régulièrement chaque jour, deux heures environ après chaque repas, étaient de véritables régurgitations dues au pelotonnement de l'intestin, à l'amoindrissement de son diamètre et à la difficulté de la circulation alimentaire. Ainsi s'explique aussi la rapidité de l'amaigrissement, le malade se nourrissait de ses propres tissus; la viande crue, l'huile de foie de morue, les lavements de vin et de bouillon ont pu le soutenir, mais étaient absorbés en trop petite quantité pour entretenir les forces vitales. L'intestin, cependant, paraissait digérer tout ce qui passait; ainsi il n'y a jamais eu de diarrhée. Les urines étaient peu abondantes, mais toujours acides et contenant parfois un excès de matières extractives azotées dénotant également une modification profonde dans l'acte de la nutrition. Malgré les adhérences du foie, les veines abdominales n'ont jamais été variqueuses, la circulation de la veine porte s'exécutait donc facilement. Il m'a paru bon de recueillir l'observation de cette maladie dont l'étiologie méritait, à plusieurs titres, de fixer mon attention et dont l'évolution a été une nouvelle consécration des assertions de Grisolle et de Lebert.

CLINIQUE DES DÉPARTEMENTS

Tumeur du creux poplité.

Par M. E. Sorrier, médecin principal en retraite.

C..., Auguste, 22 ans, tempérament mélangé, forte constitution, laboureur à Valleroy-aux-Saules près Mirecourt, vient nous trouver le 7 mai 1877 pour une grosseur qu'il a dans le jarret gauche depuis 18 mois.

Il raconte que, sans cause connue, il a vu cette bosse, d'abord du volume d'une noisette, augmenter lentement et acquérir la proportion d'un petit œuf d'autruche, sans gêner la marche, la flexion, l'extension du membre, sans même déterminer de claudication, ni d'autres troubles que de légers fourmillements au pied et quelques varices au mollet.

Cette tumeur, qui mesure 44 centimètres de circonférence et remplit tout le creux poplité, est dure, élastique, complétement irréductible, un peu mobile, faisant une légère saillie, sans adhérence ni changement de couleur à la peau, indolente.

Quelle est la nature de ce néoplasme?

Le diagnostic hésite entre un anévrysme, un névrôme, un kyste synovial, un ganglion lymphatique, un abcès, un lipome, un fibrome.

Un anévrysme? Absence de frémissement vibratoire, de bruit de souffle ou de *thrill*, de mouvement d'expansion correspondant à la systole ventriculaire et isochrone aux pulsations artérielles; en un mot, la tumeur est silencieuse, irréductible à la pression directe et nullement diminuée par la compression de la crurale; ajoutons qu'elle n'entrave en rien le cours de l'ondée sanguine, ainsi que le prouvent les battements artériels de la tibiale postérieure derrière la malléole interne. Ce n'est donc pas un anévrysme.

Un névrome? Un névrome n'atteint jamais de pareilles dimensions; il est ordinairement d'une dureté de silex, occasionne des fourmillements douloureux dans les fibres nerveuses périphériques et se traduit par des modalités fonctionnelles curieuses, dans la zone cutanée qui n'est plus animée par ses ramifications nerveuses. Ce n'est donc pas un névrome.

Un kyste synovial? On ne les remarque guère dans la région poplitée, mais bien dans l'interstice des muscles et dans les digitations de la patte d'oie de la partie interne. Ce n'est donc pas un kyste synovial.

Un ganglion? Cela n'est guère probable, par cette raison qu'au creux poplité les ganglions sont à l'état rudimentaire pour ne pas dire négatif, et que, chez notre malade de tempérament sanguin, il n'existe nulle part aucun indice d'engorgement, strumeux ou lymphatique. Ce n'est donc pas un ganglion.

Un abcès? Qui dure depuis 18 mois, sans poussées inflammatoires, sans rougeurs à la peau, sans fluctuation. Nous ne nous arrêtons à cette hypothèse que pour la rejeter. Ce n'est donc pas un abcès.

Que reste-t-il encore de cette élimination?

Un lipome et une tumeur fibreuse? Peut-être bien l'un et l'autre, un lipome fibreux.

Nous aurions pu, de suite, par une ponction exploratrice, faire jaillir le diagnostic des profondeurs obscures de cette tumeur pleine de doutes; mais nous ne l'avons pas fait parce que nous sommes persuadé que nous avons affaire à un fibrome et que de cette poche charnue il ne sortira rien. En effet, la tumeur est dure, résistante, élastique, irréductible, indolente et muette, et présente tous les caractères d'un fibrome.

Je passe sous silence tous les remèdes secrets, signes de croix, eaux miraculeuses puisées à des sources fécondes... en déceptions, qui en vidant sa bourse n'ont pas encore épuisé sa confiance, pour arriver de suite à l'opération qui est pratiquée avec le concours de M. le docteur Soyeux. Le malade, couché sur le ventre, n'a pu être chloroformé: incision cruciale de 12 centimètres. Après avoir enlevé l'aponévrose qui est adhérente, on aperçoit la tumeur, enfouie comme enchâssée dans le losange formé par les muscles biceps, muscles de la patte d'oie et jumeaux. Après une dissection minutieuse, longue, nous pouvons facilement la séparer des gros troncs vasculaires et nerveux qu'on voit dans ces profondeurs, sans qu'aucune hémorrhagie abondante vienne effacer les points de repère ni faire hésiter notre main.

Ce néoplasme ovoïde pèse 550 grammes; son enveloppe fibreuse, résistante, semble formée par le refoulement du tissu connectif ambiant. Sa trame légèrement friable, criant sous le scalpel, est d'un jaune grisâtre et d'une homogénéité parfaite, sans trace de vaisseau et ressemble, à s'y tromper, à de la tétine de vache. C'est donc un fibrome à un degré de condensation peu avancé, peut-être en voie de régression adipeuse.

Les suites de cette opération ont été assez simples, sauf une fièvre muqueuse, expression morbide d'une constitution médicale régnante qui a prolongé le travail de réparation, avec suppuration abondante; enfin, par des injections détersives médicamenteuses nous avons pu modifier les surfaces cruentées, et hâter la germination des bourgeons charnus qui, en poussant des profondeurs, ont comblé ce vaste hiatus et ont servi de trameaux premiers linéaments toujours si délicats d'une cicatrice naissante.

Un mois après, la guérison était parfaite et permettait à notre opéré de se livrer sans gêne et sans claudication aux rudes travaux des champs.

REVUE DE LA PRESSE.

Occlusion intestinale complète ayant duré six semaines et causée par un rétrécissement infranchissable, annulaire (carcinome colloïde), occupant la fin de l'intestin grêle. — M. Leroux, interne du service de M. Dumontpallier, a rapporté à la *Société anatomique* cette observation, que nous résumons brièvement :

Un homme, âgé de trente-sept ans, imprimeur, ayant eu la fièvre muqueuse à l'âge de quinze ans, a ressenti pour la première fois, en octobre 1876, des coliques abdominales assez fortes avec des alternatives de diarrhée et de constipation : il est repris, en avril 1877, des mêmes symptômes : la constipation augmente et persiste malgré l'emploi des purgatifs. Au 25 avril, les selles sont totalement supprimées : le ventre se ballonne, éructations, nausées. Entré à l'hôpital le 10 mai. Les contractions intestinales déterminent des gargouillements et peuvent se percevoir facilement à travers la paroi abdominale. Distension énorme de l'abdomen, pas de circulation collatérale. Les purgatifs ne produisent aucun effet. Jamais le malade n'a vu de sang ni de matières glaireuses dans ses selles. Les accidents deviennent de plus en plus graves, les vomissements surviennent; le malade meurt le 15 juin : *la rétention absolue des matières ayant duré cinquante jours.*

L'autopsie fait reconnaître la cause de cette occlusion intestinale :

Les anses intestinales ont triplé de volume et la distension est telle qu'en les écartant, il se fait spontanément une rupture. Les parois de l'intestin grêle sont ramollies, peu résistantes, la muqueuse est à peu près saine. En déroulant l'intestin, on arrive à l'obstacle qui est situé tout à fait à son extrémité terminale en avant de la valvule iléo-cæcale, où l'intestin grêle forme avec le cœcum et le mésentère une masse dure et agglomérée, l'intestin grêle est extrêmement rétréci dans les dix derniers centimètres, dur et presque infranchissable. Les parois sont fortement épaissies, dures à la coupe, blanchâtres, lardacées, ne fournissant pas de suc laiteux à la pression : des ganglions volumineux, ayant subi une dégénérescence caséeuse ou bien gélatineuse, sont compris dans les feuillets adhérents du mésentère.

L'examen histologique, fait sur une coupe au niveau du rétrécissement, montre la muqueuse complétement infiltrée d'éléments embryonnaires, ses glandes plus ou moins détruites, mais sans grande altération ni ulcération de sa surface libre. Le tissu sous-

Tuberculose locale

Friedlander. La tuberculose en général est une tumeur maligne d'après Virchow, c'est à dire qui peut se développer et se multiplier par infection, mais comme les autres tumeurs malignes elle peut rester d'une façon ± permanente partielle ou locale.

Il cite à ce propos, les örtliches fungosa, les dérivées tuberculeuses qui dans certains cas sont suivis de tuberc. miliaire et dans d'autres non.

– Nach Friedlanders Ansicht Jedwede Bösartige Entzündung die Schwindsucht hervorruft, wie z. B. in den Lungen in den meisten Fällen auch einen Tuberculösen Process vorstelle.

Ann. Brodowski.

Tuberculose locale.

Le tubercule du lupus est généralisé
(?) — Mais il y a d'autres formes tuberculeuses
qui restent solitaires

Il y a en pareil cas malignité locale
mais non malignité infectieuse

La malignité locale c'est la
tendance à la nécrobiose.

— Dans les tubercules des ganglions,
du testicule, des centres nerveux, la
généralisation est plutôt rare.

Ainsi d'ap. la remarque de Recklinghausen
c'est dans le cerveau qu'on trouve
surtout la tuberculose locale.
malgré leur longue durée ils restent
solitaires ou confinés au cerveau.

Koster (Centralblatt. 1873. 20 Decemb.
est arrivé à des résultats analogues.
Seulement l'opinion de Koster diffère
de la nôtre.

Koster dit qu'il a trouvé les tubercules
dans des lupus déjà primitivement

enflammés et les considère
comme secondaires.

Il ne dit pas que cela ne puisse pas
arriver, mais dans le [illegible] [illegible] [illegible]
les tubercules dans les parties saines.

Il semble pourtant à la vérité le
tubercule est souvent consécutif à
l'inflammation.

Friedlander p. 32.

A. 60.

Volkmann. Tuberculose
Locale de
~~l'anus~~ la muqueuse
rectale, point de
depart des fistu. à l'anus
des tuberculeux. –
– Resumé par Lannois
Spillmann.
p. 207.

Carricature primitive
gangliomaire

Par MMM. Colrat et Lepine.

Revue mensuelle
Pr 366.

10. mai. 1898 -

Cas un cas très rare.
Femme dans l'aisselle
gauche.

Tuberculose locale

La tendance à la généralisation n'est pas un critérium absolu de la nature tuberculeuse d'une affect. locale. Le tubercule peut être solitaire

ainsi la généralisation peut faire défaut dans la tuberculose des jointures, du testicule, du syst. nerveux central.

— Le gros tubercule du cerveau qui est énorme reste souvent jusqu'à la fin, solitaire

exemple le lupus.

~~Volk~~ Friedländer. Volkmann's Samml.

p. 31.

On n'a pas assez considéré cette tuberculose locale qui a des dans généralisation

Vorträge 6.5.18.

Exemples de tuberculose *locale*.

1° Lupus.–

2° 1 cas d'Ulcérat. tuberculeuse du col, sans aucun ~~signe~~ tubercule ailleurs chez une femme (autopsie

2° Gomme tuberculeuse du lobule de l'oreille chez une autre femme.

3° Forme d'ulcère rodens de vieille date chez une femme – sur la joue

4° Dans le derme d'une tumeur cancéreuse.

3° Tuberculose locale des jointures

4° Tuberculose du testicule de date ancienne sans rien dans les poumons

5° La péricardite tuberculeuse est [illegible] solitaire.– et elle est en ce cas souvent miliaire et très petite.

6° On peut aussi trouver de telles localisations de petits nodules, dans la plèvre, le péritoine, l'intestin, le poumon.

La tuberculose locale a son analogue dans une forme de syphilis.–

_Il y a dans ce cas malignité locale.

Mode de propagation de
la tuberculose dans les
voies urinaires et générales.

Recueil, t. 42.

— Ce n'est pas par contiguïté,
comme cause déterminante
dans les cas primitifs, le
testicule et la prostate
étant malades.

Locale Tuberculose

Friedlaender

Volkmann's Sammlung.
N° 64.

La tuberculose ([illegible]) propre des animaux, n'a rien de commun avec la tuberculose de l'homme.

[illegible] des Riesenzellen pr. le diagnostic anat. du tubercule.

Tubercules du Lupus, des fongosités, du testicule, etc.

Enlèvement prématuré
des membranes. Lésion

Péritoine et Plèvre.

Péricarde.

Tuberculose locale

Cruveilhier. dit. p. 136. — Il y a des tubercules locaux qui tiennent à une cause locale. Ex. tub. testiculaires. — Il y a aussi des tubercules qui tiennent à une cause générale.

Lereboullet. — Le poumon étant à peine affecté — Pleurésie seule — et pleine de pus [illegible] — Willemin. p. 154 — bien des pleurésies tuberculeuses guérissent, pus réapparaissent. — on dit alors que la pleurésie a altéré à [illegible] seule la plèvre.

— Si le poumon est affecté, il y a toujours en même tuberculisation de l'un ou des deux plèvres — C'est la loi de Gaudier.

Péricarde — Eichhorst. Tuberculose primaire du péricarde.

Dans Charité Annalen. t. VII. p. 219.

— C'est chez les vieillards surtout que se trouve la péricardite tuberculeuse isolée — cependant dans le cas présent il s'agit d'une sujet de 36 ans — femme.

Il s'agit de tuberculose primaire, isolée, il n'existait pas trace de foyers caséeux ou de tubercules nulle part.

Il y avait une pleurésie double. Les fibrineuse.

— on trouva dans le péricarde une grande quantité de liquide sanguinolent, dans le fond duquel [illegible] caillots. — qui de liquide environ 500 gram [illegible], dans les feuillets du péricarde sont [illegible] de sang, épaissis, granuleux, tous [illegible] de nombreux nodules miliaires [illegible] sur la [illegible] feuillets pariétal, [illegible] 3 petites de [illegible], ulcéreux [illegible] de [illegible] gravement [illegible] [illegible]

Cas de tuberculose primaire du péricarde. Eichhorst. Charité annalen. p. 229.

Dans ce cas qui paraît unique il s'agit d'ulcérations sous-muqueuses de la face pariétale du péricarde tuberculeuses — analogue à ce que l'on voit dans l'intestin tuberculeux — après être restées sans symptômes ces ulcérat. ont donné lieu tout à coup à des hémorragies qui ont déterminé la mort en 24 heures.

— Bamberger a démontré que les ulcérations du péricarde dans péricardite purulente sont on ne peut plus rares.

— Le cas cité est un cas de péricardite tuberculeuse primaire et isolée " il n'existait pas traces de foyers caséeux ou de tubercules nulle part —

— c'est chez les vieillards en général que se trouve cette péricardite tuberculeuse, tandis que dans le cas précédent

Il s'agit d'un sujet de 36 ans.

Anatomie diagnostic.

Péricardite tuberculeuse granulaire hémorragique

Ulcère tuberculeux péricardique

Atrophie jaune aigue myocardique

Pleurésie double séro-fibrineuse -

Hyperplasie ganglionnaire générale

Thrombus du ventricule droit.

Hydropisie anasarque.

L'hémorragie a été la cause de la mort. elle est due à une érosion d'un de petites vaisseaux qui ne pouvait trouver dans le fond de ulcère.

Tandis que la Lésion Tuberculeuse
intestinale est Secondaire, Lors de
2e. Stade — Elle n'en est pas de même
de la Méningite et de la Péritonite
Tuberculeuse qui se déclarent
chez des sujets vierges, au moins
fonctionnellement — En tous
cas l'altération pathologique
prédomine.

Spillmann. 6. 158

~~Répartition~~,

Immunités

sûretés

Immunités p. la tuberculose

— Les muscles animaux } Laennec et Bayle le savaient déjà

Et Rokitansky — Virchow ne l'a jamais vu.

— Il n'en est pas de même de muscles du coeur. { Rokitansky / Virchow

autre, immunité — glandes salivaires, ovaires, oesophage, vagin (Rokitansky).

Virchow n'a jamais vu — gl. salivaires, parotides, pancréas, les mamelles —

— l'ovaire et la gl. thyroïde — rare.

Pour les muscles — Zenker a vu que tubercules miliaires autour de foyer caséeux — [illegible] dans le tissu cellulaire

Il y a des immunités qui sont dues plus — Vagin — Thyroïde dans tub. miliaire,

Muscle. — Myocardium und gehirn. Waldeyer. Virch. Arch. 1866. T. 35. p. 218 —

Marchand. Virch. Archiv {1878. 72. Bd. p. 142 et Lancet. May. 4. 1878.
arsenical. de la branche — syphilitique miliaire — gommeuse. muscle.

femme de 20 ans. — tub. miliaire — myocard. tub. de presque — cirrhose — ontiques — les 2 poumons — un tubercule lenticulaire avec ... la face convexe du foie — plusieurs anneaux de la surface ... de la rate, le parenchyme du rein droit ... en dehors de cela le péritoine et la plèvre libre de tubercules.

— Coeur — péricarde et endocarde sains
— sur le ventricule gauche au voisinage de la pointe, 4 ou 5 tumeurs — les unes font saillie dans la cavité, les autres enveloppées dans la substance musculaire du coeur. —
dimensions de 5 — 9 mm. long, 7 — 11 mm. large.
une thèse a paru sur la tuberc. de myocarde — à Breslau 1865. — Haberling.

Cruveilhier aussi a vu de 2 tumeurs blanches ... c'est dans le ...
... que ... Zenker ...

Muscle

Tuberculum verrucosum.

Paul Chavasse

Étude sur la tuberculose des organes urinaires

(Reins. Uretère. Vessie)

Paris 1872

" au point de vue pratique la tuberculose des organes urinaires offre réellement peu d'intérêt)

— causes déterminantes toujours inconnues — Symptômes peu nets — diagnostic difficile — traitement palliatif —

Le principal intérêt est dans l'anatomie pathologique.

Tuberculose urinaire : D[r] Tapret arch. de Médecine. mai 1878

près Civiale.

— Il faut séparer la tuberculose urinaire — de la génitale.

Il y a une tuberculose primitive de la vessie. — c'est Jacques... Guyon.

— aussi Jean. ... (France médicale 27, avril 1878. —

S'il est vrai que fréquemment la tuberculose urinaire est un complément dans le cours de la tuberculose pulmonaire, il a des formes les malades se présentent à une époque avec des symptômes de cystite spéciale, alors que rien n'a éveillé l'attention du côté du poumon.

— Rein. Aucun ... a peu ... affirmé que la phtisie rénale peut évoluer seule. —

— Tapret ... démontré que la vessie peut être le siège exclusif, ou primitif, d'où la lésion rayonne vers le rein, d'un côté, l'urèthre de l'autre, jusque vers la prostate et les testicules ce qui est le plus ordinaire.

Il distingue
- 1° Tuberculose isolée du Rein.
- 2° Tuberculose isolée de la Vessie.
- 3° Tuberculose isolée ou primitive de l'urèthre.
- 4° La tuberculose isolée de la prostate.

revenant primitive.

Si à chacune de ces formes, il ... un tableau symptomatique correspondant il ... à permettre le diagnostic !

Intestin grêle. Gros
Testicule.

Reclus, testicule p. 75

Il y a des prédispositions locales — le cœur incomplet, l'ectopie, la cryptorchidie — alors la santé générale peut être indemne. L'organe exposé seul souffrira sous l'influence de la moindre cause traumatique.

Il y a comme dit Cruveilhier, des tuberculisations localisées qui tiennent à des causes locales, et des tuberculisations générales qui tiennent à des causes générales !!

— La tuberculisation solitaire est fréquente dans le testicule. Elles naissent, se développent, restent stationnaires ou même s'évacuent et disparaissent, sans retentissement, sans généralisation.

— Il est homme qui il y a 10, 20 ans, ont été tuberculeux — et se portent bien et n'ont aucune trace de lésion pulmonaire.

— Ce n'est pas que les tubercules des testicules aient été éloignés, préservés à l'abri des hôpitaux, qu'on les a vus fréquemment à l'abri des généralisations comme le croyait Velpeau.

La tuberculisation est partout la même. Le tubercule du testicule n'est qu'un cas particulier de la tuberculose.

— Mais il n'est pas vrai de dire avec Dufour, contrairement à l'opinion professée de Velpeau, Roux, Vidal de Cassis, que la tuberculose génito-urinaire doit tôt ou tard se généraliser.

Car il y a certainement des cas où elle ne se généralise pas —

Statistiques de Reclus.

1° Cas où l'état du poumon a été étudié

Seulement par l'auscultation — 30 cas

Tub. génitale et pulmonaire 16

Tub. génitale — solitaire — 14

Salleron qui plus loin il dit que chez les soldats sur 51 cas le poumon pris seul^t une fois

Cas d'autopsie — 30 cas

— Tub. génitale et pulmonaire 20

Tub. génitale seule — 10.

---

avant 15 ans, la tub. testic. est très rare — ainsi sur 185 enfants tuberculeux, le testicule est pris 1 fois

Vieillesse, rare aussi mais rare. Sur 104 autopsies Dicter Dupont pas un seul testicule tuberculeux.

C'est entre 15 et 35 ans au moment de l'activité fonctionnelle de la glande.

Sur 500 phtisiques de Bordeaux. 11 seul^t tuberculeux du testicule. — sur 1317 tuberculeux à l'hôpital Lariboisière 33 tub. du testicule.

—

Orchite tuberculeuse { 15 cas Reclus . p. 87 [illegible]

Début des plus brusque

Pris à coup dans le cours d'une bonne santé

Douleur vive du testicule

Le testicule qq. semaines, mais le gonflement porte sur l'épididyme qui déborde de toutes parts épididyme dur rénitent, surface inégale [illegible] le testicule rougit et la [illegible] de bosseler.

— il y a de l'hydrocèle

[illegible] d'aspect [illegible]

Vers le 4e jour la douleur diminue — alors le gonflement diminuant —

[illegible] de l'épididyme

suppuration rapide vers le 25 jour

du pus crémeux [illegible]

mais une fistule persiste [illegible]

à la surface granuleuse —

La tuberculose [illegible]

envahit le 2e testicule —

— Le malade devient } tuberculeux quand qq. le suit

En somme — L'orchite se trouve la première manifestation de la tuberculose et parmi les contusions parfois dans le testicule, dans tendance à la généralisation

Tuberculose génitale de l'homme.

Testicule — Le siège de prédilection est l'Epididyme — Il est bien remarquable que le testicule soit pris indépendamment de l'Epididyme 3 cas seulement cité par Reclus.

— Ricord a dit qu'on dirait trop d'Inoculation = quand il y a des tubercules dans qq. parties des voies génitales, il y en a dans l'epididyme =

qq. l'Epididyme seul, mais le testicule souvent envahi en même temps.

Ainsi sur 24 cas, Reclus en trouve [illegible]
avec [illegible] — Epididyme et testicule — 27 cas
Epididyme seul — 7 cas

Le Canal déférent lésé à son origine et à sa terminaison

La tuberculose des vésicules séminales et de la prostate est habituellement liée à celle du testicule quoi qu'en pensent des auteurs, comme Dufour et Cruveilhier
Ces réunissent dans une même description.
Cependant qq. prostate atteinte à l'exclusion d'autres organes

La muqueuse uréthrale est aussi siège de lésions importantes — les petites pertes et membraneuses.
(un cas pour Naciculair. Reclus. Cellul. glande)

Fièvre: Rétention ~~urine~~ ~~granulation~~ urinaire { combinaison de la lésion génitale et urinaire
— alors vessie — et à partir de la Rétention vers les uretères, les bassinets

Reclus. p. 105

Le tubercule du testicule est une affection à marche essentiellement chronique —

mais à côté de ces formes bénignes de Velpeau et de Vidal de Cassis — Tuberculose locale de Cruveilhier.

— Il y a des formes graves, à généralisation rapide — qui s'étendent aux voies urinaires —

Köster. Ueber fungöse Gelenkentzündung
Virchow's Arch. t. 95, Berlin 1869.
t. 48.

Cornil. Arch. de Physiologie 1870. { manus, tubercules [illegible] / (Nouveaux [illegible]) / Arthrite tuberculeuse [illegible] }

— [illegible] de [illegible] — tuberculose du poumon
— douleurs perte de mouvement de l'articulation
du coude droit, [illegible]: douleur à la pression
pas de craquement.
on diagnostique tumeur blanche.

Autopsie — granulations, tuberc. [illegible]
[illegible] tuberculeuse.
Cavernes au sommet du poumon droit

— Coude — cavité [illegible] pas bien [illegible]
— [illegible] articulaires fongueuses, molles
— [illegible] et [illegible] de pus.
— [illegible] opaques, [illegible]
— on remarque petites graines saillantes, opaques
au centre jaunâtre.
— Synoviale épaisse de [illegible]
cartilages articul. blanchâtres un peu
— opaques.
— Les épiphyses de l'humérus, radius et cubitus
ne présentent pas d'amincissement des trabécules
du tissu spongieux — les trabécules paraissent
plutôt épaissies.

— Histologie — tissu [illegible] granulat. tubercul.
par [illegible] —
ces granulations [illegible] dans ces tissus comparables
à celles qui dans les tubercules pleuraux
forment la couche au centre de la
quelle se développent les tubercules.

— Cartilages — vascularisation et ulcération, dans les couches superficielles —.

Cellules des capsules en prolifération.

— Dans les trabécules osseux des synoviales, les corpuscules osseux se remplissent par des granulations graisseuses.

— Dans formes spéciales d'arthrite chronique & bien des cas de ce genre dit sous le nom de tumeur blanche.

J'ai pas tumeur blanche

cas — 18, par de dégénérescence granulo graisseuse

intermède des cellules du cartilage.

1. par d'accroissement des trabécules osseux

2. par de l'usure des corpuscules osseux.

3. par [illegible] raréfiante des ostéophytes.

4. ni carie, ni ostéite raréfiante des [illegible]

— Il est vraisemblable que cette affection confondue avec arthrite scrofuleuse — les 2 affect. cependant sont [illegible] bien distinctes —

La symptomatologie de l'arthrite tuberculeuse

est tout entière à faire.

Trouve avec les granulat. dans tous les genres de fongosités, non seulement dans celles qui proviennent de la tuberculose des synoviale en dehors, mais encore dans les trajets fistuleux, les abcès périarticulaires. dans 2 cas, les os.

— dans la tuberculose miliaire (un fréquemment) des jointures, les superficielles dans une synoviale les injectée mais peu semblé.

— La tuberculose fongueuse. tout des genou stationnaire.

J'ai vu 10 cas — 5 cas analysés

5. cas d'amputation — dans 1 seul il y avait tubercules au poumon des poumons ? —

arthrite blennorragique

Six observations.

Trois garçons. 9 ans 12 fr 14.

Trois filles 3 ans fr 6. 9

Antécédents héréditaires. Dans trois cas l'un des parents est mort phthisique. Le début a presque toujours été sans une cause appréciable; une fois, il y aurait eu chute sur le genou, mais l'enfant s'est relevé et a continué sa journée sans se plaindre; ce n'est que le surlendemain qu'il a commencé à boiter. Aucun d'eux n'a eu d'atteinte rhumatismale antérieure, et ils ne paraissent pas avoir été exposés aux influences rhumatismales. Ils sont presque tous une apparence chétive, délicate, aucun d'eux n'a eu ni maux de Pott ni affection thoraciques graves; ils n'ont pas non plus de traces de scrofules. Ils étaient atteints de l'épanchement du genou depuis un temps variable entre huit jours et un mois, lorsqu'on les a conduits à l'hôpital. Cet épanchement a pour caractère d'être abondant sans être douloureux, sans amener une tension extrême de la synoviale; il est sujet à quelques variations et chez deux ou trois de nos sujets, le repos au lit a suffi pour diminuer la quantité du liquide, mais cela n'a été que une amélioration passagère, car l'épanchement a été reproduit dans des proportions aussi considérables. Ce fait m'a conduit à suivre une pratique que j'indique maintenant pour n'y plus revenir et qui a consisté dans l'immobilisation du membre dans une gouttière

fabrication avec adjonction de cautérisations au fer rouge disposées en rayons autour du genou en ménageant la rotule par devant. J'ai dit que lorsque encore appelé à l'immobilisation par la compression et d'autres méthodes.

Chez deux de mes malades après deux mois environ de traitement, la guérison de l'épanchement a été obtenue. L'un deux a repris ses jeux et ses habitudes depuis plus d'un mois lorsque il survint une méningite qui l'enleva en peu de jours. J'ai envoyé l'autre à Berck où il est encore et paraît bien portant.

Chez les autres sujets, l'affection a eu une marche analogue ou un peu différente. Deux sont encore dans mon service avec un épanchement articulaire datant de plusieurs mois et qui ne paraissent pas à se résoudre. Chez eux comme chez les précédents, la synoviale ne présente pas un gonflement comparable à celui des tumeurs blanches ordinaires, elle est bien un peu épaissie en certains points de son trajet, mais ces épaississements sont partiels, à peine reconnaissables et ne font nullement corps avec les os. Le squelette d'ailleurs examiné avec la plus scrupuleuse attention ne révèle ni augmentation de volume, ni douleur à la pression ni à la percussion. Il n'y a pas non plus d'élévation de la température appréciable à la main. Pourtant chez les six malades sans exception, en explorant, tant la pression et pour

fait point de synovite du genou. J'ai développé avec la plus grande facilité de la tuberosité interne du tibia une tumeur que je n'ai pu rattacher qu'à un bourgeonnement osseux fait à l'épanchement avec l'attache du ligament latéral interne à ce niveau. Cette tumeur est constante dans mes dix observations.

Les deux derniers sujets sont morts de méningites tuberculeuses après avoir eu comme les précédents au début un épanchement du genou. Cet épanchement avait succédé un épaississement de la synoviale qui me fit croire pendant longtemps chez l'un d'eux à une tumeur blanche ordinaire fongueuse sans lésion osseuse reconnaissable. Je la traitai pendant huit mois par l'immobilité et la révulsion. Cet enfant allait bien et je le considérais comme guéri avec une ankylose fibreuse, c'est à dire incomplète, lorsqu'elle fut prise de méningite à laquelle elle succomba. Le dessin non coloré indique l'état du genou et montre des adhérences fibreuses réunissant le genou qui n'offrait plus de fongosités.

Chez le dernier sujet enfin, l'épanchement du genou résorbé, on trouvait que la synoviale présentait des plaques molles fongueuses qui indiquaient qu'une transformation s'était opérée. Tout me portait à penser cependant que l'affection était encore curable lorsque survint la méningite. Le dessin colorié représente l'état de ce genou.

Dans les deux autopsies que j'ai faites il coexistait avec les

lésions ordinaires de la méningite tuberculeuse que l'on granulations dans les poumons, sur l'un des deux cadavres, il y en avait aussi sur la plèvre ; mais elles n'étaient ni chez l'un ni chez l'autre abondantes dans le poumon où elles se présentaient sous la forme de noyaux plus petits que la tête d'une épingle, semi transparents, isolés et quelquefois groupés en petit nombre. J'ai vainement cherché des tubercules dans les os, et je n'ai trouvé qu'une fois dans le tibia une tache blanchâtre, peut être tuberculeuse dont l'examen histologique sera fait.

En résumé : Un épanchement articulaire subit aux moyens ordinaires, sans gonflement de la synoviale, sans altération des os, tel est le début de l'affection. Deux fois déjà cet épanchement s'est résolu complètement et tout me porte à croire qu'il en sera ainsi pour deux autres que j'ai encore sous les yeux. Tandis que il a été suivi de cet épanchement de purulence chez mes deux derniers faits. Trois fois la méningite est survenue et une fois après quelques jours seulement de fièvre d'un mois. Mais je n'ai pu faire l'autopsie de ce genre.

Tuberculose primaire cutanée

Tuberculide = Gomme

Scrofuleuse !

Lupus.

Tuberculum
genitale.

Tuberc.

Tableau du malade.

debut par le rein, prostate, vesic.
les symptômes significatifs de participation du col vesical.
Le Rein et la prostate se prennent sans revelation bien nette.
— C'est la tub. de vessie qui donne à la maladie sa physionomie circonscrite
à ses 20 à 40 ans. — a perte de sang sans douleur, sans cause locale
passives —
— L'hemorragie recidiva —
Le col vesical d. pred de contraction spasmodique — il y a retention
d'urine, besoin à uriner
— envies frequentes d'uriner impérieuses et douloureuses au commencement et
à la fin.
a petits dépôts purulents striés de sang
— et qq urines claires, qq normales, aqueuses
— ou troubles, louches, [illegible]
— on explore urethre — blennorrhée prolongée
— Vessie petite et dilatée. Col douloureux, [illegible]
induration limitée au bas fond.
Sans corps, étrangers.

[illegible] [illegible] [illegible] [illegible] [illegible]

Tuberculose des organes genitaux. chez l'homme.

Thèse de Reclus. p. 75.

— La tuberculisation solitaire est fréquente dans le testicule. Le tubercule naît, meurt, s'élimine, sans généralisation.

On rencontre des hommes qui il y a 10, 20 ans ont eu le testicule tuberculeux — Ils se portent bien et n'ont que les cicatrices des trajets fistuleux —

Ce n'est pas que la tuberculisation du testicule ait été clinique spécial comme le voyait Velpeau, Ricord, Vidal de Cassis.

Dufour combat cette opinion en disant que tôt ou tard les tubercules génitaux arrivent à généraliser. Cela n'est point nécessaire et à l'appui de son dire M. Reclus donne une statistique.

~~[illegible]~~ 2. statistiques: 1° 30 cas où on étudie seule la tuberculisation seulement. —

Tuberculose génitale et pulmonaire. 16

Tuberculose génitale solitaire. 14.

Sallem a été trop loin quand il a dit que chez les soldats

Sur 50 cas, le poumon fut seulement une fois —

Cas avec autopsies — 30.

Tuberculose pulmonaire et génitale — 20

Tub. génitale seule — 10.

— C'est entre 15 et 35 ans. au moment où l'activité fonctionnelle de la glande — donc exception radicale à la loi de Louis.

organes génitaux

— Chez la femme,

Tubercul. des org. genitaux chez la femme 1865

- Brouardel, p. 8. — La Lésion tuberculeuse se dépose ici en même temps dans le poumon et les organes génitaux — 99 fois elle se développe primitivement dans les organes génitaux constituant alors une sorte de phthisie pelvienne primitive — cette thèse on [illegible] à cela — forme à tort considérée comme très rare —

— chez la femme la tuberculose génitale ne passe guère aux voies urinaires — ainsi sur 115 cas [illegible] une seule fois — chez la femme la tuberculose urinaire existe, mais indépendante de celle des [illegible] la tuberculisation génitale paraît souvent chez l'homme au contraire la tuberculisation génitale précède celle des organes génitaux —

— Comme preuve de phthisie pelvienne primitive. cite une observation de Siredey — pas de tubercules ailleurs.

— dans 2 autres tuberculose du poumon croissante, mort entraînée par tuberculose pelvienne —

— . Tubercul. des trompes, utérus, vagin. —

— Klob. tuberculose isolée du vagin dans [illegible] — mais les poumons ?

Virch. archiv Bd 62 — p. 264.

Ensorcelle, Solitaire
des cerveaux noires.

Bien étudiés par Cruveilhier — Virchow

Gracds. Tubercules solitaires des centres nerveux volume, formes solitaires — conglomérats — volume : pois, grain de raisin, noix — occupent rarement la substance blanche — ces substances grises de la périphérie ou des noyaux centraux. — Le plus souvent on les rencontre à la limite des 2 couches.

— Le cervelet est l'organe le plus fréquemment atteint — Les tubercules du pont de Varole, leur siège —

— Dans l'âge avancé les tubercules sont de plus en plus rares — cependant Cruveilhier Tubercule spinal

— Ils se développent toujours dans la substance grise —

Presque toujours ils coexistent avec tubercules cérébraux — Lésion Cliniques. latente ? ou localisation —

Rech. lésions tuberculose locale.

Virchow — presque toujours on trouve en même temps qu'eux des tubercules dans les autres organes.

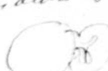
Mais dans la plupart des cas l'évolution des tub. cérébraux est tellement avancée, qu'il y a lieu de penser que ces foyers ont précédé les autres

— Il y a lieu de considérer certains de ces cas comme des exemples de tuberculose cérébrale primitive —

Rarement cette tuberculose cérébrale survient comme métastase —

Jaccoud et Wernher avec Hasse une cause traumatique (Virch. p. 112)

Cruveilhier : exprime le fait actuellement ses doutes 1° La tubercule cérébrale font rarement partie de la diathèse tuberculeuse — 2° Les tubercules cérébraux peuvent exister indépendamment de la tuberculose des autres organes.



Dans le cas de Virchow symptômes mentaux. Céphalée — neuralgie à gauche — épaule gauche, membre gauche de la poitrine, membre supér. gauche paralysie — muscles fessiers très pâle — région des 3e et 4e vertèbres cervicales — nerfs du plexus brachial — leurs racines au niveau des trous de conjugaison

Friedlaender

Recklinghausen.

gros tubercules sébacés ou multiples, (malgré leur longue durée), restent isolés

(du cerveau.

Loi de Louis.

Exceptions à la Loi de Louis. ou phthisies annuelles de Sidney.

Recherches anatomiques, pathologiques et thérapeutiques sur la phthisie. 2e édi. 1843. — 1re édi. 1825.

— 125. observat. étudiés et calculées d'après la méthode qui lui est propre —

Loi p. 182 — ~~[illegible]~~ les tubercules ou les granulations grises ou transparentes, ou la matière grise ou mate, paraît propre aux phthisiques.

Je n'en ai observé dans aucun cas, et dans aucun individu, après 15 ans, qu'il n'y en eut dans les poumons.

De manière que leur existence, dans ces derniers organes, semble une condition nécessaire de leur développement dans les autres parties.

— D'ailleurs, j'ai toujours vu, à l'exception d'un cas la matière tuberculeuse beaucoup plus avancée dans les poumons que partout ailleurs.

Les tubercules qu'on trouve dans les autres parties du corps sont à peu près au même degré de développement.

— Il cite 2 exceptions — 1° Sujet mort de fièvre typhoïde
poumons sans tubercules. — petite granulation
2° [illegible] tub dans glandes mésentériques. ([illegible]
par tubercule — mastication. —

— Autre exception. Sujet de 40 ans. rien dans le poumon, mais tubercules dans le cerveau et le cervelet.

(Lésions concomitantes tuberculeuses ~~ou~~ ~~[illegible]~~ peut considérer comme telles.

1/5 des cas. Ulcérat. du larynx. trachée

5/6 — ulcérations larges de l'intestin grêle et gros intestin

1/16 des cas rate. Reins idem

plèvre, prostate — utérus

— quelques cas, granul. tuberculeuses dans les méninges (Sylvius)

— quelques cas

primaire

Tuberculose localisée

du foie ;

Orth. Virchow ... t. 66.

Il y a une sorte d'antagonisme entre la tuberculose localisée et la dissémination, au moins en général.

autrement dit la tuberculose primaire et la secondaire diffèrent en ce que = Les organes qui comme le Cerveau, les organes génitaux, sont peu disposés pour la forme disséminée sont disposés aux tubercules primaires.

au contraire, la rate et le foie qui sont qs. toujours affectés dans la tub. généralisée (le foie surtout) où les tub. solitaires sont plus fréquents qui ne sont pas lésés primitivement.

Foie. = Il se trouve là sous 2 formes 1° tuberculose miliaire disséminée.
et 2° tuberculose des conduits biliaires —

1° Tub. miliaire disséminée. C'est de beaucoup la plus fréquente
— elle se distingue par la petitesse des nodules, qui va jusqu'à nécessiter l'emploi du microscope.
— C'est pourquoi sa fréquence a été jusque dans ces derniers temps inaperçue.
— Ils peuvent atteindre le volume d'un grain de mil. Ceux d'un pois sont rares.

2° forme — plus rare. affecte les voies biliaires — les nodules peuvent acquérir le volume d'un pois
— Ils ont une couleur verte, qu'on voit au centre du nodule et tient au mélange de pâte biliaire.

3° De gros nodules, comparables aux tubercules solitaires du Cerveau, ne se rencontrent dans le foie que rarement
— Je ne trouve cela mentionné que par

— quel... ...ais en ... depuis
mais il [illegible] ... ou dans cette forme. ... , ...
... — c... [illegible] qu'ac... il y a des conduits biliaires

... rapporte 2 cas

1° cas — Tuberculose miliaire generalisée, nombreux tubercules disseminés du foie — en outre dans le lobe droit un nodule gros comme une amande. et plusieurs autres plus petits dans connexion avec les voies biliaires #.

2° cas — Peritonite tuberculeuse, caséification des ganglions porte et retro peritoneaux, tubercules disseminés peu nombreux du foie — une tumeur tuberculeuse du foie lobée, grosse comme le poing

---

1°

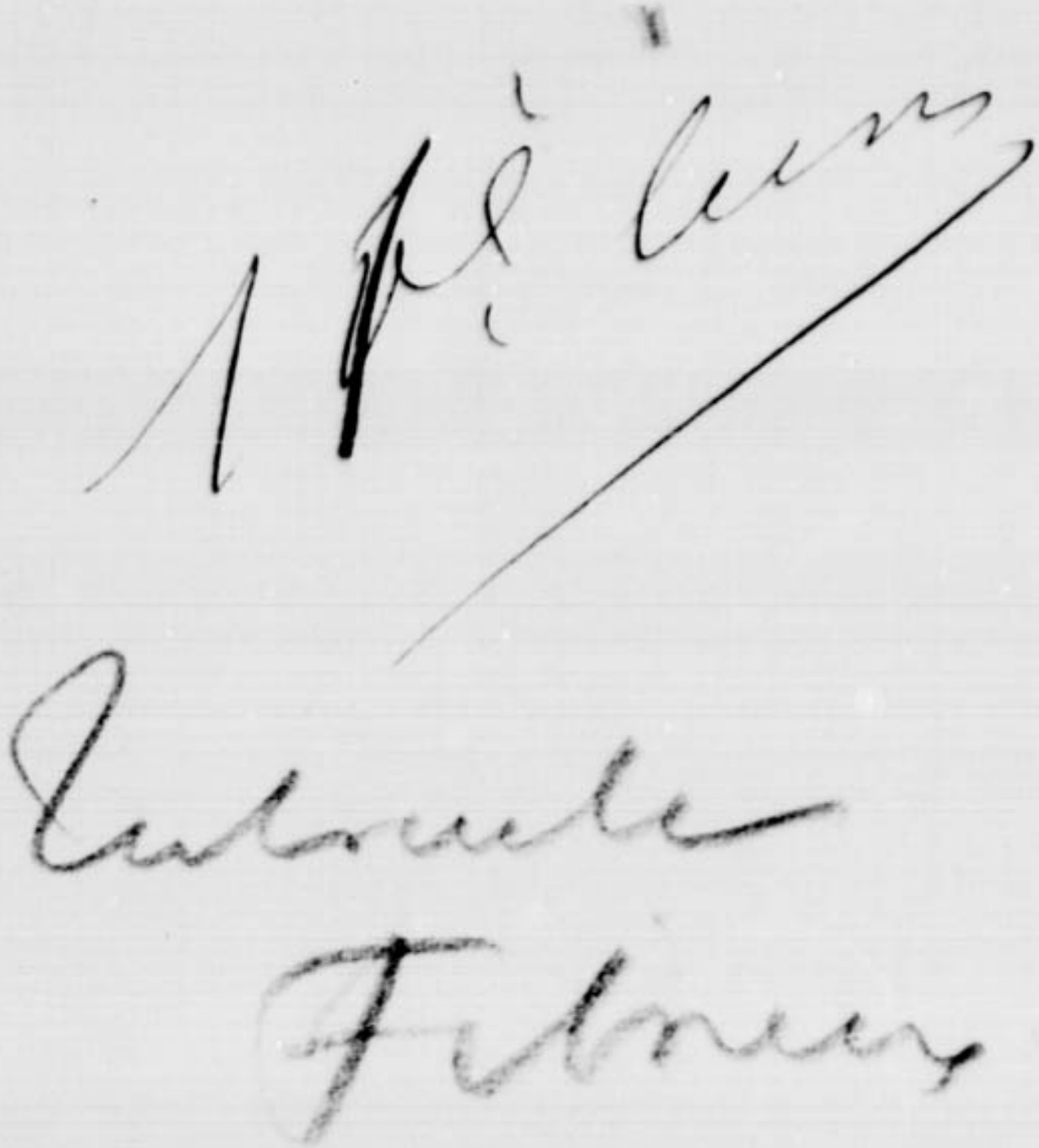
#. Tuberculose miliaire generalisée, tub. dans les poumons, rate, reins foie, testicule droit choroïde —
Gros tubercule caseeux dans le lobe droit du foie —
ulcerat. tuberculeuse du larynx, epiglotte, pharynx —

#. Le 2e cas. Interessant parceque les nodules étaient pendant la vie sentis à travers les teguments et avaient donné lieu au diagnostic. Leberkrebs.

— Le noyau etait constitué par une certaine ... de la ... comme de petits nodules gros ... comme voyant autour,
dans le cerveau —
Ces petits nodules etaient des tubercules reticulés avec cellules geants —

Il y a lieu de croire que dans ce cas l'affection du foie etait primaire — et que consécutivement l'infection des glandes lymphatiques, porte et retroperitoneale —

— chez une femme de 59 ans
— Il y avait probablement cholecyste chronique.
— La vesicule du fiel comprise dans la tumeur etait ...



Mais il est un dernier mode d'évolution des sécrétions folliculaires : il est de ceux question intéressante à laquelle se rattache celle de la création des tubercules. Questions connexes, mais qui à présent ne se confondent point absolument.

— Vous savez que le tubercule est essentiellement constitué des éléments cellulaires qui sont formés les plus tempérés sur du ... un tissu adénoïde. 1° granulation grise de ... constituée un tissu ... 2° granulation réticulée — 3° granulation fibreuse ... granulation tuberculeuse. ... tissu tuberculeux ... un ... de tubercule de ... on désigne ... sous le nom de tubercule de ...

Tubercule fibreux.

Evolution fibreuse.

Malmet p. 9

transformat. fibreuse du tubercule miliaire

Bayle (épais pluralité) distingue. la phth. tuberculeuse — de granul. mil. à châtaigne — Ils se ramollissent au centre

Et dans la phth. granuleuse — granul. miliaires transparentes, luisantes — cartilagineuses — grand. millet à grain de blé — Jamais opaques, ne se fondent pas

ceci les distingue des tub. miliaires qui sont toujours gris ou blancs et opaques et se fondent

Il y avait pour lui (unicité) dualité (en tuberculeux) — Laennec ne l'a pas admise. — il croit que l'un passe à l'autre

Cruveilhier appela les granul. de Bayle des granulat. de guérison.

— Virchow: de la nature tub. phtisique. Le nodule se compose en grande partie d'un tissu connectif dense — Ils se distinguent des cellules par leur aspect gris clair, perlé, transparent.

Leur concomitance avec des tubercul. plus ou moins; leur tendance à la dég. caséo. graiss. les rapprochent des tubercules.

— Thaon. dit que la granulat. fibreuse est bien un tubercule. —

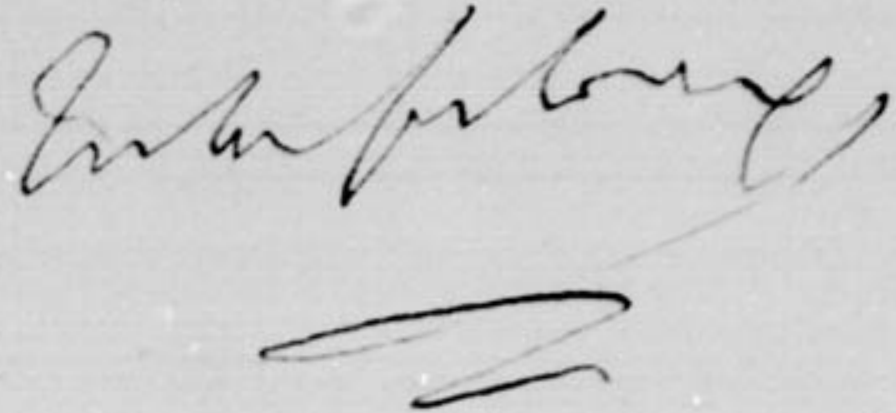
— Grancher 2. manière. Mais seulement le tubercule peut guérir par sclérose, mais toute granul. qui se développe lentement devient fibreuse et guérit. C'est donc la transformation en une fausse anatomique sclérose inoffensive.

— aussi appelle-t-il le tuber. une néoplasie fibro-caséeuse.

— Le tub. fibreux, contrairement à ce que dit Virchow, ne subit pas la dég. caséo-graisseuse — c'est le dernier terme de l'évolution de tubercule cellulaire — C'est lorsque l'évolution du tubercule se fait lentement et qu'il parcourt tous les stades : embryonnaire, adulte, fibreux.

Mais il peut être arrêté dans son développement, devient alors calcaire. —

— Les 2. évolut. peuvent se faire chez le même individu — mais elles constituent souvent alors des types séparés et cliniquement Bayle a trop séparé... en admettant phthisie tubercul. et phthisie granuleuse — Tous deux sont subordonnés à la diathèse tuberculeuse.



Cruveilhier.

Curabilité de la phthisie pulmonaire. divers modes de guérison — p. 614.

— Il résulte de mes recherches, 1° que anatomiquement et pathologiquement les tubercules en général et les pulmonaires en particulier, sont curables à toutes les périodes de leur développement — granulation solitaire, agrégats tuberculeux.

2° qu'il existe des granulations de guérison, des tubercules de guérison, des agrégats de guérison.

Laennec ne s'était occupé que du mode de guérison des cavernes tuberculeuses.

— J'ai pu reconnaître à la Salpêtrière que le tubercule est curable, à toutes les périodes de son évolution —

1° granulation miliaires solitaires — granulation de guérison

2° granulations groupées — guérison

3° ~~agrégats~~ petits foyers. — enkystement.

—

1° granulat. miliaire de guérison — petit kyste avec centre crétacé — matière

2° Un gros du volume d'une grosse noix — il ne coupe gris ardoisé — il ne paraît pas de centre caséeux

— d'autres enkystements du même.

Flammen.

— Tubercule fibreux.

a — Dans le tub. miliaire très habituel, que zone interne fibreuse avec éléments, granule et confluent le centre caséeux

b Dans Agglomération tuberculeuse cas de granulation —

C. Agglomération tuberculeuse et tub. miliaires fibreux

— ulcérat. tuberculeuse du caecum.

— broncho pneumonie disséminée et nodules fibreux, avec cell. embryonnaires et qq. cell. géantes

—

Transformation du tubercule en un tubercule fibreux. —

Schüppel. dans les ganglions lymphatiques fig. 103. —

— Le tubercule fibreux ne diffère en rien d'après lui de l'origine du tubercule à cellules

La transformation fibreuse se fait parce que les mailles du reticulum s'épaississent, aux dépens des cellules qui s'atrophient

— les mailles du reticulum se changent en une masse fibro conjonctive transparente, 109 homogène, cependant striée vaguement.

— Mais pendant que cela se fait à la périphérie, le centre se nécrose et le tubercule fibreux ressemble assez à un petit fibrome fibreux [illegible] le centre caséeux [illegible] avec centre caséeux. Le centre caséeux peut se résorber, et alors on rencontre un petit fibrome de la grosseur d'un tubercule.

En même temps, dans la zone externe les cellules se disloquent dans le type fascicul de manière à former un kyste.

1° Tubercules fibreux. — Expos. elementaire

Schappes — C'est la transformation du tubercule en tub. fibreux.
Les mailles du reticulum s'epaississent — dans les espaces des elements cellul. qui s'atrophient. Schappes
— c'est une masse fibro-conjonctive, homogène, plus ou moins transparente — vaguement striée. Keratoïde.
— mais il semble que cela se fait à la peripherie le centre se necrose. Le tubercule fibreux ressemble à un petit fibrome miliaire avec centre caseux.
— Le centre caséeux peut se retracter.
— La zone externe les cellules se developpent deux
— La zone externe les cellules se developpent deux — la zone externe les cellules se developpent deux

le type [illegible] — dans le poumon très souvent aspect. Typ. classique : cirrhose. [illegible]

2° agglomerat. tuberculeux — [illegible]
— Idem.

2° cas — centre caseux. cellules geantes
— Rien. vide caseux — Cas de gangrene [illegible]
~~ant. [illegible]~~ — agglomerats fibreux
— Ici laryngite — [illegible] tubercul. [illegible] — les cas

(partiels Mercredi. Lalou

femme de 40 ans. sans. 10 ans après

Cette question le rattache. celle de la curation des tubercules. C'est un sujet que Cornil a traité avec prédilection et son talent.

p. 604.

Il résulte de ses recherches et de ce que avait dit Lalouy les tubercules en général et les pulmon. en particulier sont curables à toutes les périodes. Aussi bien le granulat. Laennec que les ayst. anatomistes.

— 2° qui il existe des granul. de guérison, des tub. de guérison — des agrégat de guérison.

Laennec ne s'était occupé que des modes de guérison

des Cavernes.

1° granul. de guérison — petits kystes crétacés
2° — d'autres. pois. — cellules pr...
3. d'autres — qui ...

Tub. aigu { miliaire / massive
Tub. subaigu { miliaire / massive
Tub. chronique primitive { miliaire. / massive

Ceci est la granulation de Bayle

Manu f. 13? — Bayle n'a pas connu la granulation de la phthisie aiguë.

Il n'a jamais parlé que des formes chroniques

" La phthisie granuleuse est une espèce des plus chroniques

— La granulation décrite par Bayle — est demi transparente, très dure, grain de grêle — elle ne devient jamais opaque

c'est le terme très commun dans phthisie chronique et alors presque chronique.

Phthisie granuleuse [illegible] de 99 ans

— Jamais dessous de fibres primitives ou fibrose contractive. Car dans lequel on pourrait dire granulation de guérison.

Marèt 2,

3. ca. Eud. cutaneus.
Eud. fibro-cutaneus
Eud. fibrosus

— Réaction contre la
doctrine de Virchow,

en Allemagne

Schaffer. p. 138.

- Es besteht für mich der Verdacht, dass noch manches Kärtige Tuberkelleben Ursprungs sein könnte, was man jetzt nicht dafür gelten lassen will. Das eine solche Revision namentlich der Lehre von der chronischen Lungentuberculose zu gute kommen würde, und dass die Grenzen zwischen infectiösen und tuberculösen Processen sich hier wesentlich anders gestalten, als man bisher anzunehmen pflegte, davon habe ich mich bereits durch eine Reihe von Untersuchungen neuerdings kommen

Confluence et Infiltration

Schuppel. — Infiltration et
coalescence.

— Calcification.

Schueppel, p. 108 Infiltrat Tuberculose 1

quelquefois " die Tuberkel sind so reichlich und auf grösseren Strecken in so gleichmässiger Vertheilung vorhanden dass sie vielfach unter einander zusammenfliessen und dass das sie noch scheidende Zwischengewebe selbst mit in die Necrose und Verkäsung hereingezogen wird. dies ist die sogenannte Infiltrirte Tuberculose,

Ce n'est pas ici de l'infiltration proprement dite en ce sens que les élém. tuberculeux ne sont pas infiltrés dans l'intervalle des élém. de la glande. Toujours le tubercule est dans la forme nodulaire. Mais les nodules peuvent devenir coalescents. le tissu intermédiaire se trouve compris, privé de vaisseaux. et pris avec l'aspect à l'œil d'une pomme de terre. (que l'on considère comme le 2e stade de la scrofule.

Pour moi (p. 110) L'état caséeux des glandes scrofuleuses est de provenance tuberculeuse — Dans l'hypothèse de Virchow on voit que la calcification

dépend de ce que l'inflammation a pour résultat l'accumulation dans la glande de corpuscules lymphatiques qui subissent-là dég. graisseuse.

Mais je suis revenu de cette idée parceque toujours j'ai trouvé au microscope des parties caséeuses des tubercules, et dans l'amas lui même des vestiges de tubercules; soit des cellules géantes.

— autrefois on regardait la glande scrofuleuse caséeuse comme tuberculeuse. aujourd'hui Virchow a changé tout cela — il faut y revenir.

p. 112. Wenn ich auf die Anwesenheit von Tuberkeln in der sog. Drüsentumoren die Ansicht basire, dass die käsige Masse wesentlich aus Tuberkeln hervorgegangen ist, und dass eine Verkäsung in der Mehrzahl der Fälle überhaupt nicht vorliegen würde, wenn keine Drüsentuberkulose bestände, so wird mir von gewissen Seiten wohl der Einwand gemacht werden, dass die vorhandenen Tuberkel erst secundär

Zur Verkäsung der Drüsenzellen hinzugetreten sein können. als le caseum dépendant de la fonte des ganglions non tuberculeux, a produit une infection de voisinage et en conséquence une éruption tuberculeuse secondaire.

Mais a fait tu une remarque de forme pe- Il faudrait demander d'abord que le caseum résulte bien de la glande hypertrophiée simplement – D'autre côté dans les amas caséeux on trouve de vestiges du tubercule et en particulier les cellules géantes – puis dans une série de glandes venant d'un même sujet, on trouve les unes caséeuses seulement et les autres tuberculeuses non encore caséeuses.

— Ich möchte uebrigens nicht den Irrthum aufkommen lassen als wollte ich die Möglichkeit des käsigen Zerfalls von hyperplastischem Lymphdrüsengewebe überhaupt bestreiten.

Er kommt in der That neben
und in Folge gleichzeitiger Tuberculose
vor. Er kommt vor in den
hyphosen Lymphomen der Mesenterien,
und vielleicht auch, aber
gewiss nur in sehr beschränktem
Umfange, in denen (nicht
Tuberculösen) Lymphomen bei
Scrofulosen, Was ich übrigens
noch nicht als erwiesen
ansehe.

Cerveau. p. 479

Wagner. Les agglomérations de follicules peuvent atteindre le volume d'une cerise, d'une noix — ainsi dans le cerveau, le testicule, les Lorenzini.

Le follicule a de 0,35 à 0,15 de diam.

Cornil et Ranv. p. 208

Granulations confluentes.

– Dans le poumon les ilots
– de gran. confl. sont confondus
avec les ~~[illegible]~~ fr. caséeuses.

Mais il en est de même dans
les os.

Infiltration

Comp et Ramier. Infect. tuberculeuse.

Dans le poumon: la granulation
tuberculeuse, pneumonique et
intra alveolaire. Mais on voit aussi
des tubercules qui occupent l'intérieur
de plusieurs alvéoles dont les
cloisons élastiques sont conservées.
Le tissu embryonnaire dans ces cas
végète des parois alvéolaires dans
l'intérieur des alvéoles, et il en résulte
que l'épithélium qui y est
contenu entre pour sa part
dans la production du tissu
embryonnaire nouveau.

fr. 206

Varchow. 6. 3. Fran. 1.89 Infiltrations tuberculeuses

Conglomerat. tumeurs — Les tubercules gros comme une ~~noix~~ noisette, sont des tubercules conglomérés

Ces tubercules conglomérés sont constitués par des milliers de petits tubercules — dans les grands tubercules du cerveau, ce sont des centaines de mille qu'il faut compter

— aussi gros tubercules conglomérés des parois costales — masses de 4 à 5 pouces de long sur 1 à 2 pouces d'épaisseur.

Plaques conglomérées — Dans les plèvres et dans les séreuses.

p. 90 — Dans l'intimité des parenchymes où l'éruption tuberculeuse part d'un endroit pour envahir une étendue plus ou moins considérable, l'agglomération finit par revêtir le caractère de l'infiltration.

Je ne parle pas ici de l'infiltration tuberculeuse des poumons qui est plutôt une espèce d'hépatisation —

Mais je parle des enveloppes, des reins, des testicules, où l'infiltration qui devient plus tard uniforme était à l'origine un grand conglomérat tuberculeux et non pas quelque foyer inflammatoire.

Il faut donc faire abstraction de ces grandes agglomérations quand on veut étudier l'histoire de la granulation tuberculeuse isolée

car l'histoire generale des grandes agglomerations depend des processus qui evoluent dans les petites granulations

Or je le demande, qui fait mieux établi ; quelle opinion plus traditionnelle et plus médicale que celle qui proclame la tuberculose une affection foncièrement générale diathésique, qu'elle soit héréditaire ou acquise

J. G.

Chauffard répond sur Colin.

...

Cornel et Ramus.

fo. 201.

Tubercule en general.

Cornil & Ranvier. t. 2 p.

La granulation. Tuber. ou granul. grise.

- nodosité - de 1/10 de mm à 2 - 3 millim. ce dernier cas rare.
- relief, dure, opaque { transparente d'abord, puis opaque et jaunâtre au centre
- Entourée d'une zone rougeâtre vasculaire.
- qq. isolés, qq. confluents

- Elements - Rareté de la cellule.
  1°, Cellule aussi grande que dans le corps muqueux et plus noyaux
  2°- Cellule fusiformes.
  Cellules embryonnaires
  3°, Elem. pourvus qui y dominent. Petites cellules de 4 à 9 µ
  à noyau entouré d'une faible qté de protoplasma.
  Ce sont des Cell. embryonn. en voie d'atrophie.

Les Lymphatiques sont plus volumineux
que ces éléments embryonnaires des tubercules - Robin

Cornil et Ranvier. — arrangement des parties du tubercule

3ônes.

1°. Zône periphérique ou de prolifération. on voit sur de grosses cellules mères —
Zône qui plus étendue.

2° — Se centrale où les éléments se trouvent presque en s'atrophiant et finissent par tomber en détritus granuleux.

Tous les éléments émettent autour d'eux une subst. fondamentale glutineuse ou fibrillaire qui les agglomèrent et les tiennent fortement réunis les uns aux autres — en outre g. le centre de la nodos. devient fibrinoïde

— quand il y a des vaiss. au centre ils s'oblitèrent.
mais il peut y en avoir à la périphérie.

Cornil et Ranvier -

Developpement du tubercule

1: Le developpement se fait au milieu de tissu embryonnaire, de telle sorte que zone de proliferation - La forme est donc fonction d'inflammation du tissu conjonctif, ainsi, dans le foie il y a d'abord hepatite interstitielle dans l'os, ostéite.

2°, Mais dans le tissu conjonctif, ou cellules, analogues. mais peuvent naître aussi depuis des cells epitheliales.

— Dans le poumon. { 1° Tissu pulmonaire { interlobulaire / peribronchique / interalveolaire

mais 1° intérieur des plus. alveoles dont cloisons élastiques aussi. conservées alors de telle végété des parois. l'ovule y prend sa part.

p. 205 — 4

Nature : Cornil et Ranvier.

— C'est un genre de fibrome dans lequel les éléments cellul. disposés en nodules s'atrophient au centre du nodule.

placé tout près de la gomme dont ils ne diffèrent que par l'oblitér. des vaiss. dans le tubercule.

— Variétés — { soulitaires / ou agglomérés { une zône rouge les entoure. Vaisseaux { cette zône fait ressortir la granul. qui est opaque.

{ alors masses, volume d'un pois, noisette, noix.

— ~~une zone~~ { dans les granul. confluentes toutes se confondent par la partie centrale en une masse anémique — alors impossible de reconnaître les nodules isolés. bientôt toute la masse devient opaque,

p. 208

De pareils clots, devenus caséeux sont souvent rapportés, même par les observ. les plus en renom, à la pn. caséeuse lorsqu'ils siègent dans le poumon

— La même chose a lieu dans les os, les reins, les ...

il y existe encore p. 210.

Phases. (Cornil et Ranvier. 209)

5°

La transf. caséeuse est constante dans tout tubercule adulte
— une petite nodosité dont le centre est opaque et jaune, est un tubercule.
— est-ce l'adult. de Vaccinau?

De l'état caséeux on passe au ramollis! — Il en résulte un foyer
rempli de débris, qui peut s'ouvrir dans un canal muqueux.
Il en résulte une ulcération ou caverne

(2°. enkystement, et transf. calcaire.

— Structure. — Il est certain par l'analyse clinique que les granul. peuvent rester petites, se fusionner que des granul. intraparenchymateuses se joignent confusément au sein des masses indurées résultant d'une pn. interstitielle.

aussi chez les vieillards nodosités dures, remontant à un grand nombre d'années bien que un peu dégénérées à leur centre.

— Les tub. agglomérés ainsi peuvent rester longtemps inertes. mais la généralisation est toujours à craindre. On ignore quant à présent quel est le mode de généralisation.

Tuberculosis

In general,

Die Lichtenwalde.

von Billroth

Kellen und Billroth.

Handbuch.

1ter Bd. 2te Aufl.

1ter Lief. 2 Lief.

Combination von Krebs und
Tuberculose.

von Friedreich,

Virchow's Archiv. Bd. 465.
36. Bd. 1866.

permettez aussi à ~~l'école~~ évoquer ici comme de
plus proche une ombre pleine de
Majesté - Rendre hommage au
Clinicien incomparable, à l'anatomo
pathologiste Illustre, à l'homme
de la médecine Française, gloire
au grand Laennec !

Tuberculeux. Lymphatique

Tubercule. 3 zones (Tubercule adipeux)

Grancher — cellules embryonnaires { 1, 2,

Périphérie — { Subst. fondamentale { 1° Cellules rares se touchant
2° séparés en groupes linéaires par subst. brillante, réfringente, amorphe ou fibrillaire

Si alcool — carmin — cell. se colorent
ac. acétique — subst. se gonfle, noyaux apparaissent plus nettement — donc ce sont cellul. embryonnaires + subst. conjonct. en voie d'organisation

— soude — cell. se détruisent et fibres élastiques

Centre — en dégénérescence
— au voisinage du centre, dots orangés — au voisinage de la masse de génération { ce sont vaiss. oblitérés Cornil et Ranvier
{ ce sont des alvéoles (Grancher

Au centre même par soude — fibres élastiques dessinant alvéoles, squelette du tubercule — donc s'établit dans tissu du poumon
donc cell. alvéol. st. englobées

— Point de départ — vaisseau, bronches, paroi alvéolaire

— Donc granulation —

Bizzozero — les plus petits sont composés d'un certain nombre de follicules.
les follicules ont environ 0,4 mm —

granulie

Formes cliniques.

1° Adhérence lente = 2° Adhésite galopante - 3° Adhésion aiguë
ou Tuberculeuse. ou Tuberculeuse ou Caséeuse. granuleuse.

1°. Adhésite lente. durée 1 ou 2 ans. — autopsie.
2. Adhésion caséeuse. 2, 3 mois, 6 mois.
4. Adhésion aiguë =

Granules

Granulations tuberculeuses — Virchow

— Petite tumeur et la véritable tubercule est organisé [illegible], bien qu'il ne contienne pas de vaisseaux ; il est organisé c'est-à-dire il a une structure cellulaire — cellules.
— entre les cellules, il y a de petits réseaux de fibrilles de tissu conjonctif.
— au centre dégénérescence caséeuse — cette zone centrale est entourée d'une zone de cellules bien vivantes et tassées.
— une 3e zone de cellules plus grandes.

En parallèle au tubercule tumeur. — Le tubercule infiltré.

Granules. granules. — c'est dans la zone [illegible] et péri-lobulaire, dans le tissu conjonctif et le [illegible], que se développent les 1res altérations.
— c'est là que se développent les cellules lymphoïdes, ou cellules [illegible].

Si autour d'un vaisseau } 1° lésion du vaisseau — endartérite — endartérite — [illegible] rayonnante

Si autour d'une bronche } — altération de l'épithélium de la paroi — [illegible].

0,50.

Rabuis.

~~Globulins~~

Leucocytes — 0,008 } 0,012
0,004 }

Leucocytes
globulins à noyau libre — 0,004
0,003
0,005

Cytoblastim à noyau libre. 0,005 à 0,006. —

Rud Fleisch

L. Rindfleisch.

Medicinische Gesellschaft.
Berliner klin. Wochenschr. No. 6 et No. 7

Ueber Tuberkulose entzündung.

Il existe une inflammation tuberculeuse - c'est une inflammation spécifique -

Il y a une forme d'inflammation spécifique d'où un des caractères est la caséification - Cette inflammation est le produit d'une diathèse particulière -

La tuberculose miliaire a été à tort considérée comme la caractéristique de la tuberculose ; il n'est qu'un des modes de l'inflammation tuberculeuse

Les formes diverses de l'infl.on tuberculeuse, locale.

1° affection primaire de divers organes Membranes ou parenchymateux ayant le caractère de la phthisie ou de l'ulcération.

2° Des affections secondaires, ayant le caractère d'infection de voisinage - ce sont les tubercules initiaux des lymphatiques et du tissu cellulaire, et les tumeurs tuberculeuses des glandes.

3° affect. tertiaires. qui représentent une infection générale de l'organisme - Tuberculose miliaire des divers organes avec dialyse pneumonique autour des petits vaisseaux.

La 2e affection secondaire s'exprime souvent en pratique par la coexistence de l'affection tertiaire. (Tuberculose vasculaire) se portant par une multiplication des cours d'inflammation simples dans le foyer de l'inflammation tuberculeuse.

Tubercule miliaire

Ponfick.

Ueber Entstehung und
Verbreitungsweg der
Acuten Miliartuberculose

Etat du Canal thoracique.
Il en est tout autrement dans les cas
où il y a tuberculose generalisée

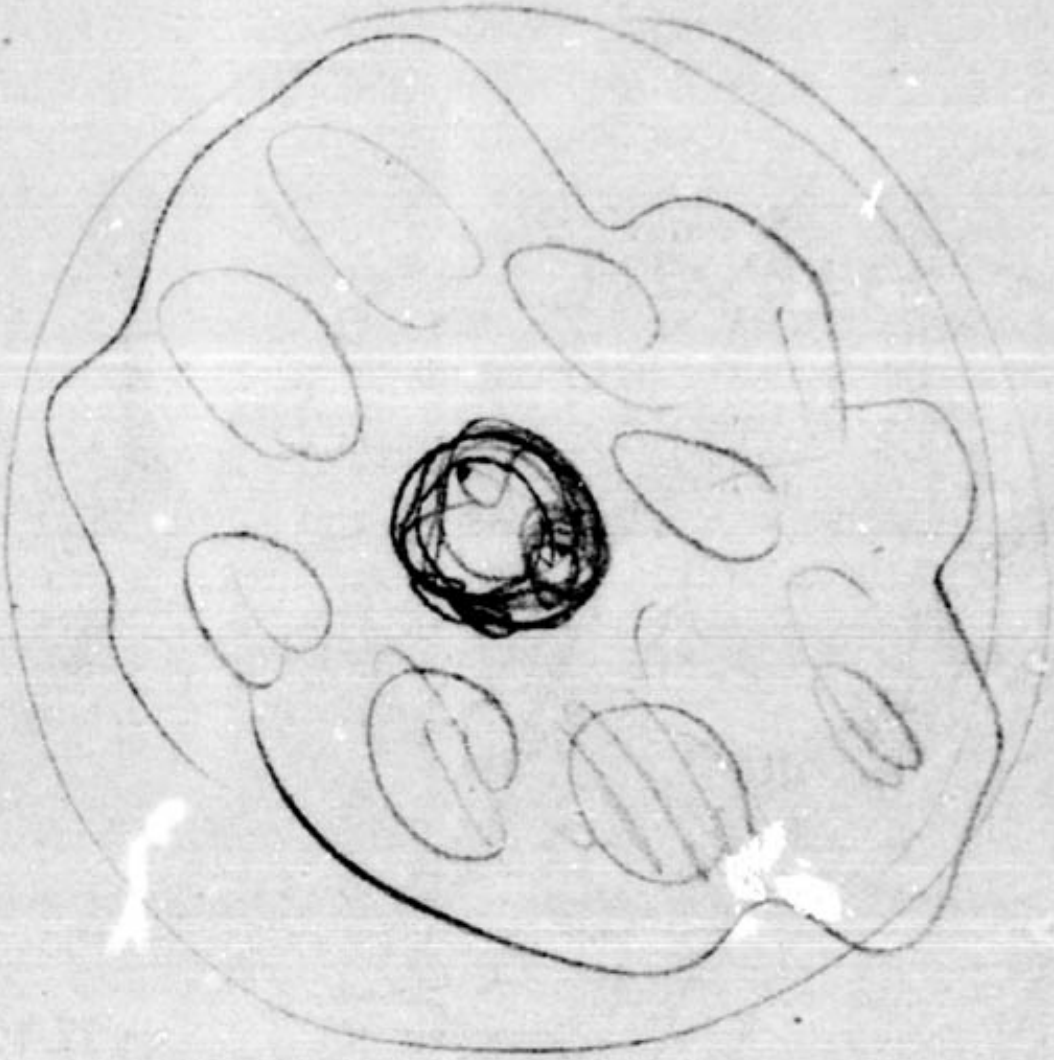
Im Berliner. klin.
Woch.
v. 6/3.
12. 9. 1889

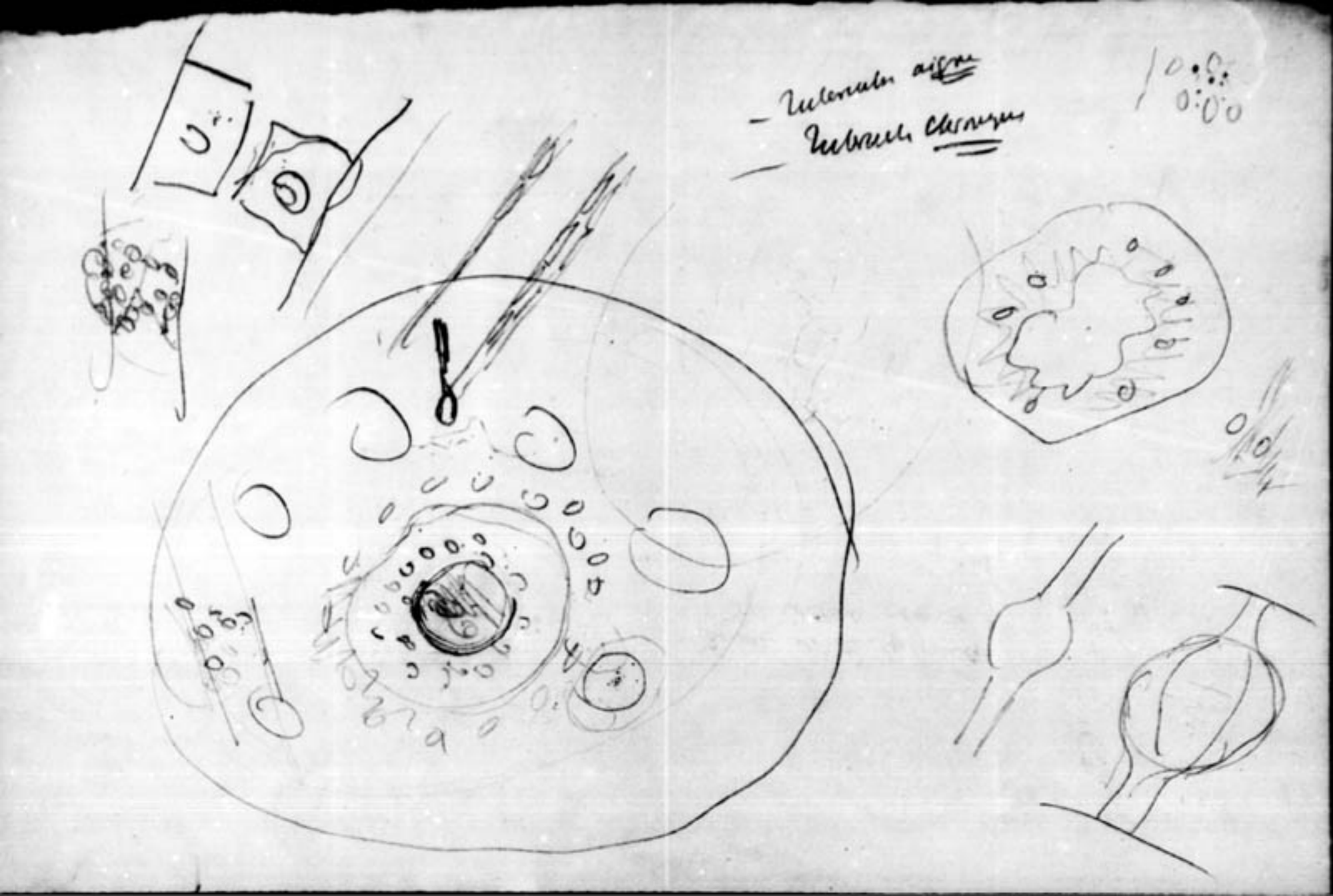
Miliar Tuberculose und
Emphysem. — Burkart.

B. 27. t. 12.
Deutsch. Archiv.

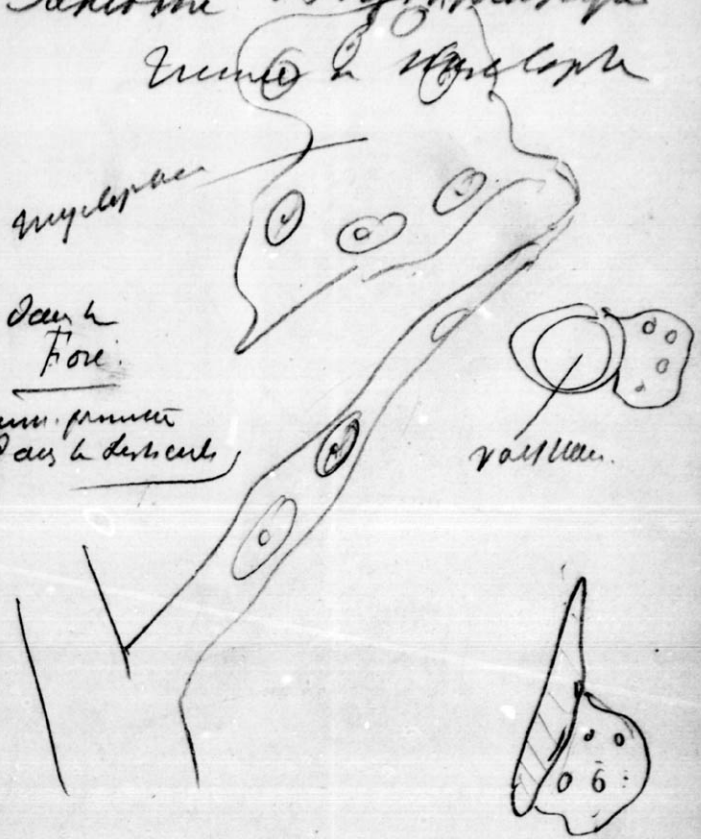
Malassez

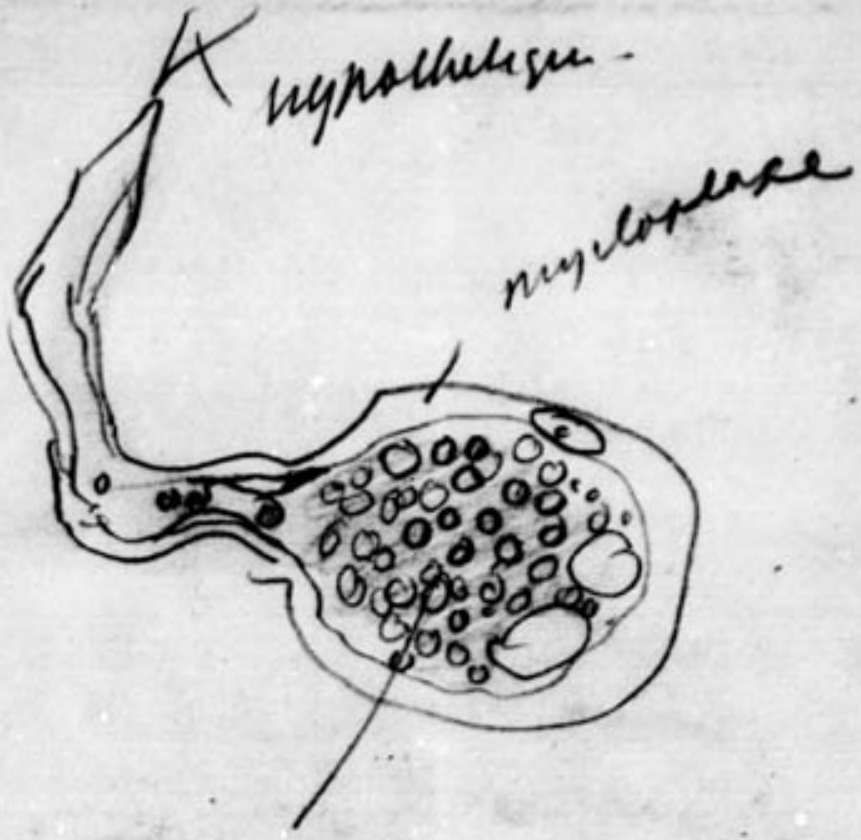
Tubercule du Cysticerque
et Cellules géantes.



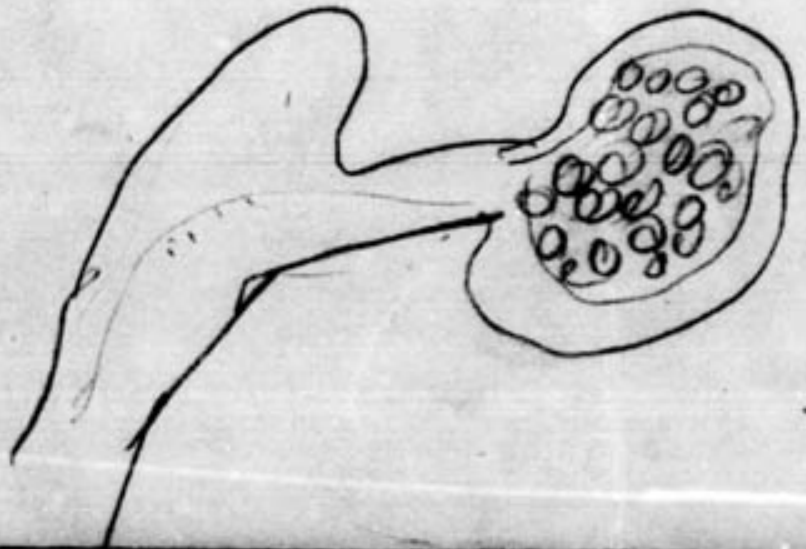

- tubercules aigus
- tubercules chroniques

Sarcome Angioplastique
Tumeur de Kaposi





Globule du Sang.



Les Tumeurs, ont presque toutes pour point de départ des cellules embryonnaires.

Leur marche est d'autant plus rapide leur vitalité d'autant plus grande, que l'élément globulaire, (cellulaire) domine davantage dans leur texture.

Trés de Küss. par Lancereaux.

Gaz. hebdomad. n° 43. 1872

25. 8bre p. 691

Kleb und Valentin

Geschichte der Tuberculose. —

origin Lymphatique
de Tuberculose.

Virchow's Archiv.

v. 242. 1808 44. vol.

4. poly. 4 tabs

Tubercule dans les alvéoles. — point de départ épithélial.

D'après M. Küss le tubercule pulmonaire n'est pas dû à la prolifération des cellules plasmatiques, mais à celle de l'épithélium alvéolaire — un amas [illegible] d'un épithélium très minces — l'atrophie de l'épithélium et celle des cloisons constituent l'emphysème.

L'épithélium pulmonaire est le point de départ du tubercule.

Thèse de Küss. — Laveran.
gaz. Hebdom. n° 43. 1872.
25. 8bre 691

Schüppel

S. 4.

der Tuberkel nicht in den Lymphgängen, sondern in den Follikulargebilden der Drüse sich entwickelt, und daß die grossen vielkernigen Mutterzellen welche in den Lymphsinus der Rinde und den cavernösen Gängen des Markes liegen, mit der Tuberkulose der Lymphdrüsen als solcher gar nichts zu schaffen haben, sondern einem Reizungs Zustande angehören, welchen Einige als Katarrh der Lymphdrüsen bezeichnet haben.

S. 16

Der Tuberkel entwickelt sich nur um gefäßhaltig. Gewebe; seine Beziehungen zu den Blutgefäßen sind mehrfach, wenn auch in verschiedenem Sinne erörtert werden.

In Zukunft wird vorzugsweise darauf zu achten sein, ob die Riesenzellen, deren Vorhandensein in den Lymphdrüsen immer den Beginn der Tuberkelbildung anzeigt, mit den Blutgefäßen in irgend welcher näheren Beziehung stehen -

Vergl. Rumpler. Das Verhalten der Tuberkel. Virch. arch. 24 Bd S. 571. —

Schüppel. - Zur Histogenese der Lebertuberkeln. Arch. der Heilk. IX. 1868. S. 561

contribution anatomique
du tubercule

Discipline poétique

Le Tubercule (Chauffard 6. 20. discussion sur le rapport de Colin

Qu'est-ce en effet, que la granulation tuberculeuse; Est-ce une formation pathologique d'une organisation avancée, un élément histologique d'une structure et d'une forme nette et distincte, d'un caractère propre et accusé? Loin de là. La néoplasie tuberculeuse est une néoplasie pauvre, qui ne se développe pas, dont l'organisation demeure misérable, dont la vie est courte, qui s'infiltre promptement de granulations graisseuses.

Cellules Tuberculeuses

•

Schüppel: Tuberkelzellen
die Kinder der Riesenzellen
— etwas der Epithelartigen
Zellen. —

Voir. Lymphdrüsen Tuberculose

V. 95.

Cellules Epithelioïdes, dans le
Lupus on serait des cellules
Epitheliales. Lange J. Rh.
med. Jahrbuch. 1875.

Wagner. manuel. p. 479.

— c'est un Néoplasme non vasculaire, nodulaire
— composé { de cellules analogues ± grosses. } toutes à la fois dans un
{ de qq. Riesenzellen } tissu réticulé.
— constamment ramollissement.

A
1° Tubercules { circonscrits – nodulaires — granulation — miliaire.
B { les conglomérats – qq. cerveau, œil { tubercules dans le cerveau le
testicule, les séreuses.

C Infiltrat. tuberculeuse — dans les membranes muqueuses. (voir nouveaux génito-
à la périphérie par de Castale (?) par les tubercules ; il y en par pas notable.

— on distingue les tubercules frais – ou gris
et les tub. – jaunes.

. Éléments constitutifs. du tub. frais. { Beaucoup prétendent qu'il y a toujours un protoplasme.
a Noyaux libres. ovalaires. 0,005 0,008. transparents
b cellules. analogues aux leucocytes mais plus grosses ou plus petites,
c. Grandes cellules rondes, rares, ovales ou étoilées, avec 10 ou 100 noyaux
— disposés ± régulièrement, dans une substance fondamentale ±
fibroïde, reste de l'ancien tissu.

Dans la majorité des cas, et dans les deux organes, que le tubercule soit infiltré ou nodulaire ou circonscrit, il présente une structure analogue au tissu réticulé ou cytogène — on dit alors tubercule réticulé ou cytogène —
lymphadénome tuberculeux —
un tel tuber. est composé de nodules ou follicules (de 0,35 à 0,15 diam.)
— chaque follicule = Reticulum dans les mailles duquel élém. cellulaires.
— Le Reticulum répond à celui de la subst. conjonctive réticulée normale
mais il est plus large — il consiste en cellules serrées ± distinctes
— le follicule est non vasculaire —
— les éléments cellulaires sont ceux décrits.
— au centre du follicule il y a constamment une ou plusieurs
Riesenzellen. – leur diam. est de 0,04 μ. – rondes ou
longues, granuleuses, tendues, avec 20, 50, 100 noyaux.
— En général un tubercule miliaire visible contient de 5 à + follicules.

Le tubercule fibreux paraît être une forme plus développée du tuber. ordinaire.

au centre des tub. anciens et souvent des nouveaux

— le follicule est non vasculaire
— Les Elements contenus dans ces deux dents.
— au centre du follicule il y a constamment une ou plusieurs
Riesenzellen — leur diam. est de 0,04 μ. — rondes ou
longues; granuleuses, arrondies, avec 20, 50, 100 noyaux.
— En général un tubercule miliaire visible contient de 5 à + follicules.

Le Tubercule fibreux paraît être une forme plus développée du Tuber. ordinaire.

Atrophie Nécrose. commence au centre du Tub. avance et s'arrête du nouveau
— Elle s'empare des cellules et des pellicules.
— Les cellules géantes, plus rond.

— ramollissement — résulte d'une Met. chimique inconnue.

Développement. — tissu conjonctif, vaisseaux, lymphatiques. — de l'endothel probable.

accroissement.
— extension. —
— tissu ambiant — Dans ... comprimé.

Virchow. p. 77 t. 2 - Trad. — Caract. des tubercules

Le caract. anatomiq. particulier de la tuberculose est dans le ~~son petite~~ développement pour spécifique du produit.

C'est la plus petite forme connue de tumeurs.

on dit tubercule miliaire — mais le grain de millet est plus gros — le volume est celui des plus petits follicules lymphatiques — analogie avec les corpuscules de Malpighi de la rate.

— C'est un produit organisé

— Pour caractériser le tubercule il faut s'entendre sur le stade qui doit servir de type dans la description.

Il prend la période de développement et de maturité.

— et prend le tub. des séreuses.

— or il est organisé - bien que non vasculaire.

La petite nodosité consiste en une prolifération provenant du tissu conjonctif —

La jeune prolifération au début ressemble à un produit de tissu de granulation.

La cellule est une cellule, avec un noyau. ressemblant aux éléments des ganglions lymphatiques — cellul ronde plus petite ou plus grande de 2 ou 3 fois que les glob. incolores du sang.

Les cellules les plus grandes renferment 25 de 30 12 noyaux et même davantage;

— Entre ces cellules se trouvent de petits réseaux de fibrilles de tissu conjonctif et quelquefois aussi des vaisseaux qui appartiennent à la partie ici disparue.

— Ceci est le tubercule cellulaire

— mais il y a le tubercule fibreux — p. 85.

+ Tuber. agglomérés . p. 89