of the disorder, are such as to show that it is the result of local disturbance, not general deterioration.

And it has been further shown, both by Dr. Galabin and by Dr. Sibson, that simple hypertrophy of the heart, which may probably be taken without error as an index of the cardio-vascular change, may result from the incapacitation of one or both kidneys by calculous disease. I can see no escape from the conclusion that disease, at first local to the kidneys, may be attended by, or, in other words, may produce, the vascular and cardiac thickening. And, as to the *modus operandi*, none other can be suggested than functional disturbance of the gland.

To take note in passing of a prominent clinical landmark which opens to our view, it may be accepted as a general truth, that hypertrophy of the heart in connection with albuminuria is a sign that the kidneys, however affected originally, have become the subjects of fibrotic and irrevocable disease. The association is, no doubt, partly one of time. When the disorder has lasted long enough to thicken the ventricle, it will almost invariably have extended to the interstices of the gland.

To speculate on the essential nature of the cardio-vascular change, it has been shown that, though differing in degree, it is similar in kind, whether the renal affection be of inflammatory origin or have begun, however obscurely, as granular degeneration. The change is compounded of muscular thickening, arterial and ventricular, which deserves in both situations to be called hypertrophy, notwithstanding its admixture in the arteries with degenerative changes, and, as coincident with or successive upon it, an exaggeration of the outer or fibroid sheath which is as truly as the muscular coat hypertrophied. These structural alterations are concurrent during life with increased tension or tightness of the arterial system. Where there is no increase of tension, to anticipate a fact with regard to the lardaceous disorder, there is no arterial or cardiac thickening. When both ensue, and the time of their accession can be observed, as happens when they succeed upon an inflammatory attack, the tension appears early, the hypertrophy late, in the disease. Thus, it is impossible to attribute the tension to change of structure; but, on the other hand, the two may be the consecutive results of a common cause, or the change of structure may be directly due to the increased tension. Whether we seek the cause of the cardiac hypertrophy or of the arterial tension, we can find it only in obstruction. After what has been recently brought before the College, I need not recur to the evidences that the increased tension of Bright's disease depends upon retardation of the arterial outflow. Bright, with a sagacity which served him in the place of microscope and sphygmograph, was led by the cardiac hypertrophy to infer the existence of this condition, and to attribute it to a difficulty with which the impure blood penetrated the capillary vessels. And at present the only question which calls for consideration is, whether we are to place the difficulty with Bright in the capillaries, or with Johnson in the arterioles. In the view of the later pathologist, one to whose observations, however we interpret them, a large importance must be attached, the circulating system is the seat of divided councils. The arteries resent the contact of the impure blood, seek to shut it off from the tissues by an action which has been likened to that of a stopcock, and by their continued efforts become hypertrophied. The heart at the other end becomes hypertrophied by its endeavours to overcome the resistance thus occasioned.

If we regard, however, the ventricle and the arteries, as the facts of comparative anatomy enable us to do, as but the subdivisions of one contractile vessel for the propulsion of the blood, which act not antagonistically, but in unison, an arterial effort succeeding upon and carrying on that of the heart, it becomes difficult to conceive a perversion of function such as this theory supposes. We may fall back upon the elder hypothesis, locate the hindrance in the capillaries, and assign it to a loss of fluency in the unpurified blood, a want of consent between it and the tissues, or in some other mode to a lessened facility of capillary transmission.

In this hindrance, whether in arteriole or capillary, in the smaller vessels or the smallest—and, perhaps, it matters practically little in which we place it—it is easy to see the explanation of the complicated change. We may, as of old, attribute the ventricular hypertrophy to the difficulty with which the heart empties itself; and with as obvious reason extend the same explanation to the hypertrophy of the arteries. The fibroid thickening, no less real than the muscular, may feasibly be assigned to the increased pressure of the blood against the arterial wall and the consequently increased soakage into it. The arterial coats are normally nourished by this process, and it is consistent with all we know of the effects of congestion elsewhere to attribute fibroid hyperplasia to its exaggeration. It is possible that the change in the muscular coat may be somewhat helped by this process, more especially in imparting

a want of distinctness or appearance of fusion to its elements. As touching the degeneration early found in the nuclei, later about the inner aspect of the muscular coat, and possibly at last universally, this may, perhaps, be fairly attributed to the irritative effect of the unpurified blood. An irritant in the blood, whether excremental or alcoholic, will cause atheroma, and may be credited with the allied result in fatty change of nuclei and fibre.

But, thus to trace the cardio-vascular change directly to renal incompetence, does not exclude the possibility that it may have other modes of origin. Cases present themselves, and by no means unfrequently, in which the lesion of vessel is marked by cardiac hypertrophy, retinal hæmorrhage, and hardness of pulse, and with which, nevertheless, the urine is either free from albumen or contains but a scarcely appreciable or intermittent amount. I think it is not to be doubted that in such the disease is less often absent than hidden, not declared by external symptoms, though effective in its internal results. Albuminuria is no such constant sign of the granular kidney as of tubal disease. Even when the gland is hopelessly crippled by interstitial growth and death by uræmia is impending, the urine, whatever be its faults of deficiency, may, if not critically examined, pass as free from albumen. The vascular mischief, therefore, is often associated with latent renal disease. Whether it ever occurs independently of renal disease, is as yet *sub judice*.

THE GOULSTONIAN LECTURES ON THE NATURAL HISTORY OF PULMONARY CONSUMPTION.

Delivered at the Royal College of Physicians, London.

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LECTURE II.

MR. PRESIDENT, FELLOWS, AND GENTLEMEN,—In my first lecture, I attempted to show that certain cases of disease included under pulmonary consumption by writers living in different countries and at different periods of time—all of them workers of the most trustworthy authority—ought not to be classed under that affection. My reasons for taking up that position were, that in none of them, or at any rate in the most typical cases, could death be said to occur from disease of the lungs; that the series of clinical symptoms was essentially alike in all, and absolutely characteristic of a disease to which the term “tuberculosis”, if it be preserved at all, may be given, but given only as a name for a certain and well defined group of symptoms, and not in relation to any particular growth or product. I stated reasons why I am prepared to hold, with Buhl and others, that this disease is one of infection, never causing independently any peculiar destruction of lung-tissue; and I think I demonstrated that, excepting in cases where the disease supervenes, as sometimes it does, on some former destructive affection of the lungs, the most marked characteristics of pulmonary consumption, as defined by Aretæus, by Sydenham, by Morton, were absent. Almost every writer without exception tell us that there was no emaciation; that the lungs, beyond being more red than normal, and surcharged with innumerable minute granulations, were sound. Bayle (*op. cit.*, p. 130; Barrow's translation, Liverpool, 1815, p. 143), in contradiction of his own definition of phthisis, exclaims in reference to his second case, that of a postilion aged 24, who died from apnoea twenty-two days after being attacked with a dry cough, his lungs being found crammed with miliary granulations, “In the beginning of November, the cough not having yet commenced, where is the physician who, on examining this young man, who seemed to enjoy the finest health, and whose chest was perfectly well formed, would have suspected a consumption?” And it was from cases of this kind, as compared with others of which I shall speak to-day, that I hope to show that this is never the commencement of the ordinary phthisis of this country. Therefore it was that I insisted that the terms “tubercle” and “tubercular”, vague in their primary use, have become vaguer in their secondary one; and in any degree I have been able to assist in encouraging some strict definition of terms so commonly used, I shall not have wasted time in a fruitless strife of words.

I pass on to consider the ordinary form or forms of consumption, keeping before me the definition of Morton, to which may be added

the sanction of Bayle: "The lung is the only organ which constantly undergoes organic change in pulmonary phthisis"; and that of Portal (*Observations sur la Nature et sur le Traitement de la Phthisie Pulmonaire*, Paris, 1792, see p. 539), who, in his chapter on the examination of bodies dead of this disease, says, "The lungs always undergo change".

One of the great facts which Addison set himself to demonstrate was, that pneumonia (*Guy's Hospital Reports*, ser. ii, vol. iii, p. 3)—I quote his own words—"leaves behind it divers forms of induration of the pulmonary tissue, all of which have been somewhat indiscriminately, but I believe erroneously, regarded as modifications of tubercle or of tubercular infiltration. Such indurations of the pulmonary tissue, altogether independent of tubercle, are, I am more and more persuaded, of very common occurrence; and moreover venture to predict that the time is not far distant when it will be generally acknowledged that they have not by any means commanded the attention they deserve." I am content to confess my belief in Addison's prediction, though its fulfilment is hardly yet come. But since the time of Morton with sixteen varieties of phthisis, Sauvages with twenty, Portal with forty, Baumes with three, Bayle himself with six, and yet doubting whether there may not be more, the tendency has certainly been to arrive at some simpler and truer statement of the phthisical process; even though Buhl, perhaps the clearest writer of to-day on the subject, must needs divide and subdivide varieties. The division I take to-day for my own convenience is to some extent similar to that adopted by Dr. Peacock in the paper to which I have already referred, though I must still insist upon distinguishing between the effect of intra-alveolar and extra-alveolar products; and so far, in its larger characters, my division agrees with that given by Buhl. At any rate, I cannot but feel that the clinical corresponds here with the pathological grouping.

All forms of phthisis may, I think, be classed according as to (a) whether dead or dying tissue-elements are found choking up the alveoli and bronchi, and leading to further destruction of the respiratory organ; (b) whether changes occur in the walls of the vesicles and bronchi, robbing that organ of its function in a different way; or (c) whether the interstitial tissue undergoes metamorphosis, interfering in yet another fashion with that same necessary function—on the imperfect and abnormal performance of that function depending all the symptoms of innutrition and fever which go to make up pulmonary consumption. To the first group belongs that form of the disease to which Buhl has given, I think correctly, the name of "desquamative pneumonic phthisis"; to the second is due that form next common in frequency, caused by long-standing bronchitis and emphysema; and to the third, those fibroid changes resulting in the same true symptoms of consumption, differing somewhat clinically and pathologically, as they are caused by alcoholism, syphilis, or anthracosis. All these forms of consumption fall under the definition of Morton: all owe their origin to primary disease of the lungs; all are accompanied by symptoms due to that lung-disease; all alike bring in their train that general wasting of the bodily structure and strength which is included in the very term "phthisis"—"consumption". Upon the first and second forms, if not upon the third, a further process, resembling that of "acute tuberculosis", may and does frequently supervene, constituting Waldenburg's "phthisis combinata"; but the series of symptoms in these cases is in strong contrast to those alluded to in my first lecture. I need scarcely state, after what I have already put forward, that my own work, clinical and pathological, leads me to support the proposition of Addison (*Guy's Hospital Reports*, ser. ii, vol. iii, p. 29), "that inflammation constitutes the great instrument of destruction in every form of phthisis".

I proceed to speak of the first form of morbid change, which, leading to destruction and death of the breathing surface, originates, as I believe, in catarrhal inflammation of the epithelium lining the alveoli and smaller bronchi, and is the form under which the common pulmonary consumption of this country most frequently occurs. This view I do not intend to support by reference to the numerous authorities who from the time of Broussais downwards have upheld the inflammatory origin of phthisis. I propose, on the other hand, to show, from writers who uphold its tubercular origin, how far their descriptions are consistent, as they profess them to be, with the supposed development of an extra-alveolar miliary nodule into an intra-alveolar and interstitial exudation or infiltration. I shall, however, press into my service especially such plates as exist, by whomsoever prepared, in illustration of what certain writers mean by certain *post mortem* appearances, the correctness or the reverse of such appearances being open to the judgment of those who inspect them. The agreement of these drawings with what each of us can see for ourselves in the *post mortem* room being allowed, the general experience of all is conclusive that a draw-

ing, however inartistic, represents those facts which the draughtsman most certainly appreciates for himself,

"Quæ sunt oculis subjecta fidelibus, et quæ Ipse sibi tradit spectator."—HOR., *A. P.*, 181.

Of the clinical symptoms of the ordinary phthisis of this country it is not for me to speak. The sequence which Morton sketched—the general wasting of the body, due in the very first instance to the local mischief in the lungs, and accompanied from the very commencement by cough, dyspnoea, and other signs pointing to those organs, joined with symptoms of pyrexia, at first of a low and hectic type, but increasing in intensity as it becomes also more frequently remittent—such is the every-day picture of that phthisis which, as Hippocrates noticed, occurs most frequently between the ages of eighteen and twenty-five, and of which Sydenham declares, "Atque hæc est præcipua Tabis species" (*Proc. Integri*, cap. lx, [1]). Modified by age, at any rate, in its rapidity of march, here swiftly striking down the young and growing, there clinging to the full perfected man, till, with a grim satire on the "survival of the fittest", it robs him of the later years of manhood—it preserves, with this one exception of rapidity, the same marked outlines, to recall which further to your minds would savour of impertinence on my part. It is on the interpretation of the morbid appearances found that differences of opinion alone arise. Death in this form of phthisis is brought about, as Aufrecht (*Die chronische Bronchopneumonie [Lungenschwindsucht] und die Granulie [Tuberculose]*, Magdeburg, 1873) has already stated, either, though this is rare in adult life, by the supervision upon pulmonary disease of longer or shorter standing of what, with Empis, he calls "granulia"—that is to say, of "acute general tuberculosis"; secondly, as an acute galloping consumption; or, thirdly, as the termination of a long chronic process.

The appearances found in the second and third class of cases seem to me to be the most unmixed, though more difficult to unravel in the type of galloping consumption, and are, as far as I can see, the probable key to the commencement of this affection. The signs during life are, of rapid consumption, those of consolidation at the apex generally of both lungs, followed very rapidly by signs of breaking down of the lung-tissue, both consolidation and destruction creeping swiftly downwards to the base. The signs in chronic consumption are a very slow procession of the same features over one or both lungs. On *post mortem* examination, these organs, in addition to the cavities of larger or smaller size present, and portions of tissue in a condition easily broken down by the finger, exhibit here and there those grape-like masses which are most marked in the chronic form of the disease. To this arrangement in the latter form Laennec evidently refers when, in a description of one of his plates, he recognises it as a collection of "crude tubercles", at the same time comparing it to the club on playing-cards, drawn even at this day more correctly on the cards of Paris than on those of London, as a stalked trefoil. (*Op. cit.*, plate i, fig. 7.) This same racemose arrangement is seen in Carswell's plate (*Pathological Anatomy*, Lond., 1838, "Tubercle", plate i, figs. 1 and 3), which he describes as a delineation of tubercle, a delineation which Sir Thomas Watson praises, and adopts as a representation of his own views. Bock, in his *Atlas of Pathological Anatomy*, published at Leipzig in 1855, gives in Plate xxiii a very good illustration of a most common *post mortem* appearance—cavities in the apex, and in the base clusters of round bodies. With him, again, these latter are "tubercles". Colberg (*Beiträge zur normalen und pathologischen Anatomie der Lungen*, *Deut. Arch. für Klin. Med.*, 1867, vol. ii, p. 486, taf. vii, fig. 7) in 1867 gives the naked-eye appearances of chronic catarrhal pneumonia (phthisis pulmonalis), in which the same clusters are said to be due to the cheesy degeneration of the contents of the alveoli; and Thaon (*op. cit.*, plate ii, fig. 3) in 1873 gives a plate resembling Colberg's in everything but colour, the description of which reads as follows: "Fibrous granulations, or the granulations of Bayle arranged in clusters: isolated clusters: confluent clusters: bronchial ramifications: normal pulmonary tissue." But, whether called by the various names of tubercle, chronic catarrhal pneumonia, bronchopneumonia, it must be conceded that all these plates have these points in common: they profess to represent the appearances found in the bodies of those who die of pulmonary consumption; they represent the naked-eye appearances of what we find to-day on the *post mortem* table in bodies dead with the same symptoms described in connection with these cases; they represent an intra-alveolar and intrabronchial product, with more or less surrounding inflammation and more or less surrounding induration.

To Addison's lithographs I have already referred. Bayle gives no plate of his own; but Barrow (*Researches on Pulmonary Phthisis*, from the French of G. L. Bayle, D.M.P., by William Barrow, M.D., Liverpool, 1815), who translated his work in 1815, gives two woodcuts of a lung containing ragged cavities of different sizes and bronchi cut transversely; the latter being, according to him, tubercles. Not only do

the woodcuts, but the very words of the late Dr. Hughes Bennett (*Clinical Lectures*, 1859, p. 711), point to the same intra-alveolar, intrabronchial production of what he names "tubercle". He asserts: "Numerous successful injections of pneumonic, tubercular, and cancerous lungs, in my possession, demonstrate that the exudation in all is poured out in the same manner, and occupies the same position in the pulmonary textures"; that is, as the preceding sentence tells us, fills the interior of the air-vesicles. The same growth of "tubercle" in the lung from the intra-alveolar epithelium was the view of Schroeder van der Kolk (Paget's *Surg. Path.*, ed. 1863, p. 815). The same, too, had been apparently (*ib.*, p. 814) Virchow's earlier view, though (*Cell-Path.*, p. 474), later, in his criticism of Reinhardt, he held the "tubercle to originate from connective tissue". Such, if I may be allowed to state my belief, is the view held by a large number in this room; viz., that the product is intra-alveolar.

This intra-alveolar origin of the pathological products of the commonest form of phthisis being for the moment allowed, the rapidity of proliferation of the epithelial cells, growing closely side by side, may easily be judged of by comparison with the rapid enlargement and proliferation of the cells of irritated articular cartilage, or with that of the cells of the inflamed cornea. But, with this rapid proliferation of cells, the portions of lung attacked begin to lose their function as breathing surfaces, and to take on the function of secreting glands. It is scarcely possible, I think, to overlook the likeness there is between, at any rate in their pathological aspects, the inflamed lung and the inflamed sebaceous follicle. The lung, in a state of chronic inflammation and of epithelial—or, if the word be better chosen, endothelial—growth, is very like a racemose gland, also in a state of chronic inflammation. In the description of one of his lithographs (*Guy's Hospital Reports*, ser. ii, vol. iii, p. 36, plate iv), which to my eye represents lobular products with recent pneumonia, or, in his own words, "a form of phthisis in which the disorganisation results partly from the softening of recent hepatisation, and partly from the disintegration of pneumonic induration more or less ancient", Addison draws what he conceives to be, "although rude and incorrect, some sort of analogy" between his "sthenic tubercles"—i. e., the result pictured—and facial acne. I confess that I fail to see either the rudeness or the incorrectness of the comparison. A lung-alveolus is not so unlike a sebaceous follicle that its contents, like those of the latter, proliferating more rapidly in a condition of inflammation, and, to quote Morton again, "having a difficult exit", lead to further inflammation. Both become plugged with their contents; the contents of both undergo the same cheesy (oily) change, and set up the same inflammation and death in the secreting wall itself and the immediately surrounding tissues. And, as increased cell-growth cannot but be influenced to a very large degree by the simplest mechanical processes—of which fact no histologist can doubt—we might easily expect that in the loose and spongy tissue of the lung, as compared with the dense structures outside sebaceous glands, the death of the parts engaged is at the same time more rapid and more wide-spreading.

And this, as it seems to me, is the process—the great fault of which is its simplicity—which really does occur in the very large majority of cases of ordinary phthisis. If we take a portion, which seems most recent, of one of those grape-like clusters to which we have referred, we shall find pretty well the same microscopical appearances which Professor Klein has drawn in this vertical section through the lung of a child which died of acute tuberculosis. Here the masses found agree with Buhl's description of desquamative pneumonia. The alveoli and infundibula are distended by abnormal material. The intra-alveolar capillaries, as the disease advances, are no longer permeable, seem to disappear; as does the epithelial lining of the alveoli, still visible in the regions of latest change. Here is no stubborn tissue opposing its own vitality to the invading change. The cell-proliferation in a few alveoli leads not only to its own death, to the death of the capillaries which first fed it, and to the death of the wall which supported those capillaries: by easy extension, it passes onward to more alveoli, more infundibula, till bronchioles and bronchi are caught in the devastation; or it may be that the bronchioles are the first parts affected, and the catarrhal proliferation in the alveoli due to an extension of the inflammation from them. The microscope only tells us this: that these grape-like masses consist of intrabronchial and intra-alveolar products, in a more or less advanced stage of cheesy degeneration.

It is scarcely needful in these days, I suppose, to say much with regard to that distinction laid down by Rokitsky (*op. cit.*, *Syd. Soc. Trans.*, iv, 106) and Skoda (*Differential Diagnose zwischen Pneumonie und Tuberkulose*, viii, 46, 1862), and followed by numerous writers and teachers, between pneumonia and what they called pulmonary "tubercle"; namely, that the former began, as a rule, at the base; the latter, at the apex. Skoda himself narrowed this question

very much by begging the larger premiss, and asserting that, pneumonia being an acute affection, and tuberculosis only seldom acute, healthy persons were attacked most often by apical pneumonia, while any acute apical infiltration in persons suffering from chronic "tuberculosis" was "tubercular"—i. e., phthisical—leading to destruction of lung-tissue. At any rate, from the time of Morgagni downwards to a paper published in 1873 by Schlesinger (*Zur Statistik der genuinen fibrösen Pneumonie*, Berlin, 1873)—the latest paper I think I have seen on the subject—there are numerous statistics to show that apical pneumonia is very far from being an uncommon thing. And there is a class of cases to which I think sufficient attention has not been paid, of which the following is an example. For being allowed to examine the case, and for some of the notes, I am indebted to my colleagues Dr. Broadbent and Dr. Cheadle. I may mention, in passing, that, judging probably from the sharply defined distinctions of books, the house-surgeon who first saw it took it for a case of phthisis: an opinion which in lapse of time, will probably, I think, be justified by the result. A married woman, aged 22, whose father was said to have died of consumption, was admitted into St. Mary's Hospital, having been perfectly well up to a week before, when she complained of pain under her right clavicle. The physical signs were impaired resonance with bronchial breathing at the right apex, and general bronchitis of the smaller tubes. The same night, she was delivered of a six-months birth. A month later, I examined her. There was no difference on percussion on either side; but, in addition to a general bronchitis which still existed, there was feeble inspiratory murmur at the right apex, as compared with the left.

I have notes of cases similar to this, seen in my out-patient practice—cases which all agree in the presence of a more or less general capillary bronchitis, with localised dulness at the apex of a lung. I have not been able myself as yet to follow out any one of these cases; but in a series of American lectures now being published there is one by Dr. Colwin Ellis on the frequency with which capillary bronchitis is complicated with changes in the air-vesicles, and on its relation to catarrhal pneumonia. In this paper, five cases of the kind I am speaking of are given, two with *post mortem* results. Rindfleisch (*Deut. Arch.*, vol. xiii, p. 43; see Ziemssen's *Cyclop.*), writing on "chronic tuberculosis of the lung", and intending by that name the ordinary form of phthisis we are now considering, supports this view in so far that he places the original seat of his "tubercle-granule" at the passage of the finest bronchi into the air-vesicles, into which its further infiltration proceeds. With the statements of Rindfleisch, Jürgensen, and Buhl, agrees the opinion of Dr. Hughes Bennett (*op. cit.*, p. 713), that in the phthisical process "the bronchi are necessarily involved; their terminal extremities are among the first structures affected"—those parts of the tubes which, says Andral (*Clin. Med.*, ed. cit., vol. iv, p. 3), "are on the point of forming the air-vesicles".

I have at this present moment under my care at the Victoria Park Hospital two cases in one family which link together in a very interesting way the form of phthisis I have thus slightly sketched and that other form which, exhibiting all the clinical symptoms common to the two classes, does not depend, I think—at any rate, originally—on intra-alveolar changes, but on changes in the walls of the alveoli and bronchi themselves, i. e., on chronic bronchitis and emphysema. Laennec had noticed the strong likeness between his "pulmonary or tubercular phthisis" and this "chronic mucous catarrh"—a common infirmity of old age. (*Op. cit.*, vol. ii, p. 76, 77.) He says: "There exists, in fact, the most perfect similarity between these two affections, whether it be in the sputa, the emaciation, or all the other symptoms." Of the two cases to which I referred, one is that of a lad aged 16, who presents all the signs of what I am in the habit of calling cheesy pneumonic or bronchopneumonic phthisis, with, the last time I saw him ten days ago, marked pyrexial symptoms. The other is that of his father, aged 54, who declares that up to last winter—at the very most, a year ago—he was perfectly well; while, as to his son, he asserts that, after an attack of scarlet fever, "he was pretty hearty till he caught a cold". The father's father had died at the age of thirty-five from "rupture of a blood-vessel". The two cases stand in strong contrast, yet in strong similarity; the younger dying in no long time from, it may be, the engendered phthisis, "quæ quidem leni catarrhi nomine à miseris istis ægris signari solet; donec tandem negligentia incurabilis evadat" (Morton, *op. cit.*, p. 334); the older surviving beyond the life of his son, to wait the close of a chronic and incurable phthisis, "quoniam omnes senes, qui aliquo acuto morbo non corripiuntur, hoc modo phthisicè languentes tandem ultimum claudunt diem" (*ib.*, p. 236). It is not true, as Morton seems to have thought, at any rate to some extent, that old age itself is a chronic and incurable phthisis of this kind; but it is true, as he laid down, that the "asthmatic phthisis" of which he writes is marked off from other forms of phthisis by the pre-

sence of wheezing and intense dyspnoea throughout the whole course of the disease. I should but waste the time of the College were I to enlarge further on this form of consumption. There is one fact to which I cannot but draw attention; and I think it is important, because it throws some light upon a theory which, for myself, I have utterly discarded: the relation between phthisis—consumption—as with Morton I have defined it, and the presence and development of so-called tubercle. I do not remember to have seen, though I have heard of, cases of old people dying of acute tuberculosis. But, even in those who die in the ordinary course of a chronic bronchitic phthisis, there may be seen scattered round old cavities, or in the neighbourhood of dilated tubes and patches of emphysema, more especially at the bases of the lungs, certain bodies, to the naked eye like miliary tubercles—like well-boiled rice-grains—but under the microscope vesicular lobular pneumonias. The evidence of other observers goes to show, what I have not yet been able to prove to myself, that, in addition to these alveolar proliferations, there are also perivascular products; and in so far I readily accept Dr. Klein's statement with regard to their occurrence in artificial tuberculosis, as probably true also here, that these semitransparent nodules, "due to catarrhal pneumonia, depend on the spreading of the perivascular cords" (*op. cit.*, ii, 60), and, to quote Rokitsky again, "are usually secondary processes" (*op. cit.*, iv, 83). But, beyond this, they have no special meaning; they are but the common accompaniments here, as always, of any affection whatsoever of the lungs. The font to which they owe their origin is probably, in these cases, some cheesy focus in some dilated tube: a mechanical process to which the next set of cases may lend at least a possibility.

[To be continued.]

DOUBLE POPLITEAL ANEURISM:

THE LEFT ANEURISM DEVELOPED AFTER HÆMORRHAGE CONSEQUENT ON LIGATURE OF THE RIGHT FEMORAL ARTERY: PULSATION RETURNING AFTER LIGATURE OF ARTERY, ARRESTED BY EXTENSION OF LIMB: RECOVERY: SUBSEQUENT ANEURISM OF SUPERIOR MESENTERIC ARTERY.*

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CHARLES DOLT, aged 37, a healthy labouring man, about 5 feet 10 inches, spare, muscular, and of dark complexion, who had suffered amputation in the left forearm sixteen years previously in consequence of an injury, was admitted into Addenbrooke's Hospital November 12th, 1873, with a tense very painful swelling in the right ham. The knee was at a right angle, and he could not straighten it. The swelling filled up the ham, and was so tender that he could scarcely endure it to be touched. There was no pulsation in it, and no *bruit*. I was, however, suspicious of aneurism, and directed him to keep in bed. There was oedema of the foot and leg. Next morning, pulsation, *bruit*, and the ordinary symptoms of aneurism, were marked. He was extremely restless, in much pain, without appetite, with flushed face and sordes on the teeth. The pulse was 80, full and bounding. No cardiac murmur, or evidence of disease in the heart or great vessels, was perceptible. He was formerly a runner on the Nottingham cricket-ground. Nine weeks before his admission, he walked twenty-six and forty-four miles on two consecutive days. On the following day, he was unable to walk from general stiffness. The other parts of his limbs and body recovered, but the right knee remained stiff and painful. He returned, however, to his usual avocation as helper with a steam thrashing-machine. A fortnight before admission, he noticed swelling of the ankle and an enlargement in the ham. A week later, he was running and jumping, and fell into a ditch. After this, the pain became much more severe, and he was unable to walk.

We tried pressure upon the femoral artery and flexure of the knee, but he could not bear either. Even digital pressure on the artery for a short time could not be tolerated. Accordingly, on November 20th, I tied the right femoral artery, in the usual situation, at the lower part of Scarpa's triangle. The operation was very easily done, and there was very little exposure or disturbance of the vessel. I applied a carbolised catgut ligature, tying it tightly, and cut off both ends. Carboloid oil was applied, and the edges of the wound approximated by means of sutures and plaster with carboloid dressing. The pulsation and pain in the ham ceased immediately, and never returned. He suffered, however, a great deal from pain in the foot and toes; and mor-

tification of the extremity of the pulp of the second toe took place, and subsequently the nails of the other toes separated, the separation being attended with some ulceration at the roots of the nails, particularly of that of the great toe. The wound did not proceed favourably: no healing, but a good deal of suppuration. It subsequently granulated, and appeared to be doing well, when on December 9th, at midnight, severe and nearly fatal bleeding occurred, deluging the patient. The house-surgeon was quickly in attendance, and applied pressure. When I reached the patient an hour afterwards, the bleeding had ceased, and did not return on the suspension of the pressure. I deemed it, nevertheless, best at once to reopen the wound and search for the bleeding vessel; and I tied, as I thought, both the proximal and the distal ends of the artery in the wound, using a hemp ligature. The bleeding appeared to have come from the upper end; and I supposed at the time that I had properly secured the vessel, though the sequence gave pretty conclusive evidence that I was mistaken in this. It was rather a trying proceeding in the weak state of the patient; and it was difficult to throw the candlelight, the patient lying in his bed, well into the depths of the wound. Next day, a distinct, though slight and limited swelling, was perceptible in the upper and inner part of the wound, which made me fear that all was not right. On December 16th, at 8.30 A.M., bleeding recurred, after the leakage of a few drops during the night. I again opened the wound, and exposed the bleeding upper end of the artery, in which there was a considerable aperture, and with some difficulty tied it, this time effectually. I now found that the swelling, just mentioned as near the wound, was caused by clots deep in the wound and in connection with the artery, which renders it pretty certain that I had not succeeded in tying the vessel in the night of the 9th. Both the ligatures which had then been applied were loose in the wound.

On the morning of the 18th—two days, that is, after the second hæmorrhage, and while he was in an extremely exhausted state from the loss of blood on the two occasions—the patient told me that the same disease had taken place in his left ham. He had felt something snap during the night, and had been in great pain ever since. True enough, there was a distinct aneurism, so tender that he could scarcely bear it to be touched; and he could not tolerate even digital pressure upon the femoral artery. The swelling increased, and the pain continued, or rather, after some intermission, returned with such severity that he willingly submitted to the ligature of the left femoral artery, which I did on Christmas-day. I applied on this occasion two hemp ligatures at a distance of about half an inch from each other, and divided the artery between them. The edges of the wound were simply approximated by sutures, without carboloid acid, and without any dressing or plaster. He was in an extremely feeble state, but was greatly relieved by the operation. The pulsation in the aneurism ceased. The relief, however, was but of short duration. On the day after the operation, slight pulsation was felt in the aneurism. This increased, and with it the pain, so that in four days his condition was as bad as before the operation, the pulsation being full and severe, as though the artery had never been tied. It was quite certain, however, that the vessel had been tied, and indeed divided. But we remarked that after the operation, in this instance, he did not suffer so much pain in this limb as he had experienced in the other limb after the operation upon the artery in it; and there was no indication of imperfect blood-supply in the distal part of this limb. We inferred, therefore, that the anastomosing currents in this limb were more free, and were sufficient to give new impulse to the aneurism. The case was perplexing, and the man's condition not encouraging the attempt at any further operative procedure. The limb was bent, and I bethought me of straightening it, and did so with some force, the proceeding being attended with extreme pain to the patient. I bandaged it in the straight position upon a splint; but he could not endure this, and in about an hour took off the splint. The pulsation, however, had ceased, and did not return, and he was again much relieved. In two or three days, the swelling became exquisitely tender, though not very painful. It increased in size, and there was some surrounding oedema. The patient was also delirious at night. I inferred that suppuration was taking place; and therefore, on the tenth day after the operation, I passed a full-sized trocar into the aneurismal tumour, and gave vent to some thick grumous semipurulent matter. A drainage-tube was inserted and left in the wound, and the cannula withdrawn. Great relief was given by this. The discharge through the tube was not great, ceased in a few days, and the tube was removed. The swellings gradually subsided in both hams, and the man left the hospital quite well.

The points worthy of attention in this case are, the occurrence, or, at any rate, the rapid increase, of the left aneurism during the time that the patient was lying in bed exhausted by hæmorrhage. Secondly, the

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