CLINICAL LECTURE ON THE VARIETIES OF PHTHISIS.

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THE subject of the classification of phthisis is not an easy matter, or I should not have devoted so much time to its discussion. A great deal has latterly been written upon it by earnest hard-working men, who were looking out for anatomical characteristics to guide them in separating one form of the disease from another. It became necessary for me to state their views, to show you what opinions were entertained, and whither certain doctrines must lead you if you accepted them. I was not criticising the works of the different authors who had advocated certain separate varieties, but was examining the grounds upon which they based the distinctions which they made, and was putting to you the same question I had often previously asked myself—*Cui bono?*

These varieties are various phases of one and the same complaint at different periods of its existence, or else they are distinct pathological entities, and are entitled to be called after the prevailing pathological change. Thus the same patient may at the commencement of his lung-malady have what you please to specify as an alveolar catarrhal phthisis, and then symptoms arise to make you call his illness phthisis *ab hæmoptoe*; and, after the lapse of a year or more, you will see him again, and name his complaint fibroid phthisis, or else phthisis with recurrent hæmoptysis. And then, I ask, of what clinical use has this kind of classification been to you? Has it increased your knowledge? Has it influenced your treatment? Perhaps it was difficult to tread on such delicate ground in a lecture, and not offend. I should have known that I must wound some author's prejudices.

As those of you who read the journals will have seen, some one has felt himself aggrieved by me; but this writer is in error, as he must perceive if he read through my lecture more carefully, when he states that my object was to discredit before my readers any attempt at a classification of phthisis upon a pathological basis. As to misunderstanding or misinterpreting Dr. Douglas Powell's views, how could I? He wrote a book to establish certain varieties of phthisis; and, since all his different subforms either pass or may pass one into the other, I told you, what I feel bound to repeat for your instruction, that this mode of classification is of little practical utility. Let me take this opportunity, however, of correcting one statement I made, and of which Dr. Powell has complained. I said that, in his variety of phthisis *ab hæmoptoe*, he did not specify the source of the blood. What I meant was, that he did not prove this. In his table he does particularise the blood-source to be the pulmonary capillaries, and in his text he brings what arguments by way of facts he possesses to determine this; but, if he be satisfied with these facts, I am not. There is no real evidence where the blood came from. The woman did not die then, or there is no *post mortem* examination recorded; and Dr. Powell must be a feeble reasoner than I give him credit for being, if he think he has proved the blood-source or the phthisis *ab hæmoptoe* at all, to his own or anybody's satisfaction.

I am the more warm on this point, because, as I have often told you, I do not believe in a form of consumption produced by hæmoptysis at all—whether the regurgitation, or the gravitation, or the intraction by respiration into the air-cells, of blood elsewhere effused. The facts are all insufficient, and too inconclusive to favour my acceptance of them; and it is a similar caution that I felt it my duty, as your teacher, to inculcate on you.

But let me pass on. There has been too long an interval between my lectures already.

I gave you to understand that there were three different kinds—species, if you please—of phthisis, which I had found it desirable to separate from each other, because they were or seemed, clinically as well as pathologically, distinct from each other: embolic or septicæmic, ordinary or scrofulous, and foreign-body phthisis. The first and the last forms are comparatively rare; the second is very common.

Embolic Phthisis. *Synonym:* *Acute Phthisis, Galloping Consumption.*—It is a rapid degeneration of the lung-structures, commencing in lobular solidifications of various sizes and strictly localised extravasations of blood. Multiple abscesses quickly form; the solidifications

soften into pus from the centre towards the periphery, with all the well known characters of pyæmic abscess. The primary lesion is apparently local arrest of the circulation, due to the plugging of the smaller twigs of the pulmonary artery, or to arterio-capillary embolism.

Here, gentlemen, is the first illustrative example of this form of disease.

On November 18th, 1872, a tall emaciated girl of eleven years, was admitted under my care in Faith Ward. Her mother, from whom we gathered the history of her illness, told us that the child had been ill for four weeks, having been feverish and much purged; but it was not till four or five days previous to her admission into the hospital that her state excited any serious apprehensions. Then, however, she became very noisily delirious, screaming out at times violently, but at others lying moaning and muttering. For four days she had recognised nobody about her, but took what food was given her, and lay in that semi-conscious state which you know to be so common in fever. Many of you saw her with me shortly after she had been put into bed, and heard what I said upon my examination of her. Her posture was a peculiarly uncomfortable one; she lay with her head thrown backwards on the pillow, and with her back bent in a manner that suggested a tetanic spasm. You see patients similarly bent by opisthotonos. She shrieked out directly I touched her. Nor was this because I had placed my hand upon any naturally highly sensitive or painful part; for, whether I touched her wrist, her face, or her belly, she resisted it equally, jerking about her limbs and twitching her muscles in a manner to suggest spinal irritation.

You will meet occasionally with equal restlessness and hyperæsthesia in typhoid fever in children, and especially in nervous children. There is a boy under my care at this moment in Radcliffe Ward, very ill and delirious in the fourth week of fever, who lies in a crouching attitude, bent forwards, and who is similarly hypersensitive. I augur ill of such cases. Not that they always die; but they pass very close to death's door, and make very tardy recoveries.

But, to revert to our case, the girl presented a high temperature—102 deg. The sordes about her lips, her dry chapped tongue, the rose-spots scattered more upon her back than over her abdomen, and, above all these, the history and duration of her illness, taken together, suggested the diagnosis of typhoid fever; but against this were to be set the following facts—that the abdomen was neither full nor gurgling, and the spleen was not noticeably enlarged. The possibility of tubercular meningitis had passed through the mind of Mr. Giffard, my very careful house-physician. But there had been no vomiting; the pulse, though feeble and very rapid, did not intermit at all; both pupils were of equal size; and I noticed the manner in which her eyeballs followed moving persons, and that she saw and watched what went on about her, although her eyelids were half-closed. When I came to examine her chest, the physical signs which I discovered rather embarrassed than assisted me to form an opinion of the nature of her malady. Her breathing was very shallow and rapid. I could discover no evidence of pleuritic effusion. Anteriorly and posteriorly, slight catarrhal sounds, crepitation, and sibilus, were to be heard all over the chest. The vesicular sounds were very unequal in corresponding parts of the two lungs; full and excessive compensative breathing occurred over the upper part of the right lung; faint distant respiratory sounds, marked by crackling crepitation, under the left clavicle. There was no lobar, no extensive dullness; but the resonance was very unequal. Patchy dullness existed, with nowhere the full tone which a chest-wall so thin ought to furnish. The scattered crepitation was of different kinds, but in parts was of that peculiar intermediate size and distinctness upon cough, that usually betokens breaking up of the lung-substance.

For treatment, I ordered fluid and stimulant nourishment at short intervals of time, and directed that the diarrhœa should be treated by small opiate enemata; but I held out a very hopeless prognosis. I never headed the board, but said I thought the case was one of acute consumption. The girl died four days after her admission. Her bowels acted twice only. She remained in much the same state, passing her urine under her, and only expressed herself the least intelligibly once the day before she died. I never ventured to examine her chest again, and the only feature further worth recording about her symptoms while alive is her temperature-chart. November 18th, (m.) 104.3, (e.) 103.2; 19th, (m.) 100, (e.) 104.3; 20th, (m.) 103, (e.) 101.6; 21st, 106 (m.); 22nd, 108.5 (m.), taken twenty minutes before death.

The *post mortem* examination conclusively proved the case to have been one of typhoid fever. The ulceration of the Peyer's patches was neither very extensive nor far advanced, except close to the ileo-cæcal valve; but the swollen softened state of the spleen and mesenteric glands Dr.

Gee considered quite pathognomonic. The lungs were good examples of the mode in which embolic phthisis begins. A great number of patches of deep congestion and well defined consolidation were scattered through both lungs, forming various-sized hepatisations, which often presented yellow centres when cut across. They varied in size from a marble to a millet-seed. They were spots of blood-stasis or stagnation in capillaries, of small lobular collapse, with extravasation of blood into the surrounding pulmonary tissue. In the upper lobe of the left lung, and on its anterior aspect, one such solid lobule, rather larger than a shilling in size, came close to the surface of the lung; it was a highly characteristic specimen, being sharply defined, and speckled throughout with tiny yellow dots. In many parts, small quite recent blood-ecchymoses were visible, which did not present yellow centres. The appearances were quite unlike tubercle, but exactly resembled those seen in the lungs of animals into whose veins during life any fine foreign material or filtered putrefying fluid has been introduced. There was an old cretified bronchial gland in a state of softening, and one or two of those ossifying cartilaginous plates often met with in the pleura. All the other organs were healthy.

You may accept the lung-state in this instance as a very early one of a peculiar form of scattered lobular consolidations, the intervening tissue being in parts congested, but crepitant or air-containing.

Those solidifications which consist of capillary blood-stasis, with some surrounding extravasation, of alveolar infarction or blocking up of air-cells with materials that are so deeply stained with the colouring matters of the blood that the normal histological elements are with difficulty recognisable, commence, I believe, in minute arterio-capillary embolisms. It is obvious that these solidified lobules must subsequently, if the patient live, pursue one of three pathological courses. The foreign altered blood-products must either admit of being totally reabsorbed, as pneumatic cell-products are, the alveoli reopening; or the tissues, the capillary walls, and the trabecular elastic frameworks of the air-cells, will suffer degeneration, softening, and separation from the parts whose circulation remains unimpaired by the ordinary process of abscess or pus-formation; or, as a third alternative, these lobules may partly obsolesce and partly cretify, their site remaining marked by permanent nodular pigmented masses, and themselves being circumscribed by some thickening of the fibrous interlobular tissue. Of the mode in which they degenerate and form abscesses, we have had a very recent good example. You will remember a dark sallow-complexioned woman of small stature, aged 28, who was sent down into my ward from that which is especially devoted to the diseases of women. She was said to be suffering from acute pneumonia, which, it was supposed, had supervened while she was in a state of great exhaustion after repeated losses of blood from one or more blood-tumours situated in the wall of her vagina. She had a subinvolted condition of uterus after parturition fifteen months previously, and had been in the hospital ten days, undergoing suitable treatment for her local malady, when on a sudden she complained of pain in her chest, her breathing became rapid, her temperature rose to 104, and her general condition excited grave apprehension. Upon examining her chest, a friction-sound was heard on the right side; the thoracic movements were painful; respiration shallow, 60 per minute; pulse 144, thready and feeble. Inspiration seemed to me comparatively prolonged; expiration short and whiffing. There was no complete dulness, such as marks the presence of pleuritic effusion or lobar pneumonia; and there was neither bronchial breathing nor bronchophony to be heard anywhere; but over both lungs, although especially over the right, I noticed great variation in the percussion-tone which I elicited. In spots within two fingers' breadth of each other, I found comparative dulness and excessive resonance; while, scattered irregularly throughout both lungs, my ear caught that fine crackling crepitation, accompanied by a little sibilus, so often heard in acute phthisis and lobular pneumonia. My diagnosis was acute phthisis and pyæmia; and the patient sank with symptoms of general peritonitis six days after I first saw her. I need not dwell upon the entire disease which the *post mortem* examination revealed. In brief, the apparent order of events was this: double ovarian cystic disease, with subinvolution of the uterus; this organ remaining, eighteen months after delivery, still of the size of a swan's egg, having walls two inches and a half thick, and presenting a diphtheritic-looking state of its mucous membrane. Abundant evidence of chronic as well as recent peritoneal inflammation existed. The right ovary had slipped into the posterior uterine pouch, and became bound down there by old adhesions. Its cystic enlargement in this situation had exercised pressure upon the pelvic system of veins, leading to an engorged and varicose condition of both uterine and vaginal veins, and the formation of blood-tumours in the wall of the vagina. Five such tumours were found, and one was in a sloughy gangrenous state; and hence, most likely, the final blood-poisoning had arisen. Embolic infarcta were found in the spleen

and kidneys, and also in both lungs, the parts to which I desire to direct your present attention especially. There was recent pleurisy upon the right side. Both lungs were much pigmented; and little abscesses, of various sizes and in every stage of development, were scattered through them, from the minutest halo of ecchymosis about a tiny yellow centre up to ragged cavities of the size of a walnut. The walls of the smaller bronchi were swelled and thickened, and patches of lobular pneumonia pervaded the upper and lower lobes of both lungs indifferently. It was impossible from a naked-eye examination to distinguish the ultimate alveoli, thus in parts blocked up with products of inflammation from what is ordinarily called phthisis. The consolidated lobules, which are the seat of lobular pneumonia, appear under the microscope stuffed with proliferating cells, and entirely coincide with what are ordinary pneumonic appearances.

Now here was phthisis of its most acute kind—small abscesses of various sizes, with interspersed lobular pneumonia. The disease existed in a later and more developed stage than in the case to which I first directed your attention. Having hardened portions of this lung, I made some fine microscopical sections, which I will show you. They show the blocking up of minute twigs of the pulmonary artery by emboli, as I believe. And such, I apprehend, is the origin of this form of rapid degeneration of the lung-tissue—necrobiosis, or death in a living coffin, as it has been named. The frequently attendant lobular pneumonia is perhaps determined by collateral blood-flux.

Let me briefly sum up, then, the characteristic symptoms during life of this form of phthisis. Foremost is its rapidity. To-day you may hear a slight friction-rub, with feeble breath-sounds; perhaps notice a little less than normal resonance. To-morrow there will be abundant crackling crepitation. On the third day, there will exist such crackling and churning sounds as signify the disintegration of pulmonary tissue. It begins with pain, high fever, rapid breathing. There is a short, frequent, almost dry, unsatisfying cough, very irritating to those who have to listen to it. Some of you may remember how the nurse complained that the other patients in the ward could get no sleep because of this poor woman. A little rusty pneumonic sputum was expectorated; but it was very scanty. Indeed, in neither of these two cases was there any expectation to assist the diagnosis. Perhaps, even when the softening takes place, it may communicate but little with bronchi, and have no easy channel of exit.

In some instances, dark-coloured blood is, I believe, expectorated; but I have never seen such cases. Still, when we consider the amount of ecchymosis or blood-extravasation which in both these cases was discovered round about the lobular infarction, it is easy to comprehend that dark blood, as is seen in cases of pulmonary apoplexy, might be spat up.

But, you ask, why do I connect these two cases of evident pyæmic abscess in the lung with phthisis at all? It is because the history of not a few cases of pulmonary consumption is of this kind. A patient has typhoid fever, with lobular pneumonic symptoms, who has never had any lung-affection before in his life. He recovers from his fever, but has a slow and imperfect convalescence. Within a few months, he comes again into the hospital with manifest phthisis, having cavities of some size in the middle or lower parts, rather than quite at the apices of his lungs; and dies in from a few weeks to a few months after he was first taken ill.

A woman has a painful delivery, followed by pelvic cellulitis. Of this we have had more than one example within the last six weeks. Her breathing becomes oppressed; she gets a cough, with purulent expectoration; she never has hæmoptysis, but presents scattered crepitation with tubular breathing; and anon manifests cavities, pectoriloquy, hectic fever, and general wasting. How, and when, and why, did this phthisis begin? There is no hereditary predisposition to explain it. The chest-shape is not at fault. Hæmoptysis is an exceptional symptom; although occasionally, late on in the complaint, a vomica may lead to some pulmonary arterial aneurism, and large and perhaps fatal final hæmorrhage.

The first beginning, when this is watched for, has been sudden oppressed breathing, with tubular respiratory sounds, patchy dulness on percussion, followed by crackling crepitation, which either becomes larger in character and passes into gurgling and churning sounds, or else, after an uncertain interval, disappears; its site being marked, however, by persistent dulness on percussion and impaired vesicular sounds.

You are aware that materials alien to the blood, unfit for circulation and the nutrition of the tissues, are especially prone to pass into the circulation, when glands which may be regarded for the nonce as blood-filters are breaking or broken down, and when the veins themselves are diseased, varicose, or in parts the seat of thrombosis. The blood of the whole body has to pass through the lungs. Is it surprising that unassimilated

incompatible materials should suffer arrest in the fine capillary meshes of the air-cells?

All that I am pleading for are grades of pyæmia. The acute or more rapid forms, with multiple abscesses in the lungs, are, in point of causation or pathology, well understood; but wherefore withhold a like etiology—minute arterio-capillary pluggings—from those cases of phthisis which follow fever, ague, protracted cases of rheumatism complicated by bed-sores, and those other debilitated blood-states in which embolism and thrombosis are known so frequently to arise?

If you find the term embolic phthisis still too hypothetical, call this variety blood-poisoning phthisis; but remember all its principal clinical facts. The lung-symptoms are always of sudden access. They follow or occur as complications upon some other grave disease. The fever is continued, but the morning temperature is usually higher than that of the evening. Lobular pneumonic symptoms are speedily and generally extensively exhibited; but, unlike ordinary scrofulous phthisis, early bronchial catarrhal symptoms, cough, and hæmoptysis, are not forthcoming. You will find this form of disease very little amenable to treatment. I do not say it never suffers arrest, for I believe that it does, and that I have seen such cases; but they are exceptional, not to say most rare. It happens more often to dark sallow-skinned than to lymphatic persons. The treatment which I have found best adapted to the general condition is sustenance such as can be taken; stimulants, for these are requisite in lieu of food which the stomach will not support; and, as medicine, a mixture in which infusion of digitalis, nitrate of potash, and the watery extract of opium, are the potent ingredients.

ON THE ORIFICES OF THE UNIMPREGNATED UTERUS, AND THEIR SURGICAL TREATMENT.

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IN the throes of departure from my winter retreat at Mentone, I have hitherto failed to notice Dr. Matthews Duncan's strictures on the above subject (BRITISH MEDICAL JOURNAL, March 8th). An interval of repose, however, affording me the required leisure, I again revert to this important question.

Dr. Matthews Duncan's explanation of his practice in dilation of the uterine orifices forms a most necessary corollary to his "scientific" papers. The former certainly left on the reader's mind the impression that he was a much more enthusiastic "dilator" than he proves to be. Thus, he repudiates the existence of actual strictures of the uterine orifices comparable to those of the male urethra; he admits the usual permeability of the uterine canal to the bougie or sound, and rejects dilatation as a panacea for sterility; indeed, he accepts a greater degree of natural permeability of the entire uterine canal than I myself am prepared to admit. Thus, I can generally introduce, with a little firm continued pressure, a small wax bougie, warmed, bent to the natural curvature of the uterine cavities, and used with a speculum; but I also constantly find, in the same cases, the uterine sound, or a larger bougie—even a 6 or an 8, still more, a 12 or 13—arrested at the os internum, whether introduced with or without the assistance of the speculum. Dr. M. Duncan, indeed, appears all but to confine dilatation to spasmodic or pseudo-membranous dysmenorrhœa, and that all but empirically, without seeing exactly how it acts. If so, I am still at a loss to know what are the classes of cases in which the uterine sound is bent in overcoming resistance, or in which the resistance overcome is equal to a pressure of four pounds. I repeat that, in my opinion, in uterine practice such a degree of force is altogether unjustifiable, whatever the nature of the case, even experimentally. To use it, is to court disasters and catastrophies.

Satisfactory as Dr. M. Duncan's explanation of his practice appears to me, satisfactory as it is to me to learn that he mostly agrees with the doctrines I teach, I must be allowed to add that he does not explain in what respect it is that he considers the views and statements contained in the paper I read last August before the Association at Birmingham, "anatomically, physiologically, and pathologically wrong". I consider that I have a right to ask whether these words were written hastily, without deliberation, or whether they are the true expression of his sentiments. In the latter case, the questions at issue are certainly of sufficient weight and scientific importance in gynecology to demand due discussion and consideration. I would also remark that it is on these very views that my own practice and teachings are founded. I will recapitulate in a few words the opinions which I entertain and teach respecting the uterine orifices.

1. *Anatomically*, the uterine cavities are two in number; the cavity

of the uterus, triangular; the cavity of the cervical canal, infundibuliform. The cavity of the cervical canal is limited—externally, by a contraction of the os externum, usually patulous, and easily dilatable; internally, by a more decided contraction, which separates it from the uterine cavity, and is called the os internum. This internal contraction, I maintain, is formed by the circular fibres of the cervix, and constitutes a species of sphincter, although no positive sphincter, like that of the anus, is anatomically demonstrable. This double sphincter reproduces, anatomically, the sphincters which close the other large splanchnic cavities—the stomach, the intestines, the bladder.

2. *Physiologically*, these sphincters, especially the uterine one, are vital, close and open by a vital contraction; and a more or less vital contraction and closure is the usual condition during life. After death, this vital contraction ceases, as well as that of the sphincter ani, and must not, therefore, be looked for. These sphincters open for menstruation, probably for conception; and the internal one closes on the contact of a foreign body, such as the uterine sound. They are also more or less susceptible of contraction, according to individual idiosyncrasy. Thus in a perfectly healthy woman (in the interval of menstruation) having a perfectly healthy uterus, the uterine sound or a bougie (No. 8 or 10) separates the lips of the os internum with ease, passes into the infundibuliform cavity of the cervix, where lateral motion is generally possible, and reaches the os internum, one inch and a half from the os externum, there to be arrested, generally speaking, by its vital sphincter-like contraction. Sometimes slight pressure will pass it into the uterine cavity, sometimes it will not. Nothing short of unjustifiable force will succeed; although in these same cases a smaller wax bougie, warmed and bent, will generally pass.

3. *Pathologically*.—Such being the case anatomically and physiologically, the healthy os internum in the perfectly healthy uterus usually resisting the passage of the uterine sound or of a moderate sized bougie, it is a most egregious pathological error to consider this mere impediment to the entrance of the sound, apart from other morbid phenomena, a pathological condition warranting dilatation or division. So far from resistance to the sound at the os internum being a sign of disease, it is a sign of a healthy natural vital state of the parts. The free and easy passage of the sound into the uterine cavity, without pressure or force, is, on the contrary, generally an evidence of an actually morbid state of the organ. The extension of inflammation of the mucous membrane of the cervical canal to the cavity of the uterus, relaxes and paralyses the os uteri internum; so do endometritis itself, or any morbid state that enlarges the body of the uterus; metritis, acute or chronic; non-involution of the uterus after labour or abortion; or a fibrous or other growth developed in its tissues. Inflammatory hypertrophy, however, of the cervix may mechanically approximate, partly close, the entire cervical canal. The hypertrophy removed, the canal relaxes of itself.

Such are the views and statements which Dr. Matthews Duncan has stated to be *seriatim* wrong. I repeat that it is of the most vital importance that the real anatomical and physiological condition of women should be cleared up and firmly established as a basis of pathological work. If the above views be right, any healthy woman in the kingdom, casually examined, may be pronounced pathologically wrong, and treated for strictures which do not exist, dilated or divided—a really frightful state of things. Indeed, I firmly believe that hundreds are so treated every year, conscientiously, but under the influence of mistaken ideas as to the anatomy and physiology of the uterus. Indeed, many who have written and practised during the last few years seem to look on the uterine canals as comparable to the waste-pipe that carries off the water from the roof of a house, and to them nearly all uterine pathology is centered in fancied or real obstructions; just as another class of modern gynecologists have got into their heads that the uterus, the most movable organ in the economy, the "animal in animal" of the ancients, is a mere joint, like the knee- or elbow-joint, liable to dislocations, twists, and turns of every kind, and that nearly all uterine pathology lies in these fancied and exaggerated displacements.

Under the influence of these fallacies and errors, I am constantly seeing errors in practice, cases of constitutional lifelong dysmenorrhœa (or painful ovulation) divided or dilated by one, treated by pessaries left for a year or two by another, irrespectively of all constitutional considerations, and with no beneficial result whatever. The dependence of menstruation on ovulation, of ovulation on the nervous system, of the nervous system on diathesis, on hereditary and health antecedents, is often entirely ignored; and in one of the most complex branches of pathology, imperfect surgical notions are often substituted for rational treatment or non-treatment. In many of the cases thus surgically mistreated, actual lesions, important lesions, inflammatory and nutritive, are not unfrequently overlooked and ignored.