Anatomy, on which surgery intimately and necessarily leans, was advanced during the following centuries by a host of distinguished men. In Italy, Eustachius, Fallopius, Cæsalpinus, Fabricius, Asellini, Malpighi, Morgagni, and Scarpa followed close on one another. In Switzerland, Hildanus and Haller, "the father of physiology", brought renown to their country and credit to medicine. In France, appeared Belloste, Dionis, Le Dran, Winslow, Anel, Petit, and Desault; in Holland, Scultetus, Ruysch, Nuck, and Boerhaave; in Germany, Heister, Albinus, and Richter. In England, Lord Bacon expiscated the laws which should govern observation and judgment; while Harvey immortalised his age by his great discovery; and Sydenham, "the modern Hippocrates", and others, practised and wrote. The French Academy of Surgery, founded in 1743, acquired the highest authority, and its *Memoirs* are replete with valuable observations and reflections.

Along with all this progress, there was, however, much gross quackery and ignorance. Sir Kenelm Digby, with his sympathetic powders; Crazy Sally, and others, with their unscrupulous pretensions; cancer-curers, witches, the Royal touch ("Rex tangit te, Deus sanat"), fasting-girls, and a host of other impostors, flourished in the sixteenth and seventeenth centuries. An account of their doings forms one of the most curious, as it is one of the most humiliating pages in the history of our country. The ridiculous dress and manners, too, of the regular practitioners of that time did not tend to exalt the profession in the eyes of the public, but called forth much satire and contempt. Morel made the first attempt to produce the tourniquet, which Petit completed, and thereby added a most important item to the surgeon's armamentarium.

If my time had allowed, I would very gladly have referred to the careers of two very different men who lived in the eighteenth century, and who, though not surgeons, had a great influence on the medical opinion of their day. I refer to Cullen and Brown. The former did admirable service, and, by establishing clinical teaching in Edinburgh, contributed largely to the early celebrity of that school. The latter, though said to have originated a system, and to have much interested the speculative theorists of his day, was in reality so crazy or eccentric as to have well merited the epithet of "the Paracelsus of Scotland".

[To be continued.]

USE OF THE ACTUAL CAUTERY IN PHARYNGEAL DISEASE.*

FOR the last six months, I have had in use at the Throat Dispensary the method recommended by Dr. Carl Michel (*Deutsche Zeitschrift für Chirurgie*, 1873) for granular and nodular thickenings of the mucous membrane of the pharynx. I can fully corroborate the statements made by him and others as to the admirable results of this mode of treatment in this usually intractable affection. Not only in thickenings of the mucous membrane, but also in enlarged tonsils (chronic), may a rapid and effectual change for the better be confidently anticipated from the use of the cautery.

For private practice, the galvano-cautery (a convenient form of which was supplied to me by Messrs. Mayer and Meltzer) is best suited; but, in dispensary practice, I have from the first used simple cautery-irons, which are heated in the fire and applied forthwith to the throats of the patients. These cautery-irons are made of thick wire with a tapering bulb at one end and a hook at the other end. The bulb is necessary to retain the heat, as a wire is soon cooled by the mucus, and the application thus deprived of its efficacy. The whole length of the iron is fifteen inches: the thickness of the bulb being about a quarter of an inch. The hook at the end facilitates the carrying of the iron when in a heated state. By arranging matters so that the patients sit with their backs towards the fireplace, the process of heating may be carried on without exciting any alarm; and if, as should always be the case, the iron be applied at a black or very dull red heat, the patients do not as a rule object to the use of the cautery. I have now treated eighty-three cases of chronic pharyngitis with thickening of the mucous membrane by various methods. Of these eighty-three cases, thirty-five have been treated by the actual cautery, and I have adopted it almost exclusively in the treatment of this disease.

It is important to notice that each thickened nodule should be separately cauterised. Dr. Riesenfeld uses a sort of knife-shaped cautery, with which he strokes the surface of the pharynx. This appears to be destructive of the sound as well as of the diseased mucous membrane, and therefore unnecessarily severe.

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* Abstract of a paper read before the Medico-Chirurgical Society of Glasgow.

CLINICAL LECTURES ON THE VARIETIES OF PHTHISIS.

Delivered at the Hospital for Consumption, Brompton.

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LECTURE I.—PATHOLOGY AND CLASSIFICATION.

GENTLEMEN,—The disease which we are called upon to treat in this hospital on a large scale, phthisis pulmonalis, has been the subject of many attempts at classification in the hands of careful observers. Some have taken an anatomical basis for their arrangement, others a clinical one; others, again, have tried a compound of the two, and have made the classification partly clinical and partly pathological. A fourth set have declared that it is impossible to divide phthisis into any satisfactory classes at all, and have stated that the only safe arrangement would be, according to duration, into acute and chronic cases, all other distinctions being purely dogmatic and artificial, and the disease one and indivisible. While the difficulties of any definite arrangement are very great, it must be admitted that the attainment of this object is highly desirable simply for convenience sake; for we find ourselves talking about scrofulous phthisis, pneumonic phthisis, laryngeal phthisis, and so forth, without explaining what we really mean by those terms; and, if we could separate cases of consumption into groups, not necessarily sharply defined, but each presenting some leading anatomical or clinical feature, we should considerably improve our chance of securing good records and adding largely to our stock of well arranged knowledge. In order to do this, we must take a survey of the chief pathological elements of consumptive disease, as in the preponderating influence of each of these in different cases lies often the secret of the varied forms the disease assumes. In too many instances, the *post mortem* examination of a case of phthisis reveals little of the various morbid processes which have brought about death. In the mere shell of a lung that remains, we see that not only have normal structure and function been obliterated, but that the diseased products, the very destroyers themselves, have undergone disintegration and removal. From such specimens, we learn little or nothing; but, in those less advanced, we are able to recognise the principal actors in the tragedy, and to assign to each its proper part. The pathological appearances are briefly as follows.

1. The various kinds of pneumonia: croupous, catarrhal, and interstitial.
2. Tubercle: grey and white, discrete and aggregated.
3. Necrobiotic changes, *i.e.*, caseation, ulceration, and cretification.
4. Fibrosis.
5. Lung-pigmentation.
6. Various morbid changes in the bronchi, bronchial glands, and pleuræ.

Let us now briefly consider these processes in their relation to phthisis, beginning with the pneumonias.

1. *Croupous* pneumonia is the form of pulmonary inflammation where lobes, and not lobules, are primarily affected, and is characterised by its three well known stages:—1. Engorgement; 2. Red hepatisation, marked by exudation of fibrin and red corpuscles, and leucocytes, with occasional rupture of capillaries; 3. Grey hepatisation, where further emigration of leucocytes is accompanied by epithelial proliferation and followed by retrogressive changes in the exuded products. This form of pneumonia most commonly results in (1) resolution, through liquefaction of fibrin, fatty disintegration of epithelium, and subsequent absorption or expectoration of exuded materials. It may also end (2) in abscess, (3) in gangrene, and (4) in chronic pneumonia. We need not concern ourselves with any but the first and last forms of termination, as the others are not common in phthisis. It is as well to note that croupous pneumonia is to be found in phthisical lungs, as I have heard it stoutly maintained that such a product cannot exist under the circumstances, as consumptive patients do not possess that standard of health which would enable them to produce anything but catarrhal or caseous pneumonia.

You can easily convince yourselves of the accuracy of my statement by a visit to the *post mortem* room, where you will find plenty of examples of all the stages of croupous pneumonia, especially of red hepatisation in connection with cases of recently formed tubercle. Whole lobes are seen consolidated and impervious to air, and, on section, show aggregations of miliary tubercle, either grey or commencing

to caseate, scattered through them, standing out in bold relief against the deep red of the rest of the hepatised lung. Some of these masses of consolidation become absorbed, and we can trace the process during life by the accompanying physical signs. More frequently the lung does not clear up, the exudation remains, becomes organised with blood-vessels and lymphatics, and thickening of the alveolar wall takes place; in fact, the pneumonia becomes chronic, and may lead at a future time to further retrogressive changes.

Catarrhal pneumonia or broncho-pneumonia is commonly met with in phthisis, and is distinguished pathologically from croupous by its starting from the bronchi, by its involving not lobes, but lobules; by its being accompanied by pulmonary collapse; by its limited character and histological products. The colour of the points affected are greyish yellow, and they vary considerably in size from a pin's head to a cluster of lobules, giving to the lung a marbled appearance, from the admixture of pigment contained in the alveolar wall. The alveoli are blocked and stuffed to distension with large epithelial cells, which contain several nuclei. There are also leucocytes and fibrin exuded from the adjacent vessels.

This form is very difficult to resolve; the cells in time caseate and become expectorated, or are absorbed, and the absorption of this material appears to cause irritation and consequent hyperplasia of the lymphatics; but more will be said on this head when we discuss catarrhal phthisis.

Interstitial pneumonia is a chronic inflammatory process of the lung, in which thickening of the alveolar walls and of the interalveolar tissue takes place by means of a fibro-nucleated growth, and a gradual obliteration of the alveoli is the result. It is generally unilateral. The pleura and interlobular septa are largely thickened, the lung becomes shrunken, the alveolar structure destroyed, and the bronchi dilated. These latter are sometimes the seat of secondary inflammation, owing to the difficulty of expectoration and to the retention in them of irritating secretion. Ulceration takes place in their walls, and thus excavations are found in connection with the tubes.

Interstitial pneumonia generally follows one of the acute forms of pneumonia, and is identical with cirrhosis of the lung, and, moreover, is the main element of fibroid phthisis. Such are the pneumonias found in phthisical lungs, and we must take full note of them, as they are important landmarks in separating the different varieties of phthisis.

2. The forms of tubercle are *grey*, *white*, and *yellow*, and they vary considerably in size, from a pin's head to a hempseed, and are found singly or in groups. The grey tubercle or miliary tubercle histologically consists of two elements:—*a*. Large multinucleated cells with branching processes, containing much granular material and bright nucleoli; *b*. A reticulum of adenoid, or small-celled, tissue. This latter consists of delicate fibres interlacing, and containing in the meshes a number of small lymphoid cells. There are also a certain number of epithelial cells.

The white tubercles differ only from the grey in the fact that the epithelial element preponderates. The yellow tubercle, on the other hand, is grey or white variety undergoing caseation, and being gradually converted into fat or amorphous granules.

As regards the grey tubercle and its histological constituents, there is much discussion, especially as to the part played by each and its origin.

Klein affirms that, in some of the most rapid cases of acute tuberculosis, no adenoid growth is to be found, but only desquamative or catarrhal pneumonia; and states that, in some specimens examined by him, in the central or earlier portion of a tubercular nodule there was nothing but fibrinous exudation, and, at the peripheral or later portion, there was no fibrinous matter, but only spherical multinucleated cells; or, again, else the alveolus was filled by one large multinucleated or giant-cell. Klein considers that these different appearances represent different stages of the process. First comes the fibrinous exudation and fills up the alveoli, but is gradually absorbed by the surrounding tissue, which is in a state of increased activity, as is shown by its infiltration with fluid, and by the distended blood-vessels. The exudation disappears and is replaced by groups of cells or by one giant-cell. If the irritation last long enough, the small-celled tissue appears in the alveolar wall.

The nature of the giant-cell is much disputed. Hering says that it is simply a lymphatic cut across; Ziegler, that it originates from colourless blood-corpuscles; Klein and others maintain that it is the offspring of the alveolar epithelium; and this last theory, from the position of the cell, and the possibility of its isolation, seems most probable. Dr. Green, whose admirable drawings and specimens have done much to elucidate the pathology of phthisis, considers that the differences in the histological characters of tubercle depend on difference in the ages of nodules and the intensity of the tuberculous pro-

cesses. If the process be very intense, the nodules will consist almost exclusively of epithelial accumulations and fibrinous exudations within the alveoli, and they will undergo disintegration. If the process be less intense, nodules attain an advanced age, necrosis is less rapid and complete, and, therefore, there will be more time to develop adenoid tissue and multinucleated cells. Lastly, in the least severe and most chronic forms, the formation of multinucleated branched cells and adenoid tissue reaches its maximum.

This seems to me a very reasonable view, and one likely to reconcile the apparently conflicting views held by distinguished pathologists as to the nature of tubercle; for, while Rokitsky and Dr. C. J. B. Williams maintained that tubercle was an exudation from the blood-vessels, the latter assigning a large part in the process to the leucocytes or *sarcophytes*, as he calls them, Drs. Sanderson and Wilson Fox conclude from their observations that it is entirely a lymphatic, or rather adenoid, growth; and that, in all specimens of tubercle, the small-celled tissue is present. Dr. Klein and Dr. Green's preparations show that all these processes may be present, and that their appearance or non-appearance are questions of time and of intensity of the tubercular process.

3. The *necrobiotic* changes in tubercle arise partly from its non-vascularity and partly from the adenoid growth invading the vascular walls and in time blocking up the vessels. Caseation takes place first in the grey and white tubercle, yellow tubercle being the result; the large aggregations become involved in the process, and we consequently get caseous masses of various sizes. In time, communication with a bronchus is established, and the caseous mass is removed by ulceration and expectoration, a cavity marking its site; or again, the caseous mass may remain quiescent in the lung and increase of the interlobular tissue may take place in its neighbourhood, finally encapsulating and isolating it. In this condition, the mass soon loses moisture and passes from caseation into cretification, and is converted first into material like mortar, and then into the calcareous material often expectorated by consumptives.

4. Another ending of tubercle is *fibrosis*. This in old cases is a very common transformation, the histological changes being as follows. The small-celled tissue and its reticulum exhibit a tendency to assume spindle-shaped forms, the nuclei become more and more elongated, and, in a short time, fibroid tissue has formed.

This tissue plays a very important part in the pathology of phthisis; for, whilst it is in many cases an agent of invasion, choking up alveoli by spreading along their walls, it is remarkably free from any tendency to break down, but remains firm and tough to the end of the chapter. Its chief function is to limit the area of the destructive processes, which it often does most effectually, isolating portions of the lung which are the seat of active tuberculation or of softening; and, by its tendency to shrink, it promotes the cicatrisation of cavities.

5. *Lung-pigmentation* is common in cases of phthisis of long standing, and appeared to be the result of chronic inflammation; but I believe its exact causation is not yet known.

6. The changes in the *pleura* in phthisis are various. In most chronic cases, adhesions and thickening are found, and generally overlying the diseased portions of lung. At the apices, which are most commonly the earliest portions attacked, the pleura is often enormously thickened and of cartilaginous hardness, proving, in the case of cavities, a good bulwark against any chance of pneumothorax. At the base of the lung, the thickened pleura often undergoes that change which has been so well described and explained by Dr. Douglas Powell. The layers, each somewhat thickened, are separated by a striated jelly-like material, which is oedematous connective tissue, the fine striæ of which pass vertically from one pleural surface to another. The explanation is, that the shrinking of the damaged portion of lung causes a considerable separation between the parietal and visceral layers of pleura, and this space is filled in by serous fluid effused into the meshes of the connective tissue of the stretched adhesions. In instances of phthisis arising from pleuropneumonia or pleurisy, we get the whole pleura adherent and thickened. The changes in the bronchi in phthisis partake, to a great extent, of the changes in the alveolar wall, and we find inflammatory and catarrhal products, ulceration, tuberculation, and fibrosis present in different cases. The bronchial glands undergo pigmentary change in some patients, becoming exceedingly dark in colour, and in others undergoing caseation and cretification.

Before quitting this part of the subject, let us bear in mind that the main histological changes in the lung of phthisis, as defined by Dr. Green, are: 1. Accumulation of epithelial cells within the alveolus; 2. Exudation of fibrin and leucocytes within the alveoli; 3. Thickening of walls of alveoli and of minute bronchioles by small-celled tissue; 4. Increase of interlobular connective tissue. And we must not forget that, in the formation of even the simplest form of lesion—*e.g.*, grey miliary tubercle—may

include the first three processes, so that we cannot regard any one growth or formation as a basis of tubercle; it is rather a combination of several processes—adenoid growth, epithelial proliferation, and exudation from the blood-vessels—each playing its part, and giving place in time to necrobiotic changes.

Having now before us the pathological agents concerned in a case of phthisis, let us inquire the conditions of their action. These are three:—1. Predisposition. The patient must be predisposed, not necessarily through hereditary or family taint, for this does not exist in more than half the number of consumptive patients, but there is generally some depressing cause, such as hard work, insufficient food, or great anxiety, which weakens him, and renders him more liable to the action of exciting causes of disease. 2. Irritation. This is generally manifested in the lungs, either on a portion or on the whole, by various exciting agents, such as bronchial catarrh or inflammatory attacks, involving the various pulmonary tissues, causing epithelial proliferation, exudation from vessels, followed by necrobiosis. 3. Infection; or, as Dr. Sanderson defines it, the property by which, if a chronic induration due to overcrowded corpusculum exist in any organ, it is apt to give rise to similar processes elsewhere. The process of infection varies in degree, and may be well studied in phthisical lungs. The simplest form of infection which we see is where a caseous mass is surrounded by a zone of miliary tubercle, the granulations being arranged in a circle around the caseous focus, or in lines radiating from it. In these cases, the infection has been traced to the lymphatics, sometimes those of the alveoli, and sometimes those of the bronchi.

Where only small areas are involved, and where the disease does not extend beyond the lungs, it is likely that infection takes place through the lymphatics; but, where both lungs, or where other organs are attacked, the vascular system is probably the path of infection.

Why, it may be asked, are some lesions infective and others not? This is often the result of their position in the lung, and, therefore, to a certain degree, accidental. When a caseous mass is encapsulated in fibroid tissue, and thus shut out from the vascular and lymphatic systems, it is not likely to give rise to infection; but if, on the other hand, it be in connection with one of the numerous sets of pulmonary lymphatics, or, again, if it be surrounded by a zone of vascularity, it is very likely to become the focus of tubercular infection.

[To be concluded.]

ON URÆMIC ASTHMA.*

By T. CLIFFORD ALLBUTT, M.A., M.D.,

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THE affection upon which I venture to speak to-day is one of very great interest to us both as scientific and as practical physicians. Its phenomena are as striking and strange in themselves as they are inexplicable; and, on the other hand, the affection itself is one of the most agonising by which mortals are afflicted. I am not sure, indeed, from my own experience, whether the existence of a certain dyspnoea dependent directly upon uræmia, or connected directly, at any rate, with renal disease, is generally recognised by medical practitioners.† That persons having renal disease have therewith pulmo-cardiac and pleural affections, mostly of a secondary kind; and that such persons suffer from more or less dyspnoea is, of course, familiar lore. What I mean is, that I do not find it to be by any means familiarly known that the subjects of Bright's disease are liable to definite seizures, mainly consisting in intense dyspnoea, coming and going as ordinary asthma comes and goes, and depending as little as this does upon any permanent disease of the lungs.

First of all, then, I will describe the affection as I understand it, and, in doing so, I will keep to clinical facts as nearly as possible. Uræmic asthma is seen in its simplest and, I think, also in its worst form, in that kind of Bright's disease which is known as chronic granular kidney. It may be seen in any state of uræmia, whether this be dependent upon permanent or transient renal disorder; but, in the subjects of granular kidney, the affection is often very severe and very recurrent, and is often dissociated from pleuritic effusion, valvular disease of the heart, and other permanent causes of dyspnoea. How in-

sidiously granular renal disease, with the constitutional state which belongs to it, or to which it belongs, may creep on is well enough known. For months, or even years, it may betray itself only by some loss of flesh, by some fading of the skin, by unusual fatigue after exertion, and so forth. During this time, vascular tension and cardiac hypertrophy are slowly advancing and establishing themselves unknown, it may be, to anyone. One day may then come when such an one has an unpleasant interview with his solicitor, a warning from his doctor, or a quarrel in his home, and that night he is suddenly seized by a dyspnoea so terrible that he has to spring from his bed and strive for his very life, as it seems, for one, two, or three hours before peace brings sleep to his pillow, or rather to his pillows, for never again perhaps will he sleep with less than three or four pillows under his head and shoulders. Not that uræmic asthma always occurs at night any more than epilepsy does, but, like epilepsy, its first attacks are often nocturnal, and often seize the patient in his sleep. Indeed, throughout the malady, the asthma is more terrible at night, as cardiac dyspnoea generally is likewise; still, in its repetition, uræmic asthma becomes more and more irregular in its recurrence, and finally may not wholly disappear for one hour out of the twenty-four. What happens is something as follows. The patient, if awake, becomes aware that his respirations are quickening and are shallower. The distress increases, and a throbbing labouring action of the heart intensifies it. The countenance now becomes anxious, apprehensive, or even terrified, and a somewhat peculiar general muscular restlessness comes on, which seems to be something more than the mere striving for breath or air. Now, with this intense distress, which anon becomes more than this—an agonising, almost mortal conflict—the face is not puffed, congested, and blue, but nipped and pale, and the very lips themselves are blanched. This, in my experience, is always the case, and the observation is a very instructive one. Moreover, in many instances, though by no means in all, a more or less profuse sweat is of the essence of the attack, comes on, that is, with the primary phenomena, and not as a mere consequence of effort or fear. In two cases, I remember that an outbreak of perspiration upon the usually harsh skin was the very first symptom in the train of symptoms which constituted the attack. In other cases, sweating is unimportant in degree, or is even absent. To the ordinary observer, then, the patient is alarmed, and his aspect is apprehensive and pallid; he is extremely restless; he sits upright and breathes shallowly and rapidly; he speaks in gasps or makes rapid fretful signs, and not infrequently he is covered with profuse cold perspiration. To these observations the physician will add as follows. The temporal arteries stand out like pulsating cords, the radial and tracheal arteries are of tendinous rigidity, and the blood-stream is forced into these and other arteries under great pressure; the big overwrought heart heaves over a wide area of the left chest and seems to threaten to burst its bonds. There is as yet little or no cough, and but little sound of phlegm in the air-passages. The chest expands duly, and, on listening over the lungs, the air is heard to enter freely and rapidly over the whole of them, unless their capacity be lessened by some previous disease. After this contest has gone on for a longer or shorter time, the attack relaxes its hold, the respirations become easier, words and sentences are spoken, the face recovers some tranquillity, and there is usually some expectoration. This expectoration is frothy mucus, often tinged with blood; and, in one case under my care, every violent attack was followed by distinct pulmonary apoplexy in patches large enough to be mapped out by physical examination. In this case, the hæmoptysis was, of course, considerable. In all cases, the lungs fill with râles before the termination of the attack. Such are the characters of pure uræmic dyspnoea as seen apart from complications.

Let us now ask ourselves what explanation we can find as we sit watching this awful suffering. The first thing that strikes us is, that the condition is not one of cyanosis, but rather of pallor, shrinking, and incipient collapse; it so much resembles an attack of ordinary asthma in these respects, that the name uræmic asthma may properly be given to it. It is on listening to the chest that we find the most remarkable contrast with common asthma, in the perfect permeation of the pulmonary tissue by the inspired air. It is very strange to witness this strife for breath, as it seems, while, at the same time, we hear the air passing freely throughout the lungs. Indeed, the patient tells us, and we ourselves may see that he is not, as in asthma, unable to draw his breath, but that the drawn breath brings him no relief. It is clear that the air and the blood do not meet in the air-cells, but that the fault does not lie with the air. It must be the blood, then, which does not keep its appointment. Now, in an uncomplicated case, there is no permanent obstruction to the passage of the blood through the lungs; indeed, we know that, in a short time, the air and blood will come again together and the patient will find peace. How is this? Our thoughts now turn to asthma again, and we think of

* Read before the Yorkshire Branch.

† Not only in my own intercourse with my brethren do I find doubts or hesitation about this symptom of uræmia, but I find in medical literature that the true nature and importance of these attacks is only recognised in the latest works on the subject. Dr. George Johnson has recently described uræmic asthma in this JOURNAL; and Dr. Dickinson describes it plainly in the new edition of his work. Uræmic asthma is, however, not mentioned in his first edition (1868); and it is referred to very doubtfully by Dr. Roberts in the chapter on Uræmia in his edition of 1872.