cervical spinal cord, the right anterior horn was softened. The parts affected were the origins of the hypoglossal and facial nerves, but those of the spinal accessory and pneumogastric presented less decided change. It was interesting to find, however, that the recurrent nerve seemed to retain its power over the vocal cords, although the superior laryngeal nerves had diminished sensibility.

In reference to a weakened condition of the vocal cords, or diminished tension, or loss of contractile power, the disease may be of central origin, or local, or of a reflex character. We often find, in reference to the first, that the disease is in the will; the effort to produce contraction of the cords is not made; the patient speaks in a whisper. A few months ago, a patient of this kind was under my care in Guy's Hospital who had not spoken for two years; there was no evidence of organic disease, and the first application of an electric current to the larynx produced the loudest protestations that she could speak. This state has been called by Romberg phthisical aphonia. Sudden nervous shock may also produce this loss of power of the vocal cords. Other weakened conditions of the nerves of this part are due to local disease, to diphtheria, croupous diseases; and even after catarrh the muscles become weakened, and sympathise in the inflammatory affection of the mucous membrane. The application of electricity often affords great relief, and, in milder cases, the effort to produce musical notes and to sing tends to increase the tension of the weakened cords.

Still more interesting are the forms of paralysis of the recurrent nerve and of the vocal cords which are due to reflex action. Any form of pressure upon the recurrent nerve may induce altered nutrition and hoarseness, the consequence of the paralysis; for the recurrent supplies the vocal cords and, with one exception, the muscles of the larynx. The larynx may be examined by the laryngoscope, which becomes an important help in diagnosis, and the vocal cord may be seen on one or other side to be weakened or powerless. In an interesting paper by Dr. George Johnson in the *Medico-Chirurgical Transactions* of 1875, he shows that, whilst pressure on one recurrent nerve, on the same side, will produce spasmodic action or paralysis, pressure on the trunk of the nerve will cause atrophy or spasmodic action on both sides. Aneurismal disease is perhaps the most frequent cause of this reflex paralysis—reflex in its being produced, not by direct injury of the larynx, but upon it by secondary pressure; not reflex in the sense of an impression being conveyed to the nerve-centre, and then propagated to the affected part. These instances are very different in character from those already described as due to a central cause. Enlarged bronchial glands, cancerous disease, and it is said that pleuritic thickening about tuberculous lungs will induce this change by affecting the recurrent nerve.

A very interesting case of this kind was lately under my care in Guy's Hospital. A strong man, aged 43, an engineer, who, with the exception of rheumatism, had good health till last Christmas; cough then came on, and increased in severity, with dyspnoea, till his admission on February 23rd. His face and neck were swollen and oedematous, the superficial veins of the thorax enlarged; there was complete dulness on the right side of the chest, with absence of respiratory murmur; the signs of thoracic tumour were well marked; the arms became swollen, the dyspnoea increased greatly in severity, and erysipelatous inflammation affected the right arm. The voice was feeble, but was not lost. He died on March 14th. On inspection, a cancerous tumour was found in the anterior mediastinum; it pressed upon the innominate and azygos veins; the right lung was in a sloughing condition; the right pneumogastric was involved, and there was very marked affection of the recurrent on the same side; it was involved in the growth, and, to quote the description of Dr. Fagge, "the larynx was healthy, except the right vocal cord, which was swollen and yellow". The swelling was very decided, and the nutrition was so affected, that suppurative changes had commenced; but there was no suppuration about the muscles of the neck. The patient was too ill to bear the use of the laryngoscope.

Many of the instances of reflex paralysis which are associated with functional disturbance of the uterine organs are closely allied to those which have been referred to as hysterical aphonia.

These varied affections of the larynx may be produced by disease at the origin of the nerve—the pneumogastric—or the disease may be reflex in character, the irritation of one series of nerves affecting other branches, as of the lungs, or of the stomach, or of the larynx; and, lastly, these laryngeal affections may give place to disturbance of the lungs or of the stomach, alternating the one with the other; a patient at one time having aphonia, then spasmodic irritation of the bronchial tubes, or functional disturbance of the digestive organs. In the one case, the attempt should be made to relieve the central mischief; in the second, to lessen the primary source of peripheral disturbance; in the third, to remove the general irritability of the nervous system.

THE CROONIAN LECTURES ON THE PATHOLOGY AND RELATIONS OF ALBUMINURIA.

Delivered at the Royal College of Physicians.

By W. HOWSHIP DICKINSON, M.D. Cantab., F.R.C.P.,

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LECTURE I.—*Concluded.*

2. *Granular Kidney.*—Passing now to the more chronic disorder, that which is recognised as the granular kidney, and occasioned by creeping fibrosis, we have to describe a condition which begins with, and belongs essentially to, the connective and vascular structures which are external to the proper secreting elements. The change begins in disproportioned growth, somewhat of a rank luxuriance, in the fibroid tissue with which the arterial channels are uniformly fringed. Hypertrophy of this part leads to atrophy of all the rest. Creeping along the arterial lines with slow and hesitating steps, involving the organ not all at once, but by little and little, the disease makes itself felt not suddenly, but with so gradual a departure from health, that its date is usually indeterminable, and its existence unsuspected until it has reached an advanced stage. And in other respects, too, in its progress and symptoms, it affords a precise converse to the more acute disorder. One selects the young, the other the old. One is certainly local to the gland; it is in dispute how far the other is general to the system. In one case, dropsy is generally present; in the other, it is present only occasionally. One is characterised by obstructed ducts and suppressed secretion; the other by obstructed blood-vessels and secretion, enhanced at least in its watery part.

To look more narrowly into the organic derangement proper to this more chronic disorder, it may be said to be accurately declared by the superficial loss of level to which the term granulation is applied. This condition is only produced by a drawing in of the surface at certain points by a contractile growth, the distribution of which is determined by that of the blood-vessels around which it begins. Sometimes the granulations are small, regular, scarcely changed in colour, and evidently but mere exaggerations of the minute lobules into which the blood-vessels divide the surface; in other cases, the granulations are large and hemispherical, each conspicuous by the contrast between its white colour and the purple or pink depression by which it is surrounded. The light-coloured striking granulations result from a more active and dangerous form of the disease, one which involves deeper and wider injury to the gland, and more marked symptoms than those which are less noticeable. The contractile process, of which the granulation is the sign, usually involves loss of bulk and weight; but not always so; the atrophy of the glandular structure may be more than made up for by the new fibroid growth and the epithelial accumulation which is apt to occur as the result, apparently, of a catarrhal process set up in the course of the disorder. The essence of the disease, as it first takes substance and shape, is a multiplication of nuclei with subsequent fibrillation in the interstitial tissue. Spreading from the capsule inwards along the arteries, surrounding concentrically the Malpighian bodies, imbedding, compressing, and strangulating the tubes in its path, while others for a time escape only to yield to its later encroachment, the growth overruns the gland little by little, after the manner of the invader who likened his scheme to the eating of an artichoke leaf by leaf. The new growth is widely distinguished from the original fibrous structure of the organ. It consists largely of nuclei, like foetal structure or a fibro-plastic tumour. It occurs in great breadths between the glandular structures, as having thrust itself wedgewise between them, and displaced them by its increasing bulk. It gives evidence of active growth and development, not only by its spreading to the destruction of the older structures, but by the acquirement sometimes of quite a rich supply of capillary blood-vessels. These are sometimes to be traced by the blood they contain, in appropriate distribution through large masses of the intrusive material. That they are components of the new, not remnants of the old structure, is evident by the regularity of their arrangement, by their remote-

ness from any proper glandular structure, and by their delicacy of form; such that, isolated as they are, they could not, had they belonged originally to the organ, have resisted the obliterating agency of the encroaching growth which early and markedly affects the primæval capillary channels.

It is not necessary to do more than touch upon the changes in the gland which ensue upon its occupation by this growing and contracting material. According to its date, its bulky presence or its constricting results are the more apparent. The former is best seen in the rare granular kidney of childhood; the latter with the more common and chronic form which belongs to advancing life. The tubes are variously distorted, parcelled by the fibroid tissue into insular groups, cut up into cysts, and compressed to impenetrable threads.

Of all the structures of the kidney, perhaps the epithelium is that which suffers least. Early in the disease, it is usually absolutely natural; with advancing contraction, cells can usually be found which have become distorted by pressure into a variety of angular and abnormal shapes; but this part of the gland is but passively concerned in the process by which it is destroyed.

The accession of the fibrosis by slow, concealed, and partial approaches has been already described as the rule of the disease. But another outset must be distinctly recognised with an abrupt and definite inflammatory beginning. The disease may be an immediate, though slowly declared, sequence of acute diffuse nephritis.

I have already shown how more or less of interstitial nucleation is a frequent result of renal inflammation, which at first, so far as we can judge by its cause and by its symptoms, was chiefly or wholly catarrhal. The nuclei remain after the catarrh has subsided, and grow into fibre, which may be disposed generally and somewhat abundantly through the organ. This, in the early months of its existence, will simply add to the bulk of the gland without affecting its smoothness; but, as months become years, depressions appear, at first sparsely; these in time multiply and spread themselves with some regularity over the surface, until the superficial granulation is produced which is the sign of chronic fibrosis. The organ simultaneously wasting, the granular and contracting kidney is developed, which, in its morbid anatomy, its pathological concomitants, and its symptoms, is indistinguishable from that of creeping approach and latent origin. The rise of the granular condition in acute inflammation, although not admitting of question, is one of the rarities of disease. The instances I have met with or recognised do not count as a fiftieth of those where the disorder has gradually developed. As a rule, the large white kidney of nephritis remains large, pale, and nearly smooth to the end; while the granular is a slow modification of the condition of health divided from it by no abrupt change or appreciable interval.

With this, as with the more acute renal disturbance which lately occupied our attention, organs other than renal, and structures which are common to every organ, become involved in changes, some of which have been of late taken into question as to whether they are the direct consequences of the disease, or are but collaterally associated with it. Wide differences present themselves in the nature of the lesions by which each affection is accompanied; with the acute, the more important are those which depend upon changes in the blood; while changes in the vessels, rather than in their contents, rule the pathology of the chronic disease. Foremost, therefore, among the disorders with which the granular kidney is associated, must be considered that cardio-vascular alteration which has recently received so much notice. Briefly, this consists of thickening of the walls of the systemic arteries, together with hypertrophy of the left ventricle. As the two are constantly associated, the cardiac may be taken with little error as the indication of the compound change. Taking Dr. Sibson's tables as giving recent and trustworthy information on rather a large scale, I find that in 187 cases in which granular kidneys existed, unaccompanied with any valvular or other disease of the heart which could occasion hypertrophy, the heart was increased in size in 128. Dr. Sibson has been careful to distinguish, in this summary, the instances in which the granular kidneys had shrunk below the normal bulk from those which retained or exceeded it. I have, however, thrown the two kinds together, as not involving any distinction of kind. The bulk apparently depends chiefly upon the amount of tubal obstruction present; usually a secondary and very variable complication. The issue of his statement amounts to this: putting aside cases complicated with valvular disease of the heart, the proportion of cardiac hypertrophy to granular disease of the kidney, as found in the dead-house, is about two to three. This proportion of ventricular thickening, large as it is, falls short of what would be afforded were only those cases of renal disease included which had been attended during life with symptoms. In some cases, it is possible that, though anatomically the kidneys were granular, they were yet fairly efficient,

and possibly the systemic conditions which belong to the developed disorder had not yet appeared. From a clinical point of view, regarding, that is, only those renal conditions which have been recognisable during life, the proportion of cardiac hypertrophy is so large, that this condition may almost be regarded as a necessary part of the systemic disturbance associated with this form of renal disease. Exceptions have been noticed with very advanced renal disease. The once hypertrophied ventricle may apparently lose its bulk with progress of time, as hypertrophic yield to degenerative changes, or in other modes less easy to discern. A young woman was recently brought into St. George's Hospital with cerebral symptoms, which ended fatally, and proved to have been uremic. The kidneys were extreme instances of fibrotic atrophy; the pair weighed less than three ounces and a half. The blood-vessels, as examined in the pia mater, were absolutely natural, and the heart so little hypertrophied as to weigh only twelve ounces. But, practically, there are few exceptions to the rule that, if the system be influenced, however obscurely, by this form of renal disease, hypertrophy of the left ventricle will not be looked for in vain. Enlargement of the heart, therefore, is a most important diagnostic of the granular kidney, or at least of the condition of renal fibrosis.

To pass now to the condition of the arteries found together with the ventricular thickening, we find changes which have been differently interpreted, and which require, in deference to conflicting opinions, a somewhat careful examination. I have examined specimens prepared for the microscope in different ways; some simply in serum or water, others in glycerine, and some which have been exposed to the action of spirit or chromic acid, and preserved in Canada balsam.

I have satisfied myself that the change is by no means a simple one, but that at least three broad modifications can be recognised as contributing in different proportions to the arterial change which is connected with the granular kidney: first, a thickening with alteration of texture of the muscular coat; secondly, a thickening of the outer or fibrous coat; thirdly, a degeneration which chiefly affects the muscular coat, but may in the end supersede every other morbid process, and replace all distinctions of coat by a mere confusion of granules and fat. To me it appears—and I speak with caution, for so compound is the usual result, and so variously mixed are its pathological ingredients, that specimens may be found which, taken by themselves, would seem to warrant any of the views which have been propounded as to the nature of the change—that the steps of the process take place in some such order as the following. First, there is a thickening of the muscular coat, with degeneration or partial destruction of its nuclei, and the acquirement of a coarseness of fibre foreign to its normal appearance. This is succeeded or accompanied by more or less increase of the outer or fibrous coat, which sometimes, especially in cases of long standing, may even be more striking than the hypertrophy of the muscular. With these proceed alterations of a degenerative kind, fatty and semi-crystalline, which are chiefly of muscular origin, and are the chief agents in causing the brittleness of vessel to which the hæmorrhagic proclivities of the disease are largely due.

In the large white or nephritic kidney, where, from the more acute character of the disease, the changes are correspondingly earlier, the muscular coat is chiefly affected, and that rather by hypertrophy than degeneration. With the granular kidney, the thickening is more decided, and degenerative changes are more advanced. A semi-crystalline layer is often distinctly seen lining its inner surface.

With regard to the outer or fibrous coat, the action of reagents has been drawn into question, as possibly giving rise, by a delusive *post mortem* imbibition, to the appearance of thickening which has been described. But such doubts may be fairly quieted by the consideration that in all instances—certainly in all those upon which these statements are based—the diseased vessels have been compared with healthy ones subjected to precisely the same treatment; so that any differences between the two necessarily depend upon the vessels themselves, not upon the circumstances which are common to both. Glycerine undoubtedly expands the outer coat, and displays its structure to the fullest advantage; yet its increase in association with the granular kidney can be clearly seen in Canada balsam, or after any mode of preparation by which the structures can be displayed.

To complete the catalogue of changes to which the arterioles in this disease are subjected, it must be added that they often, particularly when much degenerate, become extraordinarily irregular in outline, variously bulged, and sometimes corrugated and gnarled like the limbs of a time-honoured oak. Particles of hæmatine are often found about them, or even scattered thickly and widely in their neighbourhood.

These arterial and cardiac changes are usually accompanied during life with an increase of arterial tension, an unvarying fulness of the arterial system, in degree and continuance surpassing what is found in

connection with any other renal condition, or with any other morbid state with which we are acquainted. This is evinced by the hardness of the pulse, as evident to the touch, the absence of diastolic collapse giving to the vessel a cord-like persistence; by the force with which the sphygmograph must be brought to bear upon it before a graphic tracing can be obtained; by the abnormal pressure required to obstruct the vessel, as measured by the sphygmometer of Dr. Handfield Jones; and by the alteration in the sounds of the heart, especially by the accentuation and frequent reduplication of the second sound: evidences, as they have been interpreted, the first of the abnormal force of the diastolic recoil, owing to the repletion of the aortic system; the second, of the slowness with which, from the same cause, the left ventricle empties itself, so that the sound which succeeds upon the completion of the left systole is produced not synchronously with that which follows upon the right, but appreciably later.

It is not my purpose, however, to dwell upon this portion of the subject, since it has been brought before the College recently, and with an ability and completeness to which I could not hope to attain.

THE GOULSTONIAN LECTURES

ON

THE NATURAL HISTORY OF PULMONARY CONSUMPTION.

Delivered at the Royal College of Physicians, London.

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LECTURE I.—*Concluded.*

LET us turn for an instant to the comparative pathology of these modern days. I have here a series of diagrams, copied, by his permission, from the second part of Dr. Klein's work. On the table is a drawing of the naked-eye appearances of the lung in a case of acute tuberculosis in a child, together with microscopical preparations of a case of the same kind, made for me by Mr. Lithgow, our assistant-curator at St. Mary's; the latter show perivascular growth, with some lobular collapse, and but very slight catarrhal product. [A description of the diagrams, etc., followed here.]

In these two diagrams (Figs. XXIV and XXVI), it is possible to see in the lung of the guinea-pig, as well as in that of the child, certain histological products which it has been attempted, not for the first time in the history of "tubercle", to set up as the specific elements of that growth. Happily these giant-cells will soon be relegated—if they be not even now relegated—to the list of ordinary epithelial cells, the limbo in which rest already the pathognomonic tubercle-corpuscle and cancer-cell. As my friend Dr. Klein informs me, and as I am quite prepared to believe, the giant-cell is but an epithelial cell growing under certain conditions. But

"Each month is various to present
The world with some development",

and the specific element refuses still to be unrecognised; this time it appears under the form of the particular cells drawn and described by Rindfleisch in Ziemssen's *Handbuch des Respirations-Apparates* (vol. ii, p. 161. Leipzig, 1874). I am perfectly prepared to see these cells, also, sink to the same position as the Riesenzellen. I confess that I do not perceive why, in the pursuit of a supposed scientific truth, the same common-sense which men use, or ought to use, in the ordinary affairs of life, should not exert itself; and when through the long list of specific structures, started by one author only to be hunted down by another, we find always the same untimely death, I believe that the very best thing we can do is not to join in the shadowy chase, but to abjure, as something that oppresses us, with no fault of our own, this "specific" incubus.

With the results shown in those diagrams and described in his work by Professor Klein, the researches of Dr. Burdon Sanderson and Dr. Wilson Fox in the main agree. But the latter writer goes much further, and states that he finds "in the lungs of patients dying of phthisis almost identically the same changes as those found in the lungs of children dying of acute tuberculosis"—a sentence to which I cannot altogether give my adhesion. There are cases, no doubt—cases of which I hope to speak more at length hereafter—in which the

physical signs and symptoms of lung-disorganisation are followed by death, with all the symptoms of this "acute tuberculosis". But there are also cases, of not infrequent occurrence in the adult, of this same "acute tuberculosis" with almost the same series of clinical symptoms, and the same pathological changes, as far as I am able to make them out, as those found in artificial tuberculosis, and in the general tuberculosis of the child. The only case of the kind which I remember to have come almost entirely under my own observation was that of a young man, about twenty-one years old. In him there were no abnormal signs on percussion; the respiratory sounds were everywhere coarse. There was increase in temperature and pulse-rate; his chest and abdomen were covered with sudamina; he was admitted into hospital as a case of probable acute tuberculosis, or doubtful typhoid; within a short time, he died with symptoms of tubercular meningitis, and his body presented the expected changes.

And it was, I think, from these comparatively rare cases in the adult that observers and writers argued that "ista tubercula (seu tumores)"—I use the words of Morton—underwent a further, and that a cheesy change, resulting—not in these cases, which, if the argument be true, must be said to die before such changes can take place—but in cases the clinical symptoms of which are entirely different, in a larger or smaller destruction of pulmonary tissue. That I am not, Quixote-like, opposing some image of my own brain, is, I think, shown by the fact that writers of the present day still class these cases of "acute tuberculosis" with "pulmonary consumption"; though it is but fair to state that Dr. Douglas Powell has, in his small work, drawn the distinction which others, though by no means all, have failed to make. I need not travel far for an illustration. In a very valuable, because very true and practical, though it may be a very simple, paper on the different forms of pulmonary consumption by my colleague Dr. Peacock—a paper published originally in *St. Thomas's Hospital Reports* for 1870—are given two cases, "selected from numerous instances of the same kind"; together with a third, which differs from them in several respects; all three classed by the writer under the name "acute constitutional phthisis". Dr. Peacock's first case is that of a young lady, aged 22, who died on the twentieth day of her illness of apnoea. Four days before death, "the respiratory sounds were found everywhere harsh..... but there was no rhonchus and no marked deficiency of the resonance on percussion in any part of the chest." These signs were slightly modified the day before death. On *post mortem* examination, the body "was thin, but not emaciated". There were old pleuritic adhesions over a large portion of the left lung; the right lung was collapsed. "Both lungs were copiously infiltrated throughout their whole substance with miliary tubercles, having an average diameter of from half a line to a line, and uniformly of a yellowish-white colour..... In no part had any of them undergone softening." Nothing is said about the head or abdominal organs. The second case is also that of a young woman, aged 24, who died after an illness lasting over three weeks of capillary bronchitis; here was the same absence of phenomena on percussion and auscultation; but little emaciation of the body, a chest sufficiently broad and expanded; both lungs contained a copious deposit of small, round, yellowish-coloured and semitransparent-looking bodies, which in no part had softened; the bronchial glands were softened to a brown-coloured pulp; the abdominal viscera were free from tubercle. Dr. Metzquer, in a *Clinical Study of Galloping Consumption*, published in Paris in 1874—and I may mention, in passing, that, to the majority of French writers, galloping phthisis is synonymous with acute tuberculosis—Dr. Metzquer gives two similar cases. Case I is that of a female aged 28-30, free from history of hereditary phthisis, and always well till she was taken one day with pains in the head and back, intense fever, rigors, and ringing in the ears. There were no signs on percussion or auscultation, though a few days later there were rhonchis and *râles* in both lungs, and streaks of blood in the sputa. Death occurred a month after the commencement of the illness, and there were "tubercles in all organs". The second case, that of a man aged 43, is precisely similar: the same absence of hereditary phthisis and physical signs on the part of the lungs; the same general tuberculosis without softening. In both these cases, there was some diarrhoea and slight jaundice before death. It would be easy to multiply from modern literature cases of this kind, but I prefer to turn back again to the commencement of the century. I quite agree with M. Thaon that Bayle's cases numbered 4, 14, and 15, named by him cases of granular phthisis, do not answer (see Andral, *op. cit.*, iv, 10), either clinically or pathologically, to this "acute tuberculosis"; but, in spite of the distinction Bayle makes between the *miliary tubercles* found in the lungs of his first case, a woman aged 20, who seems to have exhibited as her chief symptoms those of "tubercular peritonitis", and the *miliary granulations* in the lungs of his second case, a postillion aged 24, who died, as the four cases I have just quoted from Drs. Peacock and Metzquer did, from