LECTURES ON THE PATHOLOGY, DIAGNOSIS, AND TREAT- MENT OF BRIGHT'S DISEASE.

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LECTURE III.—CHRONIC BRIGHT'S DISEASE.

Small Red Granular Kidney.—Synonyms.—*Outward Appearance of the Kidney in different Stages.*—*General History of the Disease.*—*Chemical and Microscopical Characters of the Urine.*—*Microscopic Appearances in the Kidney.*—*The Structural Changes are essentially tubular and intratubular.*—*Changes in the Blood-vessels of the Kidney.*—*Physiological Explanation of the Structural Changes in the Kidney and of the Condition of the Urine.*

In my last lecture I gave you some account of acute Bright's disease, and I now proceed to discuss the subject of chronic Bright's disease. Cases of chronic Bright's disease arrange themselves, anatomically and clinically, in two very distinct classes. In one class of cases, the kidney is found small, red, and granular; in the other class, on the contrary, the kidney is large, pale, and usually smooth on the surface. The clinical history of the two classes of cases is as distinct as are their anatomical characters. For various reasons, it will be more convenient to take first in order those cases which are associated with the small red granular kidney. In the Nomenclature of the Royal College of Physicians, the disease is designated "granular kidney", with the synonyms "contracted granular kidney", "chronic desquamative nephritis", "gouty kidney".

Outward Appearance of the Kidney in different Stages.—I place before you drawings representing kidneys in different stages of degeneration. At no period of the disease is there enlargement of the kidney, but from the commencement a process of wasting occurs. In the early stage, when death has occurred from some other disease, the capsule is found adhering firmly to the surface of the gland, so that it is difficult to tear it off without bringing away some of the adherent glandular tissue. The fine lobular markings are less distinct than in the normal state, and the surface of the kidney is slightly uneven and granular. As the disease advances, there is progressive wasting of the glandular portion of the kidney, with granular unevenness of the surface and diminution of the thickness of the cortex; so that by degrees the bases of the medullary cones approach nearer to the surface of the gland. In extreme cases, the kidney may be reduced to one-half or even one-third of its normal size and weight. The cortical secreting portion of the gland is evidently the part chiefly implicated, while the medullary cones are nearly intact. The contracted kidney is somewhat firmer and tougher than natural. In all stages of the disease, one or more, sometimes numerous, serous cysts may be seen projecting from the surface, and varying in size from a pin's head to a pea, but sometimes as large as a filbert, or even larger. Even in the most advanced stages of atrophy, the organ retains more or less of its normal red colour and its vascularity; hence it is called the *red granular kidney*, to distinguish it from certain cases of chronic Bright's disease to be referred to hereafter, in which the kidneys are granular, but white and anæmic.

General History of the Disease.—Some general facts relating to the disease it may be well to point out now. The disease is essentially chronic from the commencement, and rarely, if ever, a sequel of an acute attack. Its commencement, therefore, is, as a rule, insidious, and in its early stages it is often unsuspected and latent. It is a comparatively rare disease in early life, not uncommon between the ages of twenty and thirty; but the majority of cases occur in persons at and beyond middle age. It is often associated with the gouty diathesis, as one of its synonyms indicates; and it is of common occurrence in persons who eat and drink to excess, or who, not being intemperate in food or drink, suffer from certain forms of dyspepsia, without the complication of gouty paroxysms. In some cases, the disease probably results from habitual exposure to cold and wet, and consequent suppression of the cutaneous secretion. There is reason to believe that chronic poisoning by lead is, at any rate, a concurring cause of the disease amongst painters and others who are exposed to the influence of this pernicious metal. Dr. Garrod was the first to direct attention to the influence of lead in the

causation of gout; and Dr. Dickinson states that, out of forty-two men exposed to lead-poisoning who had died in St. George's Hospital, twenty-six had granular degeneration of the kidneys, which in most cases was so advanced as to have caused death (*On the Pathology and Treatment of Albuminuria*). Allowing, as we must, that the lead had great influence, it is probable that habits of intemperance and other causes may have co-operated with the lead. Granular kidney is occasionally, though rarely, found as a sequence of the albuminuria which is associated with pregnancy. I have seen one well marked instance of this. The atrophy with granulation which results from passive congestion of the kidney consequent on valvular disease of the heart or emphysema with chronic bronchitis, has a different pathological history; and I shall refer to it on a future occasion.

During the progress of the disease which results in the contracted granular kidney, dropsy rarely forms a prominent symptom, and in the majority of cases it is entirely absent. The disease is often associated with hypertrophy of the left ventricle of the heart, even when there is no valvular defect or disease of the walls of the larger arteries to explain the cardiac hypertrophy. In a large proportion of cases, the immediate cause of death is uræmia or cerebral hæmorrhage.

Now, in the course of these lectures, I shall as much as possible avoid all controversial topics; but, in proceeding to give you what I believe to be the true account of the minute anatomy and pathology of this disease, I am bound to tell you that I dissent from the opinions of some pathologists for whom I entertain great respect, but not sufficient to induce me to follow them into what I believe to be an erroneous reading and interpretation of facts. Virchow, in his *Cellular Pathology*, states that there are three anatomical elements in the kidney—namely, tubes, vessels, and interstitial tissue; and, in accordance with this, there are three forms of Bright's disease—what he calls parenchymatous nephritis, having its seat in the tubes; amyloid degeneration in the blood-vessels; and interstitial nephritis, consisting essentially, as he believes, in thickening of intertubular tissue and consequent atrophy and granular contraction of the kidney. Virchow admits that two and sometimes all three of his forms of disease may coexist in the same kidney; and I maintain that in every case of Bright's disease all the tissues are implicated; the various forms of disease depending, not upon the implication of different anatomical elements in the morbid process, but upon the varying nature of the structural changes which these elements undergo in different classes of cases. I will endeavour to make this clear as I proceed. My doctrine with regard to the minute anatomy and pathology of the granular kidney is, that it consists primarily and essentially in a disintegration and destruction of the gland-cells which line the convoluted tubes, the *débris* of the gland-cells appearing in the urine as granular tube-casts; that the destruction of the gland-cells induces atrophy and contraction of the tubes; that this shrinking of the tubes, with some thickening of their membranous walls and of the Malpighian capsules, gives a delusive appearance of interstitial or intertubular formation of fibrous tissue; and that thickening of the walls of the arteries, the nature of which I shall presently describe, forms one of the most constant and conspicuous features of the disease; although this arterial change is entirely ignored by Virchow and his followers, who erroneously assume that the so-called amyloid or waxy degeneration is the only form of Bright's disease constantly and essentially associated with thickening of the blood-vessels.

Chemical and Microscopical Characters of the Urine.—You will find that the minute structural changes in the contracted kidney are rendered easily intelligible if you study them in connexion with the clinical history of the disease, and in particular with the chemical and microscopical characters of the urine. I have told you that the disease, although not exclusively of gouty origin, is often associated with chronic gout. Examine the urine of a man who has had repeated attacks of gout, and you will not unfrequently find in it the earliest indications of incipient renal degeneration. The urine may be of normal colour and specific gravity, and without a trace of albumen; but, after standing for a few hours in a conical glass, it deposits a light cloud, which, on microscopic examination, is found to consist of scattered granular *débris* and of tube-casts such as are represented in Fig. 14. These casts contain epithelial cells in various grades of disintegration, and hence arises their "granular" appearance. Every granular cast is not of necessity composed of disintegrated epithelium. Blood-corpuscles may become disintegrated within the uriniferous tubes, and appear in the urine as granular blood-casts, distinguished from granular epithelial casts by their reddish-brown colour, and often by containing some entire blood-corpuscles; so, disintegrated hyaline casts may assume a granular appearance; but by a comparison with other casts associated with them, and by noting the various grades of change, we trace them to their true source. The presence of granular epithelial casts and of scattered epithelial *débris* is evidence that a process of epithelial de-

squamation and disintegration has commenced in the kidney. In the earlier stages of the renal disease, the granular casts are found only during or immediately after a gouty paroxysm, and, as I have already

Fig. 14.—Granular Casts, composed of more or less completely Disintegrated Epithelium and Fibrine.— $\times 200$.

said, unassociated with albuminuria. In the intervals between the attacks of gout, no tube-casts are visible. At a later stage, granular casts and epithelial *débris* are always present in a greater or less amount; and the urine becomes albuminous, at first only during a fit of gout, the tube-casts also being more abundant during the paroxysm. At a still later period, tube-casts and albumen are more or less constantly present, though both may be absent even in the most advanced stages of this form of disease. Not only is the particular form of renal disease indicated by the microscopic appearance of the urinary sediment, but the number of granular tube-casts and the amount of epithelial *débris* indicate the rate at which the disease is progressing. The more copious the sediment, the more rapid is the destruction of the gland-cells, and the consequent atrophy of the kidney. In the more advanced stages of the disease, large hyaline casts are often found associated with the granular casts. (Fig. 15.) As the disease makes progress, the urine

Fig. 15.—*a a*. Granular Casts. *b b*. Large Hyaline Casts.— $\times 200$.

undergoes remarkable physical changes. The quantity secreted is usually in excess of the normal amount; and with the increase of quantity there is commonly associated a loss of the natural colour and a diminution of the specific gravity, which, usually as low as 1010 or 1012, sometimes falls to 1005. The low specific gravity indicates a relative decrease of the normal solid constituents, especially of urea, uric acid, and extractive matters. In one of Dr. Christison's cases, the total solids discharged were reduced to one-fifth, and in another nearly to

one-twelfth, of the healthy average. This defective discharge of solids is partly explained by the rest in bed, the scanty diet, and the general anæmia. The amount of albumen varies considerably. Absent or scanty in the early stage, it may be rather copious in the middle periods, and again scanty or even entirely absent in the stage of extreme degeneration of the kidney.

Microscopic Appearances in the Kidney.—The kidneys should be examined as soon as possible after death, and before the appearance of their tissues has become changed by any antiseptic or hardening process. Thin sections of the cortex, made with a Valentin's knife, may be placed in a solution of common salt and water of specific gravity 1030, and then examined with a magnifying power of not less than 200 diameters. Dilute acetic acid brings out some of the appearances very distinctly. The chief changes will be found in the convoluted tubes, in the arteries, and in the Malpighian capsules and capillaries. In some tubes, the gland-cells have their normal appearance, or they are opaque and granular, with a clear central canal (see *ante*, Fig. 12). Other tubes are filled and rendered opaque with desquamated epithelium more or less disintegrated (see *ante*, Fig. 10). Others, again, present the characteristic appearance represented in Fig. 16. Their epithelial

Fig. 16.—Transverse Sections of Tubes, containing only Granular *Débris* of Epithelium held in position by Coagulated Fibrine. At one end of the section, the contents of the tubes have been washed away, and the sections of the basement-membrane form three empty rings.— $\times 200$.

lining has become disintegrated and removed, appearing in the urine in the form of the granular casts before described (Figs. 14 and 15). A few granular particles of epithelium only remain, and these appear to be held together by fibrinous coagula. The transverse sections of tubes in this condition have somewhat the appearance of oval or globular cysts, and many years ago they were described as microscopic cells by a very able observer (see Mr. Simon's paper on Subacute Inflammation of the Kidney, *Med.-Chir. Trans.*, vol. xxx). When, in the same section, some tubes appear transversely divided, while others present themselves lengthwise, as in Fig. 17, all having the same

Fig. 17.—Tubes more or less completely denuded of Epithelium. Some transversely divided and cyst-like; others seen lengthwise.— $\times 200$.

general structure and contents, it is easily seen that the cyst-like appearance is given by transverse sections of partially or completely denuded tubes.

The number of tubes thus denuded of their epithelial lining varies much in different cases. In some kidneys, which to the naked eye present comparatively little deviation from the normal state, the destruction of gland-cells is found to have been very extensive. Other tubes are found, as regards their appearance and contents, in the same condition as those just now described, but apparently shrunken and atro-

phied, with wide interspaces between them—the interspaces being occupied by the remains of other atrophied tubes and capillaries (see Fig. 18). Atrophy of the tubes appears to be the usual result of

Fig. 18.—Tubes in process of Atrophy and Contraction after the Destruction of their Epithelial Lining, a few granular particles only remaining within them.— $\times 200$.

the destruction and removal of their gland-cells. But the opposite condition of dilatation is found in some of the tubes, which may be seen often as large as Malpighian bodies, and even larger (Fig. 19); and

Fig. 19.—Transverse Sections of Dilated Tubes with Thickened Walls. In some sections, the open mouth of the divided tube is seen—one section of a denuded tube of normal size.— $\times 200$.

there can be no doubt that these dilating tubes at length form the cysts which are visible by the naked eye.

There is yet another appearance of the tubes in the granular kidney, which probably has a close relation with the formation of dilated tubes and cysts. This appearance is represented in Fig. 20.

Fig. 20.—Sections of Tubes, in which a layer of Transparent Cells, each with a single Nucleus, has taken the place of the Normal Epithelial Lining.— $\times 200$.

The tubes are lined by layers of delicate clear-walled cells of a more or less rounded form, and each having a single nucleus. Some tubes in this condition may be found in every granular kidney, but their

number varies considerably. In most contracted kidneys, these rounded tubes are relatively few in number; in others, they are very numerous; and I believe there is a special relationship between these tubes and the cysts with which they are usually associated. It can scarcely be doubted that renal cysts are dilated tubes; and it is probable that the tubes are dilated by the accumulation of a watery fluid secreted by these delicate cells, which I have described. This at any rate is certain, that the contracted granular kidney is the only form of Bright's disease with which, as a rule, cysts are associated; and it is in these kidneys only that we find tubes lined by the transparent cells in question. (See a remarkable case recorded by Dr. Conway Evans, *Pathological Transactions*, vol. v, p. 183. I examined the kidneys in that case, and can confirm the accuracy of Dr. Evans's description.) It is probable that the dilatation of the tubes, by their accumulated liquid contents, and their conversion into aqueous cysts, is favoured by the obstruction of their outlets through an accumulation and impaction of epithelial and fibrinous debris.

I have yet to mention that here and there in the cortex of the kidney a tube may be seen without gland-cells, and completely filled with disorganised fibrine, which, if it had escaped during life, would have appeared in the urine as a large hyaline cast (see Fig. 15). Sometimes, though rarely in this form of disease, a tube is found injected with blood which has escaped from ruptured Malpighian capillaries. In some few of the tubes the contents have undergone a fatty transformation; and oil-globules may be seen either contained within cells, or free and irregularly accumulated within the tubes.

The basement-membrane of the tubes usually appears to be somewhat thickened; and this thickening, together with the wide spaces between the tubes occupied by the atrophied remains of shrunken tubes and intertubular capillaries (see Fig. 18), has given rise to the doctrine of an excess or of a new formation of connective tissue between the tubes. Now, as I told you in my first lecture, there is in the normal condition no connective tissue between the convoluted tubes. In this statement I am in accord with Ludwig, who says, "No fibrillated connective tissue exists between the tortuous portions of the urinary tubules" (see Stricker's *Human and Comparative Histology*, New Sydenham Society's Translations, vol. ii, p. 106); and I show you thin sections of beautifully injected healthy kidneys, in which you can see that the intertubular capillaries are in immediate contact with the outer surface of the basement membrane, and that if anything intervenes, it is quite homogeneous, gelatinous, and structureless. In fact, the basement-membrane of the tubes constitutes the true and only connective tissue between the intertubular capillaries and the intratubular epithelium; therefore, whatever increase of this tissue there is—and it is much less in the granular kidney than in some large white or amyloid kidneys—is not an intertubular, but a tubular, thickening. It is obvious, however, that if we take into account the wasted tubes which remain in a shrunken condition after the destruction of their epithelial contents, there is in the granular kidney a relative excess of basement-membrane connective tissue.

There is often an appearance of fibrous connective tissue round the Malpighian bodies. I am not prepared to say that no connective tissue is ever formed external to and apart from the fibrous capsule of the Malpighian body; but I am convinced that, when the capsule is somewhat thickened, as it often is in the granular kidney, and then thrown into folds, the fibrous appearance which it presents, as different depths of the globular capsule are being focused into view, may very readily be mistaken for fibrous connective tissue outside and surrounding the capsule (see fig. 21).

Fig. 21.—Malpighian Body—the Capsule thickened and having a Fibrous Appearance. The capillaries thickened and opaque; the nuclei visible in their walls.— $\times 200$.

In consequence of wasting and contraction of the tubes, some of the Malpighian bodies are brought nearer together, and three or four may sometimes be seen almost in contact with each other. Sections of dilated tubes, such as are represented in fig. 19, may easily be mistaken for Malpighian bodies; but in the sections of tubes the open mouth of

the cut tube may often be seen, and in the Malpighian bodies the capillaries, with the nuclei in their walls, are characteristic.

Now, to recapitulate; we have found the following pathological appearances in the tubes: the epithelium opaque and granular, in a state of cloudy swelling; the tubes crowded and opaque, with degenerated and disintegrated epithelium; some tubes deprived of their epithelium; some contracted; others dilated in various degrees; some lined by transparent uninucleated cells; others filled with unorganised fibrine, rarely with blood, or with oil; lastly, the basement membrane and the Malpighian capsules thickened, this thickening being often more apparent than real. All the changes which I have described, and which you may see in the specimens which I have placed under the microscopes on the table, are essentially tubular and intratubular. You see, then, with how little reason the contracted kidney is spoken of as the result of an essentially intertubular disease.

Changes in the Blood-vessels of the Kidney.—Amongst the most constant and interesting anatomical changes are those which occur in the minute arteries. The walls of the minute renal arteries present in the normal state two layers of fibres, an inner longitudinal and an outer circular layer (see Fig. 22). In the advanced stages of contracted gra-

Fig. 22.—Normal Artery from the Kidney.— $\times 200$.

nular kidney, both these layers are much hypertrophied. The two layers remain quite distinct, and sharply defined. There is an excess of normal muscular tissue with no appearance of structural change or degeneration (see Fig. 23). The hypertrophy is usually most conspicuous

Fig. 23.—Artery, with Hypertrophied Muscular Walls, from the Kidney. An inner longitudinal and an outer circular layer of fibres, of about equal thickness. The canal is injected.— $\times 200$.

in the smallest arterial branches, and a comparison of the afferent artery of the Malpighian body in a healthy kidney with one from a small granular kidney will often show that the arterial walls in the latter are twice or even thrice the normal thickness.

A transverse section of an artery of larger size, such as is represented in Fig. 24, shows the projecting cut ends of the longitudinal layer of

Fig. 24.—Transverse Section of a Renal Artery, with Hypertrophied Muscular Walls. The inner longitudinal and the outer circular layer of fibres clearly seen.— $\times 200$.

hypertrophied muscular fibres surrounded by the outer circular layer. You may see for yourselves that, in the specimens under the microscopes, the appearances described are as distinctly visible as in the diagram.

I discovered this remarkable hypertrophy of the muscular walls of the renal arteries more than twenty years ago, and published the fact in the *Medico-Chirurgical Transactions*, vol. xxxiii. Since that time, I have found arterial hypertrophy in every contracted granular kidney that I have examined. It is not present in the earlier stages of the disease, but it comes on gradually, and proceeds, *pari passu*, with the structural changes within the uriniferous tubes. This thickening of the arterial walls from overgrowth of their normal muscular tissue is quite distinct from that structural change which I shall hereafter show you, and describe as the waxy or lardaceous degeneration of the blood-vessels.

The walls of the Malpighian capillaries are usually thickened and opaque, their surface sometimes smooth and wax-like, more commonly finely granular (fig. 21). The thickening of the capillary walls, together with that of the Malpighian capsule, tends to conceal the blood within the Malpighian capillaries, and gives the Malpighian bodies a dull grey appearance. The intertubular capillaries, and the veins into which they empty themselves, present no appearance of thickening.

Physiological Explanation of the Structural Changes in the Kidney.—In a paper published long since (*Med.-Chir. Trans.*, vol. xxx), I designated the disease which I am now describing *chronic desquamative nephritis*. The term “chronic desquamative disease” is, I now think, preferable, since it implies no theory as to the inflammatory nature of the disease. The primary and essential structural changes consist in a desquamation, disintegration, and removal of the renal gland-cells; and the pathological process admits of the same physiological explanation as that which, in my last lecture, I gave of the acute desquamative disease. The changes in the glandular epithelium are the result of a modified cell-nutrition consequent on a morbid condition of blood associated with gout or with one or other of those derangements of the general health to which I just now referred as the usual antecedents of this form of renal degeneration. Gland-cells secreting abnormal products are themselves liable to become abnormal; and, when the process is long continued, the cells are apt to undergo decay and destruction. That appears to me to be the explanation of the disintegration and destruction of the renal epithelium. The wasting and contraction of the tubes, with some real and more apparent thickening of their basement-membrane, are results of the destructive changes in the gland-cells. I cannot explain the replacement of the glandular epithelium by the delicate cells which are found lining some of the tubes, and which, apparently, are intimately associated with the conversion of the tubes into serous cysts.

It seems probable that the copious flow of urine of low specific gravity is a result of the secretion of a watery fluid by the tubes deprived of their normal epithelium, and either denuded or lined by the thin-walled cells before described. This explanation is in accordance with the fact that the urine is usually more copious in the intermediate stages of the disease, when the denuded tubes are most numerous, than in the early stage, when the structural changes in the tubes are less extensive, or in the later stages, when many of the tubes are atrophied and obliterated.

The hypertrophy of the muscular walls of the minute renal arteries is best explained by reference to the analogous phenomena of apnoea. A ligature on the trachea of a dog destroys life in a few minutes; and the immediate cause of death is the arrest of the circulation through the lungs. The chest being opened immediately after death, the left cavities of the heart are found nearly empty; the right cavities, the pulmonary artery to its terminal branches, and the systemic veins, are much distended; the pulmonary capillaries are nearly bloodless; and the lungs consequently collapse to an extreme degree as soon as the chest is opened. Now, what is the explanation of this remarkable and abrupt stoppage of the circulation? The theory is that, when the respiratory changes are suspended by the exclusion of air from the lungs, the minute pulmonary arteries, under the influence of the vaso-motor nerves, so contract as to arrest the flow of blood into the capillaries. It is probable that an impression is conveyed by incident nerves from the pulmonary capillaries to the vaso-motor nerve-centre, whence it is reflected through vaso-motor nerves to the walls of the minute pulmonary arteries, which are thereby excited to contract. Similar phenomena may be observed during a fit of spasmodic asthma. Bronchial spasm narrows the tubes and lessens the supply of air to the lungs; then contraction of the minute pulmonary arteries in a corresponding degree checks the circulation. The skin becomes cold and blue, the pulse small and feeble, and the patient is apparently moribund. When the bronchial spasm relaxes, and the air again gets ready access to the pulmonary vesicles, the arterial contraction ceases, and respiration and circulation together again become free.

You will find the proximate cause of death from apnoea very clearly explained by Sir Thomas Watson in the last edition of his lecture on *different modes of dying*. Now phenomena precisely analogous occur in the kidney and probably in all glandular structures. There is the same intimate relation and interdependence between circulation and secretion as there is between circulation and respiration; in fact, the lung may be looked upon as a gas-secreting gland. When the secreting tissue of the kidney is partially destroyed, the gland is reduced to the condition of a lung receiving only a scanty supply of air, the working power of the gland is lessened, and it requires less blood. The minute renal arteries by their contractile power now regulate the blood-supply in accordance with the diminished requirements of the gland. This regulating contraction continues and increases, month after

month, year after year; and the physiological result of this persistent overaction of the minute renal arteries is that their muscular walls become hypertrophied. I will show you, hereafter, that a similar hypertrophy of the renal arteries occurs in other forms of chronic Bright's disease, but it is most constant and most conspicuous in the contracted granular kidney which we are now considering. The comparatively small amount of albumen in the urine, and its occasional absence in cases of contracted kidney, may be explained by the fact that, while there is but little compression of the intertubular capillaries by swollen tubes, and consequently but little passive engorgement of the Malpighian capillaries, the hypertrophied renal arteries, by their powerful contraction, prevent a too forcible influx of blood. Hence, too, it happens, that hæmorrhage into the tubes, which is so common in acute Bright's disease, rarely occurs in this chronic form of the malady.

[To be continued.]

EXTRACT OF AN ADDRESS

ON

UNIVERSITIES IN THEIR RELATION TO PROFESSIONAL EDUCATION.

*Delivered before the St. Andrew's Graduates' Association,
Saturday, February 8th, 1873.*

By LYON PLAYFAIR, LL.D.,

Member of Parliament for the Universities of Edinburgh and St. Andrew's.

IN medicine special schools have grown numerous, because Oxford and Cambridge neglected their duties as liberalisers and cultivators of professions. Though rivers will not flow back to the sources whence they came, yet, in the future, the sources may supply healthier waters to the streams than they have done in past times. So our English Universities, though they have lost their hold on the medical profession, may at least adjust a preparatory curriculum to suit it, and thus secure to medical students a liberal culture bearing on their future life before they begin their purely professional training.

Universities should understand that, if they desire society to uphold their ancient academic rights, they must show themselves willing to extend modern obligations to society.

I do not presume to give detailed schemes for the construction of the various academic roads which might lead through the faculty of arts to the professional faculties. For each of these would be the best adviser how the several roads should be constructed. All I venture to press is, that the roads should be sufficiently numerous not only to lead to recognised academic professions, but also to the great occupations of manufacturing and mercantile industries, which above all require to be mellowed by liberal culture.

As I have now the honour of addressing an audience chiefly composed of the medical profession, allow me to explain the attitude of hesitation, if not of opposition, which the Scotch universities have taken up in reference to a general and popular cry for a "one-portal" system of examination. This demand has risen from a just discontent with the laxity of examination on the part of some of the nineteen licensing bodies in the United Kingdom. It is contended that a single State examination would give better security for the qualifications of medical men than the separate licensing systems. No one can dispute the right of the State to fix its own standard of qualifications for licenses involving civil rights and affecting the health of its citizens. That right, as I have shown, was exercised as early as the thirteenth century, and it now receives full expression in the *Staats Examen* of Germany. But that, both in its former and present state, is a very different thing from the one-portal system which has been proposed for this country. In Germany the State examination was always supplementary to the academic curriculum. It was simply a state door through which the university trained student had to pass before he assumed civil rights of practice. But the one-portal system proposed for this country might be anterior to university or corporate graduation, so that the State license would be, instead of a supplement, a substitute for academic graduation. Any single licensing system must aim at a minimum and not at a maximum standard of qualifications. Suppose it aimed at a maximum, like the University of London, what would follow? Necessarily the ranks of the profession must remain empty. For London University, with its maximum standard, can secure annually only some thirty medical graduates from the whole of the kingdom and the colonies; while the medical register annually requires seven hundred additions. Under such a system the demand for medical men could not be supplied, and the public would suffer. Hence, clearly,

the one-portal system can only prevent a man from passing in under a minimum standard, but it cannot ensure higher qualifications. Yet such a minimum plan of licensing would govern the whole medical schools of the country, as surely as the main motive wheel in a factory governs the motions of a thousand bobbins.

Under such circumstances, the ornamental degrees of universities and corporations would have no more influence on medical education as a whole, than the brightly polished brass-work on the standard of an engine has upon its motive power. The qualifications of medical men would then be exactly what the minimum involved, and, except rarely, would be no higher. For all experience teaches us that the great bulk of students, with a compulsory examination before them, concentrate their vision on that alone, and refuse to look beyond it; so that teaching schools and universities must then teach down to this minimum, and not teach up to their maximum, if they are to preserve their students from mere crammers. It is this that has rendered uniform standards of examination so fatal to intellectual development, in every country where they have been tried. It is this that has made Germany abandon its old centralised system of State examination; for it is now carried on at the seat of each university, chiefly by the professors and partly by assessors appointed by the State. Even in this modified form it has much injured medical graduation, because students work for the essential license, and neglect the mere academic honours. Germany is the typical country of universities, for it counts twenty-four of them, and these contain 20,000 matriculated students. But its principle is to give to each university a separate autonomy and the utmost liberty of teaching and examination. It preserves for the State a right of proof that these functions have been discharged efficiently when civil rights are conferred; but it carefully makes the exercise of this right a mere supplement to a well-ordered university curriculum. This is well illustrated in the Bills now before the Prussian Parliament in respect to theological studies. The State proposes to ensure that every clergyman shall possess liberal culture; and with this view, whether Protestant or Catholic, he must go through a curriculum of classics, literature, philosophy, and natural science in the universities, and not merely in special seminaries. The examination in these subjects is to be in the hands of the State, and not in those of the bishops. The curriculum of study belongs to the university, the evidence of its fruition to the State. Such paternal functions of the State, even though chiefly exercised through university professors, are rather incomprehensible to us. Doubt is expressed in Germany itself, as to whether it is wise for the State to secure its ends by examinations; for Professor Planck, in his recent rectorial address at Munich, counsels the State to seek other means for obtaining good professional men than "its narrow and doubtful" examining system. While no country in the world has benefited so much as Germany by its university system, none, except China, has suffered so much as France by giving a preponderance to examination, and subordinating to that the teaching functions of universities. I have shown fully elsewhere (*Teaching Universities and Examining Boards*, Edmonston and Douglas, Edinburgh, 1872) how France now admits that the poverty of intellect displayed during her recent crisis was the consequence of her having sacrificed the national intellect to an uniform State-examining system.

It is not easy, in the short time at my disposal, to show you how Germany has managed to reconcile free university teaching with a State-examining system, without injurious consequences to intellectual development, but this has been well done by Mathew Arnold. Certainly German ideas of examination are as opposite to those which prevail in our universities as they can well be. With us examination is the end of university life, while in Germany it is the mere test of a well ordered course of study. All *specielle vorstudien* are expressly discouraged, and the examination aims at the proof that the student has attained *das Wesentliche und Dauernde*, or a substantial and enduring result of study. Under our examining systems cram flourishes; in Germany it has little existence, for the examination, which is a subordinate function of their university system, aims at the proof of intellectual development fitted for a future career of usefulness.

Let us apply these national experiences to the satisfaction of a reasonable demand, that the medical practitioners in this country should at least possess a minimum standard of efficiency. While the State has a right to demand that, it is clearly its interest and policy to effect its purpose in such a way as will insure maximum and not minimum qualifications. It is not wise to have uniformity either in teaching or in examination; for differentiation is as important in intellectual as it is in physical life. But a one-portal system is based on uniformity, and it would effect it as surely on the student as the single hole of the wire-drawer does upon the wire drawn through it. To avoid this, we now find the one-portal system abandoned for a three-portal system, one door of entrance being proposed for each section of the United King-