ON THE
PATHOLOGY OF THE MORBID STATE
COMMONLY CALLED
CHRONIC BRIGHT'S DISEASE WITH
CONTRACTED KIDNEY.
("ARTERIO-CAPILLARY FIBROSIS.")

BY
SIR WILLIAM W. GULL, BART., M.D., D.C.L., F.R.S.,
AND
HENRY G. SUTTON, M.B., F.R.C.P.

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DR. BRIGHT and subsequent pathologists have fully recognised that the granular contracted kidney is usually associated with morbid changes in other organs of the body. The disease in the kidney and the coexistent morbid changes are commonly grouped together and collectively termed "chronic Bright's disease."

In this communication we propose to consider the pathology of this morbid condition. We are induced to do this because our observations tend to show that the present prevailing pathological theories do not fully comprehend the whole history of the disease.

It is, we believe, generally assumed that the kidney is the organ primarily affected, and in consequence a cachexia is induced through which other organs subsequently suffer and

undergo chronic changes. How far this opinion is well founded we now proceed to examine.

It will be advantageous first to consider the morbid changes in the kidneys, and subsequently the morbid changes which occur in other parts of the body.

The morbid anatomy of granular contraction of the kidney is generally so well known that it is needless here to fully describe it, though it is necessary to recount some of the histological changes which may be observed in this state.

For our examinations some sections were made of the diseased kidneys without any hardening process; others were made after freezing: some sections were faintly stained by a weak solution of nitrate of silver, others by carmine, and others were left in a natural state. The examination was made by $\frac{1}{5}$ and $\frac{1}{8}$ inch object-glass, and the following changes were noticed.

A fine fibroid or hyalin-fibroid substance was seen between the convoluted tubules, which made the tubules appear wider apart than normal.

In some parts this substance had a homogeneous appearance, in others it had a striated or fibroid appearance like a network, and contained minute indistinct nuclei, (?) and in others it had a coarser and more defined fibre-like character. This substance was seen in considerable quantity round the Malphigian bodies, and in still greater amount in and around the walls of the minute arteries. In some renal arterioles injected with Beale's blue translucent fluid the elastic tissue of the tunica intima was normal. The muscular tissue appeared to be changed in some of the vessels—it seemed thicker than natural, particularly when the arterioles were viewed longitudinally. The muscular nuclei were indistinct, and many of them were so altered as hardly to be recognisable. External to the muscular nuclei, there was a quantity of hyalin-fibroid substance, and the layer formed by this material was much thicker than the muscular layer (Plate VI, fig. 7). This hyalin-fibroid was in some of the arterioles bounded externally by a few coarser fibres, in some it merged imperceptibly into the fibroid tissues lying between the

tubules. The arterioles were often very much thickened and tortuous. The lumen of some of them was encroached upon and narrowed, and in some it seemed completely obliterated. The morbid material here alluded to had in and around the walls of some of the arterioles very little hyaline appearance, but was more coarsely fibrous. Many of the convoluted tubules were not appreciably altered except that their epithelium was more or less granular. Others were much shrunken and wasted. Here and there a quantity of the fibroid material was noticed, arranged in a concentric manner; and in the centre of the coil were a few indistinct, shrivelled, scarcely recognisable, epithelial cells. These fibroid coils apparently enclosed atrophied tubules. Some of the tubules were irregularly dilated, or apparently formed a number of cysts. In some situations, almost all trace of tubular structure had disappeared, and scarcely anything but fibroid tissue remained. When the cortex was very much contracted the Malpighian bodies were found lying very much closer together than normal, and surrounded by a considerable quantity of fibroid tissue (see Plate V, fig. 1).

In an early stage of granular contraction of kidney, when there are no changes appreciable to the naked eye, except that the surface of the kidney is as technically called in the post-mortem room "coarse," the following alterations may be observed. In the walls of some of the minute arteries and also outside and around them, there appears to be an excess of fibroid tissue, and the arteries in consequence seem thicker than normal. The number of arterioles thus affected varies very much. Those most altered are in the cortical parts. In making this observation we are well aware that there is normally more or less of areolar tissue around the minute renal arteries, therefore it is difficult with any degree of certainty to appreciate any slight increase in such tissue, but after careful observation it seemed to us that the outer coats of the arterioles even at this early stage were thickened by increase of fibroid tissue. The muscular tissue did not appear increased. The intertubular tissue seemed also altered in some parts. It was abnormally distinct

and clearly defined. The fibre-like appearance was unduly marked as contrasted with the normal kidney intertubular structure. Immediately under the capsule and corresponding to the depressions of the granular surface, fibroid tissue extended inwards, obscuring and destroying the tubular structure. This appearance has been well described by Dr. Dickinson. Amongst this fibroid tissue we occasionally observed a number of corpuscles and nuclei.

It remains for us to describe the morbid changes in the epithelium of the convoluted tubules. Dr. Johnson, p. 219, 1st edition of his work on 'Kidney Diseases,' states that in the early stage of the disease now under discussion the only deviation from the usual appearance is in the epithelial cells of the convoluted tubules. "The epithelial cells are," he says, "opaque and have an unusual, finely granular appearance; in some of the tubules there is an appearance as of entire cells having been shed filling the tubules and rendering them opaque; in others there is an equal filling and opacity of the tubules from contained epithelium in a disintegrated condition." Dr. Dickinson states, on the contrary, that in the early stage of granular renal disease the epithelium is as in healthy kidneys, but it is altered where the kidneys are greatly contracted. We also ourselves have observed in some specimens that the epithelium cells were finely granular, but natural in all other respects. In others we have noticed a quantity of granular or homogeneous matter that rendered the tubules more opaque and almost completely concealed the epithelium. In the advanced stages of granular disease the cells were very irregular in shape and shrivelled, their nuclei were indistinct. In some spots the epithelial cells were absent, and in other tubules of the same specimen the epithelium was observed to be not appreciably altered.

In the kidneys which had recently been the seat of acute nephritis the epithelium was more opaque and contained granules. Here and there only was an epithelium cell in its natural position, the rest of the tubule being denuded, or the cells concealed by granular matter.

From the descriptions given it would appear that Dr.

Johnson, Dr. Dickinson, and ourselves have observed similar changes in the epithelium. ' From these appearances Dr. Johnson draws the conclusion that the secreting cells of the kidney have undergone a primary pathological change. He refers this change in the cells to their removing from the blood some poisonous materials with which it is charged, and so becoming themselves changed in appearance and structure. We submit that it is not necessary to adopt Dr. Johnson's explanation in order to account for these appearances and alterations in the epithelium. The granular appearance of the epithelium is by no means confined to Bright's disease. It is usual to find it in kidneys which present no sign of disease except merely mechanical venous congestion. This form of congestion is seen more or less in by far the majority of post-mortem examinations. It occurs after death from various causes, and it is certainly not dependent on any definite pathological state beyond that of venous obstruction, which notoriously always takes place to a greater or less extent during the process of dying:—in consequence of obstruction to the circulation through the right side of the heart the blood accumulates in the vena cava and in the renal and other veins; the renal venules and capillaries become distended, and serum with albumino-fibrinous contents transudes into the tubules, the urine becomes albuminous, and the epithelial cells are coated with granular fibrinous matter. This exudation may be moulded in the tubules, and form hyaline or other casts, which may be retained in the tubules or passed in the urine.¹ Kidney epithelium may also be shed as the epithelial cells of the bladder may soak off during or after death. Urine contained in the bladder is notoriously commonly albuminous after death, and charged with columnar and other epithelium. In such cases there is often no evidence of kidney or bladder disease. It appears to us, therefore, that Dr. Johnson has attached undue importance to these slight changes in the epithelial tissue, whilst the greater

¹ Recent observations on the minute anatomy of the kidney raise a doubt whether casts formed in the convoluted tubercles of the cortex can escape from them.

changes which are seen in the advanced stage of the granular renal disease may be regarded rather as a consequence than as a cause of the atrophy of the kidney tubules. Our observations, on the contrary, seem to show that the visible morbid changes in granular contracted kidneys are due to the primary formation of "fibroid" or "hyalin-fibroid" substance in the intertubular parts, including the vessels, and to atrophy of the tubular and intra-tubular structures of the kidney.

This formation commences in different parts of the kidney, commonly near the surface, but it also seems to us to commence in the outer coats of the arterioles and in the walls of the capillary vessels. From these parts it extends round the convoluted tubes and Malpighian bodies. This fibroid or hyalin-fibroid substance subsequently contracts and draws the Malpighian bodies together, compresses the urinary tubules and vessels, and may entirely obliterate them.¹ This thickening of the capillary walls and the diminished calibre of some of the arterioles must naturally interfere with the nutrition of the tissues, and tend to produce further atrophy. Owing to these changes in the capillaries and arterioles the quantity of blood passing to the secreting cells would be lessened, and in consequence diminished activity in the secreting function would occur and promote atrophy even in normal epithelium.

We would not, however, maintain that the changes in the epithelium must be entirely secondary, for changes may be going on in it coincident with the hyalin-fibroid formation in the vessels.

Where the kidney disease was far advanced hyalin-fibroid changes were seen in the minute renal arteries precisely similar to those observed in the arterioles of the pia mater and of other parts of the body. Where the kidney disease was in an early stage the tissue around the arterioles of the kidney and between the tubules had a sharply defined, somewhat distinctive appearance. We have observed a similar appearance in the minute arteries of the pia mater.

¹ See Plate V, fig. 1.

Our examinations have shown us that this hyalin-fibroid formation commences, as regards the pia mater, in the outer coats of the arterioles and in the walls of the capillaries themselves; and observing that the same kind of formation occurs in the arterioles and capillaries of the kidney, we are led to infer that this change begins in the kidney as in the pia mater in the walls of the arterioles and capillaries, and subsequently extends to the surrounding structures.

We have now to consider the other pathological changes which make up the morbid condition known as chronic Bright's disease with contracted kidneys.

Of these the most important and, as we think, essential and primary are the changes in the vascular system, more or less general throughout the body.

Dr. Bright and subsequent observers have recognised that arterial changes are part and parcel of this state.

Dr. George Johnson in his work on 'Diseases of the Kidneys,' published in 1852, says that the walls of the minute renal arteries are usually much thickened, and that this is due to hypertrophy of their muscular layer.

Dr. Wilks, in the 'Guy's Hospital Reports,' published in 1853, remarks that the occurrence of diseased arteries in the chronic form of Bright's disease is well known. It is necessary we should state that both he and Dr. Bright referred to atheromatous changes, and to the thickening which occurs in the larger and moderate-sized arteries to changes in arteries visible, in fact, to the naked eye. Many other writers have made the same observations, but it is notorious that atheroma is common in the large vessels, not only in Bright's disease, but in other maladies; still there can be no doubt that it is exceedingly common in granular degeneration of the kidneys. Dr. Dickinson found it in the proportion of 52 per cent. Dr. George Johnson a few years ago showed that not only were the microscopical arteries of the kidneys thickened, but that the minute arteries of the skin and other parts of the body were similarly changed, and he attributed the change to hypertrophy of their muscular tissue.

Our observations confirm the opinion that the minute

arteries are thickened in chronic Bright's disease, and we gladly acknowledge the debt the science of medicine owes to Dr. George Johnson in so distinctly insisting upon the fact.

Dr. Beale has also confirmed the accuracy of Dr. Johnson's observations as regards the thickening of the minute renal arteries, and he expressed an opinion that this is due not to hypertrophy of the muscular layer of the vessel, and he has stated that the outer layer of these vessels is thickened.

We have examined by aid of the microscope a large number of vessels taken from bodies in which there was more or less chronic disease of the kidneys, chiefly granular degeneration, and we now desire to bring the result of our observations under the consideration of the Society.

For the purposes of such examination we chiefly selected the vessels of the pia mater, since they offered the greatest facilities for our purpose, and most of the descriptions of the vessels and the measurements mentioned herein have therefore reference to the minute arteries of the pia mater. In a few specimens the arteries were injected, but the major part were in the natural state, or merely stained. The portions of pia mater were usually taken from the under surface of the cerebrum where the membrane is thin. Afterwards the specimens were stained in carmine, and subsequently mounted in glycerine and camphor water. The vessels were then examined by $\frac{1}{8}$ - or $\frac{1}{16}$ -inch object-glass, and a first or second eye-piece. The outlines of some of the arterioles of each specimen were drawn by means of the camera lucida, and measured by a scale divided for the sake of convenience into $\frac{1}{1000}$ parts of an inch. This scale was adopted because it allowed of the estimation of minute differences.

It will be useful if we here mention that we generally found in the arterioles measuring about $\frac{1}{1000}$ part of an inch in diameter, that the lumen of the vessel was about twice as great as the thickness of its wall: *i.e.* on observing the vessel through the microscope, we found the thickness of its two sides or walls was equal to the lumen.

In the larger arterioles the relative width of walls and lumen was different, the lumen being proportionately larger.

In nearly all the cases from which the diseased vessels were taken the kidneys were more or less granular, and some much contracted. In a few cases only were the kidneys large, white, and mottled. The condition of the viscera is briefly given in the report of the cases, the details of which are recorded in the appendix to this paper.

The large arteries are usually not much thickened in chronic Bright's disease, but occasionally their outer layer is indurated and thickened by this fibroid substance. The greatest amount of thickening occurs in the coats of the arterioles and capillaries.

The outer layer of the arteries, measuring about $\frac{1}{1000}$ of an inch in diameter, are also not unfrequently thicker than natural. The degree in which the affected vessels are altered, and the extent to which the morbid change is diffused over the vascular system of the different organs, varies very much in different cases. In some, almost all the arterioles seemed more or less affected, in others, only an arteriole here and there, the remaining vessels not being greatly, if at all, altered, though frequently their outline is more clearly and sharply defined than natural. A practised eye can usually recognise this early stage of change by this sharply defined outline. In consequence of the occasional limitation of the morbid change to a few arterioles, care is requisite in making an examination or the diseased vessels may easily be overlooked.

The morbid change in an affected vessel is by no means necessarily uniform throughout its length, and may not extend to the branches it gives off. The lumen of the affected arterioles is sometimes distinctly lessened and the diseased vessels are not unfrequently tortuous, whilst in the walls of some of the minuter ones it is common to find groups of fat-granules aggregated together. The morbid changes vary according to the size of the vessels affected. In the larger ones, in which the three coats are distinct, the inner layer (tunica intima) is sometimes thickened to a marked degree. When this happens the elastic tissue is seen to form the inner edge of the arterial wall, and outside this the tunica intima has a fine fibrous or molecular appearance.

The muscular coat is also variously altered. Thus where seemingly normal, if placed, as fresh as possible, in a strong solution of carmine for about twelve hours, the nuclei do not absorb the carmine so readily as in healthy vessels. This is a very distinct difference. The muscular layer seems also often relatively increased. This appearance is, however, to us equivocal, for with this apparent increase it is common to find, even in the same vessel, the muscular tissue wasted, and the nuclei irregular in shape, or reduced to small globular bodies, having the appearance of large fat-granules. The muscular coat may be, in fact, degenerated into granular matter at one part, whilst in a contiguous portion of the vessel, it may seem to be relatively increased. The examination of this point which refers to the change in the muscular coat has much occupied our attention, but we have failed to discover evidence of the muscular hypertrophy so much insisted upon by Dr. George Johnson. Outside the muscular coat the morbid changes, to which we have already referred when describing the changes in the kidney, are well marked in the diseased vessels. The outer portion of the altered arterioles, as seen under the microscope, is commonly bounded by a few well-defined fibres of white fibrous tissue, within which, and immediately in contact with the muscular layer, there is a more or less homogeneous hyalin formation. Where this hyalin substance is in contact with the fibrous outer layer it has a fibrous appearance (see Pl. V, fig. 4). The general aspect of the affected vessels might give, as Dr. G. Johnson affirms, an appearance of true hypertrophy, but after full examination it seems to us the whole is due to a morbid process, and not to an increase of normal nutrition, this morbid process giving rise to the formation of the hyalin-fibroid substance we have described. In the otherwise homogeneous hyalin substance, ill-defined nuclei or corpuscles are often seen. These corpuscles cannot, when fully examined, be mistaken for normal muscle nuclei, since they are irregularly disposed in the tissue, and wanting in that definition characteristic of normal elements. Although in the more characteristically affected vessels the "hyalin" substance is, as stated, more

or less homogeneous, still this is by no means uniform, for not only may it contain the bodies just referred to, but it may itself be more or less replaced by coarse fibroid and granular material.

The change from the "hyalin" to the "fibroid" character is probably due to slowness of formation, as it is more common in aged persons. It is to be stated that the fibroid changes do not occur in the smallest of the capillary arterioles. In these the homogeneous or nearly homogeneous "hyalin" change alone occurs.

The morbid appearances here described were chiefly studied in the minute arteries and larger capillaries of the pia mater, but, as we have said, the same may be seen in the arterioles of the kidneys, and, we may now add, of the skin, of the stomach, of the spleen, lungs, heart, retina, &c.¹

In the arterioles of the spleen and lungs the morbid substance was more coarsely fibroid, and the pure hyalin change less distinct.

In reference to the seat of the hyalin-fibroid formation, we cannot pass over the question which will probably be raised by those histologists who believe in the existence of perivascular sheaths of the minuter vessels. On the existence of such canals we express no opinion, but assert only that the morbid changes we have noticed occur chiefly outside the muscular layer.

The following is a general summary of our microscopical observations :—

1. That the arterioles throughout the body in that condition usually called chronic Bright's disease with contracted kidney, are more or less altered.

2. That this alteration is due to a "hyalin-fibroid" formation in the walls of the minute arteries, and a "hyalin-granular" change in the corresponding capillaries (see Pl. VI, figs. 1, 2).

3. That this change occurs chiefly outside the muscular layer, but also in the tunica intima of some arterioles.

4. That the degree in which the affected vessels are

¹ Plate V, fig. 2; Plate VI, fig. 1.

altered, and the extent to which the morbid change is diffused over the vascular system of the different organs, varies much in different cases.

5. That the muscular layer of the affected vessels is often atrophied in a variable degree (see Pl. VI, fig. 3).

In order to ascertain the extent to which this change in the vessels existed, the pia mater was examined in a large number of cases of persons who had died of various diseases. Fifty-five cases were thus examined, and the result is found in the appendix given, and shows that this "hyalin-fibroid" change was associated with granular contraction of the kidneys in most of the cases; but, in some few, the vessels of the pia mater had largely undergone this change; and the heart was hypertrophied, whilst the kidneys remained healthy.¹

These examinations also showed that, in some cases, a few only of the minute arteries and capillaries were thickened, the left ventricle of the heart slightly dilated, whilst the kidneys were a little contracted in some of these cases, but in others not.²

We have further to state, that this "hyalin-fibroid" change was not found in the vessels of healthy persons, who had been accidentally killed, nor in ordinary phthisis, nor in other diseases whose morbid conditions are not allied to the cachexia of so-called chronic Bright's disease with contracted kidneys.³

It is now to be asked, What is the morbid condition antecedent to the changes in the minute arteries and capillaries? Dr. George Johnson considers that an impure state of the blood induces this vascular change. In order that we may not do his opinion any injustice, it will be well to quote his own words. He says, "In consequence of the degeneration of the kidney the blood is morbidly changed. It contains urinary excreta, and it is deficient of some of its

¹ See Appendix, Cases 2 and 3.

² See Cases in the Appendix, Nos. 26 to 35.

³ See particulars of cases given in the Appendix, Nos. 36 to 55.

own normal constituents. It is, therefore, more or less unsuited to nourish the tissues, more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The left ventricle, therefore, makes an increased effort to drive on the blood. The result of this antagonism of forces is that the muscular walls of the arteries and those of the left ventricle of the heart become simultaneously and in an equal degree hypertrophied. The persistent over action of the muscular tissues, both cardiac and arterial, is registered after death in a conspicuous and unmistakable hypertrophy."

This theory does not appear to us supported by the facts.

Dr. George Johnson states that the change in the minute arteries is simply hypertrophy of the muscular coat. But our examinations show that they are thickened by a "hyalin-fibroid formation," and that in fact the muscular coat is often variously atrophied.

Thus, Dr. George Johnson regards the changes in the heart and arterioles in chronic Bright's disease as a physiological result, due to a morbid change in the blood, whilst our observations lead us to regard the cardio-vascular changes as throughout a morbid one.

Further, extreme degeneration of the kidneys, and together with this, no doubt, a noxious state of the blood, to which Dr. Johnson attributes the vascular change, may be present, and the vessels may frequently be found healthy. In support of this statement we have recorded in the appendix cases of large white kidneys, and of scrofulous pyelitis,¹ in which the kidneys were much diseased, and the renal changes were chronic attended with general œdema and uræmic symptoms, and the vessels were healthy. Dr. George Johnson and other pathologists regard, as we have said, the general vascular changes as essentially consecutive to antecedent renal disease, but our inquiries show that these changes are, or may be, independent of renal disease, and that the renal change in chronic Bright's disease with contracted kidney, when present, is but a part of a general morbid condition.

¹ See Appendix, Cases 56 to 60.

We are led to conclude that the kidney disease does not give rise to the vascular change. Our investigations show the disease under the following forms:

(1) Kidneys often much contracted, heart much hypertrophied, minute arteries and capillaries proportionately thickened by "hyalin-fibroid" formation.¹

(2) Kidneys little contracted, but heart much hypertrophied, minute arteries and capillaries much thickened by "hyalin-fibroid" substance.²

(3) Kidneys healthy, whilst heart much hypertrophied and minute arteries and capillaries much thickened by "hyalin-fibroid" substance.³

These facts show that there is a morbid state in which the kidneys are contracted, the heart hypertrophied, and the minute arteries and capillaries altered by a "hyalin-fibroid" formation. Further that the kidney changes are often, but not always, a part and parcel of this morbid state. The absence of such lesions of the kidneys proves that they do not constitute an essential and indispensable part of the general process.

We have already mentioned that the granular contracted condition of the kidney is dependent on a "hyalin-fibroid" formation in its arteries and other structures. The same kind of morbid change, in fact, occurs in the contracted kidneys as occurs in the arteries and capillaries of the pia mater, of the skin, of the heart, of the stomach, and other parts. The kidney disease would, therefore, seem to be but a part but not an invariable part of the "hyalin-fibroid" change, but as the vascular system was at some part affected with this change in all the cases, we regard such vascular change as the constant and essential part of this morbid state.

We have next to consider the pathology of another morbid condition which forms part of the state known as chronic Bright's disease. We refer to hypertrophy of the

¹ See Appendix, Cases 1, 4, 6, 8, 15, 21.

² See Appendix, Cases 7, 10, 20.

³ See Appendix, Cases 2, 3, 19.

left ventricle of the heart unaccompanied by any valvular defect or adhesion of the pericardium.

The morbid appearances of this hypertrophy are so well known that it is not requisite for us here to describe them, but it is necessary we should state that we have found the minute arteries in the walls of the heart much thickened by the formation of "hyalin-fibroïd" substance already described.¹

Bright, to account for this hypertrophy, says: "The most ready explanation appears to be that the quality of the blood is altered by the kidney disease. The blood in consequence affects the minute and capillary circulation so as to render greater action necessary to force it through the vascular system." Many pathologists have adopted this explanation. Dr. Wilks, however (see 'Guy's Hospital Reports' for the year 1853), has suggested that the hypertrophy may be dependent on atheromatous changes in the vessels.

They who adopt the explanation given by Bright state in support of their opinion, that the hypertrophy of the heart and the renal disease are so frequently associated together as to show that there is some intimate relation between them. They further state that this hypertrophy occurs in all forms of chronic kidney disease; and therefore they conclude that it is consequent upon diminished excretory power of the kidney.

Dr. Johnson, who supports this opinion, further states that physiologists have demonstrated that impure blood circulates with great difficulty and creates an impediment.

There is, however, evidence on the other hand which appears to be strongly opposed to these views.

Thus the cardiac hypertrophy and the renal disease are no doubt frequently associated, as we have said; but this does not prove that there is a relation of cause and effect between the two states; for it is evident that both these morbid conditions may be dependent on a third more general one. Moreover, against this commonly accepted explanation it can be shown that in many cases of chronic kidney disease

¹ Plate V, fig. 6.

the heart is not hypertrophied. Dr. Wilks has mentioned to us that in many cases of large white kidney he has found the heart free from hypertrophy. Dr. Dickinson states that simple hypertrophy of the left ventricle is rarely associated with any form of renal disease excepting granular degeneration.

We have post-mortem records of seventeen cases of large white kidneys, and in twelve of them the heart was healthy. Dr. Grainger Stewart found in lardaceous disease of the kidney that the heart was hypertrophied in only four per cent. of the cases. In four cases, in which the kidneys were almost destroyed by scrofulous pyelitis, we found the heart healthy.

We have particulars of nine cases in which the kidneys were very contracted and the heart was free from hypertrophy.

In these various forms of kidney disease the morbid changes were chronic, and it must be supposed that the blood was altered. The morbid conditions to which the hypertrophy is attributed were, therefore, present, but the heart, at the same time remaining healthy, shows that these renal and blood changes are not sufficient to produce hypertrophy, and indicates that when present it is due to some other condition.

In some of our cases, it is true, the kidneys were large, white, and mottled, and the heart was hypertrophied, but besides the renal affection the vessels in these cases were much diseased by the "hyalin-fibroid" formation.

We attribute the hypertrophy to the vascular change.

Dr. George Johnson explains the occasional absence of hypertrophy in cases of chronic Bright's disease by assuming that the muscular tissue of the heart is imperfectly nourished in such cases. In considering this suggestion it is necessary to bear in mind that, if the blood be altered, and the circulation in consequence impeded, the left ventricle of the heart must contract with greater force than natural to overcome the impediment. To accomplish this it must acquire increased strength:—become hypertrophied; or it would be unable to overcome the obstruction, and the ven-

tricle then being unable to empty itself completely, would in consequence become dilated.

Dr. Johnson may in this manner explain disproportionate dilatation, but does not explain how a normal-sized heart acquires the additional force requisite to overcome the supposed obstruction.

Experience has fully shown that hypertrophy of the left ventricle occurs much more frequently with granular contracted kidneys than with any other form of renal disease; and our observations, supported by the particulars given, appear to show that in this form of kidney disease the hypertrophy is not induced by the renal affection, but by a morbid change in the minute arteries and capillaries. Of 34 cases in which the kidneys were healthy, excepting that they were slightly granular, or in other words "coarse," and contained some cysts, the left ventricle was hypertrophied in 18; and there was no valvular disease or pericardial adhesion to account for the hypertrophy. In these 18 cases the hypertrophy would appear to have been the older and preceding condition.

Niemeyer states¹ that Bamberger, arguing against the view that cardiac hypertrophy is dependent upon the renal disease, has shown that it begins in the earlier stages of Bright's disease. Observing, therefore, that this cardiac hypertrophy occurs not unfrequently in the very early period of the kidney affection, when the excretory function is not greatly altered; and further observing that the heart in some cases is not hypertrophied, when the kidneys are very much contracted, and the function of excretion is of necessity greatly altered, we also again conclude that the hypertrophy is not dependent on the kidney change.

The following particulars seem in fine to indicate that the hypertrophy is induced as we believe by the morbid changes in the vessels.

The heart was found (see cases in the appendix) hypertrophied in all the cases in which the vessels were much and

¹ 'Text-book of Practical Medicine,' vol. i, p. 300.

generally thickened by the "hyalin-fibroid" change :—the heart was found slightly hypertrophied where the vessels were a little thickened, or a few of them only were thickened by this "hyalin-fibroid" material ; the heart was much hypertrophied when the vessels were much thickened and there was no kidney or other disease, except this vascular change adequate to account for it.

Nor is it difficult to explain how the vascular disease gives rise to the cardiac hypertrophy.

The "hyalin-fibroid" material in the walls of the arterioles must be an impediment to elasticity, and it can be experimentally shown that greater force is required to propel a fluid continuously through a non-elastic than through an elastic tube. The left ventricle, therefore, owing to this diminished elasticity of the arterial walls, has of necessity to contract with greater force to carry on the circulation.

It remains to briefly notice a few other morbid conditions which form part of the pathological changes known as chronic Bright's disease with contracted kidneys.

The first is vesicular emphysema. It is well known that vesicular emphysema and granular contracted kidneys frequently coexist. Of 33 cases of persons about middle age in which the lungs were emphysematous, the kidneys were more or less granular, and contracted in 22 ; and there were no changes in the lungs to show that such emphysema was compensatory. In some of the cases the emphysema was great whilst the kidneys were only slightly granular ; in other cases the kidneys were much contracted, whilst the lungs were comparatively little diseased, which facts seemingly show that the emphysema may precede the kidney contraction, or the kidney disease may occur antecedent to the lung disease ; and this is fully borne out by clinical experience. We have not as yet had an opportunity of examining microscopically the vessels of a number of emphysematous lungs associated with contracted kidneys, but in a few such lungs we have found the vessels surrounded by what seemed to be an unusual quantity of fibroid tissue, and it is common to see without the aid of the microscope

the connective tissue increased around the vessels and tubes in these emphysematous lungs.

With granular contracted kidney we have also found the aorta and aortic valves much diseased and the seat of atheromatous and sometimes calcareous changes giving rise to dilatation of the aorta with or without aortic obstruction and regurgitation.

Another morbid condition to be noticed is so-called "retinitis albuminurica," which experience has shown to be frequently associated with granular contracted kidney. In this change of the retina there is sclerosis of the connective tissue. There is also sclerosis and fatty degeneration of the coats of the blood-vessels, and the vessels have been found greatly narrowed and even obliterated. These morbid changes in some cases lead to atrophy of the optic disc and of the retina.¹ The tunica adventitia of the larger retinal vessels is often considerably increased in thickness. Mr. Bader in describing these diseased vessels says,² "Sclerosis is seen in the coats of the blood-vessels, especially of the small arteries and capillaries of the retina and choroid. Their walls are thickened through a homogeneous, strongly reflecting, not quite transparent substance.

From the description given it would seem, therefore, that the morbid appearances observed in these retinal vessels were similar to those we have observed in the arterioles and capillaries of the pia mater, kidneys, and other parts.

The spleen has been found by us in instances of chronic Bright's disease: diminished in size; in some cases weighing only two or three ounces, and in some no more than one ounce and a half: capsule thickened: on section substance tougher than natural, but the most noticeable alteration was the increased quantity of fibrous tissue: under the microscope the vessels were seen surrounded by a much larger quantity of fibroid tissue than usual, and in the outer coats of some of the minute arteries, "hyalin-fibroid" changes were obvious, and similar to such as occur in the

¹ See Mr. Soelberg Wells' 'Treatise on Diseases of the Eye,' p. 358.

² See 'Guy's Hospital Reports,' 1866.

arteries of the kidney and pia mater. In some cases where the kidneys were greatly contracted the spleen was very much affected in the manner described, but in some cases the spleen was much wasted, and the kidneys were little contracted; in other cases the spleen was very little wasted, whilst the kidneys were very much contracted.

We also found in many instances of granular kidney disease that the convolutions of the brain were much wasted, whilst the minute arteries of the pia mater were thickened by "hyalin-fibroid" substance. In other cases of contracted kidneys, in which the brain was healthy, no "hyalin-fibroid" changes were found in the vessels of the pia mater. In a few cases the convolutions of the brain were wasted, and the vessels of the pia mater were thickened by this "hyalin-fibroid" change, whilst the kidney and heart were healthy, showing the independence of this change of heart or kidney.

Fibroid changes with atrophy have also been observed by Drs. Fenwick and Wilson Fox in the intertubular portions of the stomach in chronic Bright's disease. The arterioles of the stomach have been found thickened. We have also observed in chronic Bright's disease with contracted kidneys great fibroid thickening in the walls of the minute arteries of the stomach and fibroid changes in the intertubular parts.

The clinical history of this morbid state enables us to get clearer views of its pathology. Old age is not an entity, but it is pre-eminently a condition or set of conditions predisposing to that state which is called chronic Bright's disease with contracted kidneys. To demonstrate the extent to which man in different periods of his life is exposed to granular degeneration of the kidney, we have collected from post-mortem registers particulars of 336 cases, death being due to various diseases. These cases were grouped according to the age. The following is the proportion of granular degeneration of the kidneys, and the proportion of granular to healthy kidneys in each decennial period of life.

From the age of	The total number of deaths from all causes.	The number of those cases in which the kidneys had undergone granular degeneration.	The proportion of granular to healthy kidneys in each decennial period of life.
10—20 years.....	44	1	1 granular kidney in 44 healthy ones.
20—30 „	69	2	1 ditto in 34 ditto.
30—40 „	84	10	1 ditto in 8·4 ditto.
40—50 „	82	31	1 ditto in 2·6 ditto.
50—60 „	39	28	1 ditto in 2·3 ditto.
60—70 „	13	12	1 ditto in 1·08 ditto.
70—80 „	5	4	1 ditto in 1·2 ditto.

It here appears that after the age of forty a large proportion of persons who die of various diseases have more or less granular contraction of the kidneys as shown on the surface of these organs; and it further appears that after forty years of life granular degeneration greatly increases as age advances.

Granular degeneration of the kidneys, therefore, belongs principally to the period of life at or over forty years of age.

Dr. Dickinson shows that of 308 persons with granular kidney only 75 died before forty years and 233 after forty years of age. It is evident, therefore, that chronic Bright's disease with granular kidney is allied with the conditions of age and experience shows that it is caused by all those influences which are recognised as tending to bring about senile changes whether prematurely or not.

Children are occasionally the subject of granular contracted kidney, and there is given in the appendix to this paper the case of a girl, aged 9 years, in whom occurred granular and very contracted kidneys, hypertrophied heart, and very thick “hyalin-fibroid” arterioles.

We here mention that occasionally in young subjects not

over twenty years of age the kidneys may undergo extreme contraction and degeneration, and be apparently strictly a local affection, and death may occur from the so-called uræmic poisoning without any signs of the cardio-vascular changes characteristic of chronic Bright's disease with contracted kidneys of a later age. These cases, we believe, have another causation.

It has been shown above, and it is notorious, that many organs are diseased in the morbid state known as chronic Bright's disease with contracted kidneys, namely, the kidneys themselves; the minute arteries; and the capillaries; the heart; the lungs; the aorta; the brain; the retina; the spleen; the stomach; and the skin.

But these various parts and organs are not constantly affected in the same order, nor is there any constant proportion between them as to the morbid changes each may undergo. In some cases all these organs are much diseased, and more or less equally so; in other cases the morbid changes are confined to a few and isolated parts.

In some cases the changes seem to commence in the kidney or in the heart, sometimes in the lungs or in the brain, or perhaps in other organs.

Clinical medicine, especially as followed in private practice, enables us often to predict and trace these changes onwards until the morbid formation is general. Thus, a patient may come under care for headache and other allied symptoms in whom, at a given stage, the renal and cardiac functions may be normal, and as the case goes on the urine first or the heart first, or the breathing may first give signs of further lesion until, as the malady progresses, that state called chronic Bright's disease with contracted kidney may be fully produced, as shown by the thickened heart, the pale watery urine, the shrunken skin, the troubled brain, and the dimmed sight.

Observing that the pathological change may commence in various parts of the body, it might readily be surmised that the symptomatic phenomena must be very different in different cases. Experience fully shows that the symptoms are very

varied in chronic Bright's disease with contracted kidneys; and we would maintain that its prodromata and the prominent symptoms in its course vary with the organ which is primarily or predominantly diseased. But whether many or few organs are affected, the minute arteries and capillaries of the diseased parts have been found by us thickened by "hyalin-fibroid" formation. With this "hyalin-fibroid" formation in the arterioles there is an atrophy of the adjacent textures. This appears to be a characteristic of this morbid change in whatever organ it occurs.

It will follow from these facts that we cannot, as is sometimes done, regard the functional disturbances which occur in many organs during the course of chronic Bright's disease with contracted kidneys as dependent on blood changes only or chiefly. For instance, pain in the head, discomfort after food, palpitation, dry skin, epistaxis, are we believe due, not so much to changes in the blood, as to the changes we have spoken of in the tissues themselves.

The conclusions to which we have arrived may be briefly summed up as follows:

1. There is a diseased state characterised by hyalin-fibroid formation in the arterioles and capillaries.

2. This morbid change is attended with atrophy of the adjacent tissues.

3. It is probable that this morbid change commonly begins in the kidney, but there is evidence of its also beginning primarily in other organs.

4. The contraction and atrophy of the kidney are but part and parcel of the general morbid change.

5. The kidneys may be but little if at all affected, whilst the morbid change is far advanced in other organs.

6. This morbid change in the arterioles and capillaries is the primary and essential condition of the morbid state called chronic Bright's disease with contracted kidney.

7. The clinical history varies according to the organs primarily and chiefly affected.

8. In the present state of our knowledge we cannot refer

the vascular changes to an antecedent change in the blood due to defective renal excretion.

9. The kidneys may undergo extreme degenerative changes without being attended by the cardio-vascular and other lesions characteristic of the condition known as chronic Bright's disease.

10. The morbid state under discussion is allied with the conditions of old age, and its area may be said hypothetically to correspond to the "area vasculosa."

11. The changes, though allied with senile alterations, are probably due to distinct causes not yet ascertained.

Should it be considered necessary to distinguish this morbid state by any special term, we venture to suggest for that purpose the name "arterio-capillary fibrosis."

APPENDIX.

CASE 1.—William L—,¹ æt. 43, who died January 1st, 1871. The autopsy showed the kidneys were very granular and contracted. They were mottled on their surfaces, and in their cortical parts there was a quantity of yellowish-grey matter, similar to what is seen in acute nephritis. The left ventricle of the heart was dilated and its wall much hypertrophied. The heart weighed seventeen and a half ounces. There was no valvular disease. Many of the arterioles of the pia mater were much thickened, and the measurements were as follows:—

The relative width of the vessels.		Of the channel.		Of the sides or walls of the vessel.
11	...	4	...	7
15	...	5	...	10
17	...	6	...	11
10	...	2	...	8
15	...	5	...	10
28	...	8	...	13
14	...	5	...	9
16	...	6	...	10
30	...	14	...	26

¹ Further details of this patient and the cases following may be seen in the post-mortem registers of the London Hospital.

The muscular layers of the arterioles were not increased. The thickening was due to the outer layer of the vessel being increased in size. This part had a very fine fibroid appearance. In some parts it appeared to be made up of homogeneous-looking matter. Many of the arterioles were in this manner diseased.

CASE 2.—Sarah S—, æt. 63, who died January 25th, 1871. Autopsy showed that the cause of death was rupture of left ventricle of heart. The heart weighed fifteen ounces, and its left ventricle was dilated and hypertrophied. The kidneys weighed fifteen ounces; their surface was almost smooth: there was no decided granular change; the cortical parts were not contracted; the kidney substance was red, and showed venous congestion only. Very many of the arterioles were much thickened, and this was caused by a quantity of hyalin-fibroid substance outside the muscular layer, and the tunica intima in some of the vessels also was thicker than natural.

The relative width of vessel.	Of channel.		Of walls.
13	...	6	10
10	...	3	8
16	...	4	12
12	...	3	9

The muscular nuclei were very imperfect in some of the arterioles. In some parts they were normal; in others they were replaced by large clear granules, or they were entirely absent.

CASE 3.—James D—, æt. 77, died of peritonitis following strangulated hernia. The autopsy showed that the left ventricle of the heart was dilated and hypertrophied. Its valves were healthy excepting a little atheroma in the mitral. The kidneys were venously congested, otherwise they were healthy. The walls of many of the arterioles were much thickened, and this was due to a granular fibroid growth outside the muscular tissue. The outermost part of the

wall of the arterioles had a distinct fibroid appearance. Within this there was a homogeneous finely granular substance. Lying amongst this granular material were numbers of short, imperfectly developed fibres and oat-shaped nuclei. In some arterioles the muscular layer was well defined.

Relative width of vessel.		Of channel.		Of walls.
24	...	8	...	16
12	...	4	...	8
22	...	8	...	14
21	...	9	...	12
29	...	12	...	17
38	...	18	...	20

And in some of the arterioles the nuclei were faintly stained with carmine; in many other parts the muscular nuclei could not be seen, or they were very irregular in shape. The tunica intima had a coarse fibroid appearance, and in other arterioles a granular appearance. In some of the arterioles it was thicker than natural. Many of the vessels were in this manner affected.

CASE 4.—James R—, æt. 58, died of suppurative pericarditis apparently pyæmic, July 25th, 1871. The left ventricle of the heart was much hypertrophied, and the aortic and mitral valves were normal. The kidneys were small, their surfaces markedly granular, and they contained cysts.

Relative width of vessel.		Of channel.		Of walls.
20	...	8	...	12
28	...	12	...	16
22	...	8	...	14
25	...	10	...	15
40	...	20	...	20

Some of the arterioles were much thickened, others were not. The thickening was not so great as is observed in some cases. The outer and inner coats of the thickened arterioles contained a quantity of hyalin matter, and to this change

the increased size of the arteriole wall was due. The muscular nuclei were in parts shrivelled and very much wasted. The most marked disease was external to the muscular layer. The walls of the thickened capillary arterioles had a homogeneous granular appearance. A number of fat-granules were aggregated in some vessels.

CASE 5.—James S—, æt. 39, died suddenly, apparently in consequence of dilatation of the left ventricle, on 15th May, 1871. Heart weighed 1 lb. 14½ oz. The left ventricle was very much dilated and its wall hypertrophied. The left auricle was also much dilated. Valves were normal. The spleen was small. The kidneys were faintly granular and much puckered on their surfaces; on section they appeared congested, otherwise the cortical and medullary parts were natural. Some of the arterioles were much thickened, but this change was mostly seen in the very minute ones.

Relative width of vessel.		Of channel.		Of walls.
22	...	9	...	14
23	...	9	...	14
31	...	14	...	17
30	...	10	...	20
20	...	8	...	12
20	...	6	...	14
30	...	12	...	18
33	...	15	...	18

The layer outside the muscular nuclei was much thicker than natural, and it had a distinct hyalin-fibroid appearance. The muscular nuclei were very defined and normal in some parts of the arterial wall and absent in others. The capillary arterioles were also thickened, and they had a fine granular appearance.

CASE 6.—John H—, æt. about 45. He was brought into the London Hospital dead on May 20th, 1871. The left ventricle of the heart was dilated and hypertrophied. The aortic valves were calcareous and incompetent. The lungs

were emphysematous. The spleen was small. The brain was wasted. The kidneys were moderate in size, and they were very granular, and contained numbers of cysts. The cortical parts were smaller than natural. Many of the arterioles of the pia mater were much thickened. External to their muscular nuclei there was a hyalin-fibroid-looking substance, which made the external coat much thicker than normal. Many of the large capillaries were also very much thickened, and their walls had a granular or homogeneous appearance. The tunica intima of some of the arterioles was also thicker than natural.

Relative width of vessel.		Of channel.		Of walls.
28	...	9	...	19
27	...	7	...	13
25	...	8	...	17
21	...	8	...	13
21	...	9	...	12
33	...	10	...	23
20	...	7	...	13
13	...	5	...	8
23	...	9	...	14
11	...	3	...	8
25	...	9	...	16
25	...	9	...	16
14	...	4	...	10
10	...	2	...	8
30	...	9	...	21

CASE 7.—Edward L—, æt. 42; died of erysipelas secondary to a scalp wound. Autopsy Dec. 28th, 1870. The lungs were very emphysematous, more or less, throughout, and there was capillary bronchitis. Many of the minute bronchial tubes were filled with pus. The left ventricle of the heart was dilated and somewhat hypertrophied, also the right ventricle. The spleen and liver were natural; the kidneys were faintly granular, otherwise healthy.

Many of the arterioles and capillaries of the pia mater were much thickened. In the coats of the arterioles external to the muscular nuclei a quantity of hyalin-fibroid substance was seen, and to this change the thickening was due.

The nuclei of the muscular tissues were very indistinct in some parts and absent in others. The walls of the capillaries were thickened by the formation of a granular or homogeneous substance.

The relative width of vessel.		Of its channel.		Of its walls.
7	...	2	...	5
10	...	3	...	7
9	...	3	...	6
11	...	3	...	8
13	...	3	...	10
12	...	5	...	7
34	...	12	...	22
32	...	12	...	20
44	...	18	...	26
42	...	16	...	26

CASE 8.—Emma C—, æt. 9 years. The kidneys were very much smaller than natural, especially the left; one weighed 2 oz., the other $1\frac{1}{2}$ oz. The kidneys in consequence of being greatly contracted were much out of shape; their cortical parts were much reduced in size; their surfaces were very irregular and puckered, not finely granular. The heart's left ventricle was dilated and hypertrophied; its valves were healthy. The spleen was small and tough. Some of the arterioles of the pia mater were very much thicker than natural, owing to the layer outside the muscular layer being much increased.

The relative width of vessel.		Of channel.		Of walls.
30	...	12	...	18
32	...	12	...	20
36	...	14	...	22
34	...	14	...	20
36	...	10	...	26
20	...	6	...	14
20	...	6	...	14
18	...	6	...	12
20	...	8	...	12
20	...	8	...	12
18	...	6	...	12
20	...	8	...	12

The relative width of vessel.	Of channel.	Of walls.
18 ...	8 ...	10
18 ...	5 ...	13
14 ...	6 ...	8
20 ...	8 ...	12

The layer external to the muscular nuclei was thicker than the tunica intima and tunica media, taken together, and in the larger arterioles it had a fine fibroid or hyalin-fibroid appearance. The thickening was most marked in the capillary arterioles or larger capillaries, and this change was due to the formation of granular hyalin substance in the walls of these vessels. The arterioles of the kidneys were much thickened by fibroid changes.

CASE 9.—Margaret B—, æt. 68, died of acute meningitis following iridectomy. The kidneys were granular and cystic, but not apparently contracted. The left ventricle of the heart was slightly dilated and slightly hypertrophied. Some of the arterioles of the pia mater were much thickened, and their outer coats were much increased in size. This portion of the vessel was thickened by the formation of a hyalin-fibroid substance.

The relative width of the vessels.	Of the channel.	Of the walls.
35 ...	14 ...	21
52 ...	19 ...	33
25 ...	7 ...	18
38 ...	10 ...	28
21 ...	5 ...	16
27 ...	6 ...	21
15 ...	6 ...	9
22 ...	5 ...	17
25 ...	8 ...	17

Many of the arterioles appeared to be healthy. The outer coats of the larger arterioles were thickened by a fine fibroid substance. The thickened capillary arterioles had a homogeneous appearance.

CASE 10.—Thomas B—, æt. 69, died of senile gangrene, May 4th, 1871.—The kidneys were granular and cystic, but not contracted. The heart weighed $16\frac{3}{4}$ oz., the left ventricle was dilated and its wall hypertrophied; there was no valvular disease. The lungs were emphysematous and the brain atrophied.

The relative width of the vessels. Of the channel.				Of the wall.
29	...	12	...	17
30	...	14	...	16
21	...	9	...	12
28	...	16	...	12
22	...	8	...	14
32	...	13	...	19
38	...	16	...	22
30	...	10	...	12
20	...	6	...	14
42	...	20	...	20
26	...	8	...	18
24	...	8	...	16

The outer coats of the arterioles were much increased in size, and the vessels in consequence were thickened.

Almost all the arterioles of the pia mater were thickened more or less, but the amount of thickening varied very much in the different vessels; in some it was little, so that it did not much increase the diameter of the vessels; in others it was very great, and it was due to hyalin-fibroid changes in the walls of the arterioles external to their muscular coats.

The tunica intima of some of the arterioles was also increased in size. The walls of the minute capillary arterioles were very much thickened, and they had a molecular hyalin appearance. The muscular nuclei of the arterioles were stained in parts and they appeared to be normal; in other parts they were wasted and indistinct. A number of fat-globules were aggregated together in the walls of many of the minute vessels.

CASE 11.—Frederick B—, æt. 31. The kidneys weighed

10 $\frac{3}{4}$ oz., they were markedly granular, and the cortex was somewhat wasted and they contained several cysts. The left ventricle of heart was hypertrophied and dilated; its valves were healthy; its right side was also dilated; the spleen was large and dark.

Relative thickness of vessel.		Of channel.		Of walls.
23	...	7	...	16
30	...	8	...	22
18	...	6	...	12
25	...	8	...	17
29	...	7	...	22
21	...	6	...	15
26	...	9	...	17
28	...	9	...	19
16	...	6	...	11

The cerebral arteries were atheromatous, and the brain was wasted; many of the arterioles of the pia mater were much thickened by hyalin-fibroid substance outside the muscular nuclei.

CASE 12.—Sarah R—, æt. 32, died August 24th, 1871. The kidneys were granular and contracted. The post-mortem appearances have not been described in the register.

The relative width of vessels.		Of channel.		Of walls.
21	...	8	...	13
22	...	8	...	14
23	...	9	...	14
24	...	6	...	14
20	...	8	...	12
20	...	7	...	13
31	...	9	...	22
27	...	8	...	19
23	...	7	...	16
21	...	8	...	13

The arterioles were very much thickened and their coats outside the muscular layer were much increased in size; some of the arterioles were apparently unaffected, but in many were thickened. The layer outside the muscular

nuclei of the thickened arterioles was seen to consist of a hyalin-fibroid substance, and the thickening was caused by this new material. In this new formation a number of minute nuclei were seen.

CASE 13.—James P—, æt. 49. The kidneys were not granular; but large and mottled, and their appearance indicated that they had been recently the seat of acute nephritis. The left ventricle of the heart was dilated. The liver was in the condition known as incipient cirrhosis.

Many of the minute arteries of the pia mater were much thickened, and the layer outside the muscular tissue was increased; it was much thicker than the middle and inner coat taken altogether, and this thickening was due to a hyalin-fibroid substance. In some of the arterioles the muscular nuclei were very indistinct and they were replaced by fat-granules; in others they were very distinct. The arterioles were not all equally involved. Some of the arterioles of the left ventricle of the heart were much thickened by fibroid formation, also the arterioles of the kidneys and skin. In the capillary walls fat-granules were aggregated in greater numbers. The relative size of the different parts of the vessels are given below.

Relative width of vessel.	Of channel.	Of walls.
15 ...	5 ...	10
13 ...	5 ...	8
15 ...	6 ...	9
12 ...	5 ...	7
30 ...	10 ...	20
19 ...	7 ...	12
25 ...	10 ...	15
28 ...	12 ...	20
15 ...	5 ...	10
9 ...	3 ...	6

The outline of nearly all the minute arteries were unusually clearly defined. The channels of the thickened arteries were not appreciably encroached upon.

CASE 14.—George T—, æt. 67, died of cerebral hæmorrhage. We regret that we could not obtain detailed particulars of this case.

Many of the arterioles were very much thickened outside the muscular layer by a hyalin-fibroid substance. Amongst this new material were a number of indistinct corpuscles or nuclei which at first sight looked like muscular tissue, but the muscular layer was observed to be entirely distinct from those imperfectly formed elementary bodies.

The relative width of vessel.		Of channel.		Of walls.
24	...	9	...	13
22	...	7	...	15
57	...	21	...	36
38	...	9	...	30
57	...	21	...	38

CASE 15.—Charlotte A—, æt. 55. The kidneys were coarse on the surface, they were also wasted. They weighed $7\frac{3}{4}$ oz. Heart weighed 1 lb. $1\frac{3}{4}$ oz.; its left ventricle was dilated and hypertrophied. Many of the arterioles of the pia mater were very much thickened, and the coat outside the muscular layer was much increased by hyalin-fibroid formation. The new tissues in some of the thickened vessels were coarsely fibroid. The muscular nuclei were much altered in some of the arterioles.

The relative width of the vessel.		Of the channel.		Of the walls.
36	...	16	...	20
32	...	12	...	20
28	...	11	...	17
36	...	16	...	20

CASE 16.—James B—, æt. 48. Cause of death was dilatation of the left ventricle. The kidneys weighed $12\frac{1}{2}$ oz., they were very granular, and contained a few cysts. The cortical parts were very little contracted. Liver capsule was thickened. Heart weighed 1 lb. 8 oz., and the left ventricle was much dilated and hypertrophied. Very many of the arterioles

of the pia mater were very much thickened, and their outer coats were much increased by the formation of hyalin-fibroid substance. The tunica intima in some arterioles was also much increased in size by the formation of hyalin-fibroid substance, and the larger capillaries were thickened by homogeneous or granular material.

In some of the arterioles the new tissue was very distinctly fibroid, and the hyaline appearance was not well marked. The muscular nuclei were wasted in some parts of these vessels, and the muscular layer seemed thicker than natural in other parts.

The relative width of the vessel.		Of the channel.		Of the walls.
32	...	11	...	22
33	...	8	...	22
33	...	8	...	25

CASE 17.—A man, æt. 64, had a cyst in the brain, died in the Whitechapel Workhouse. The kidneys were markedly granular, but not much contracted. The left ventricle was hypertrophied and dilated. For these particulars we are indebted to Dr. Hlott, the resident Medical Officer of the Whitechapel Workhouse.

The arterioles in the pia mater were very much thickened, and the coat outside the muscular layer of these vessels was greatly increased by fibroid changes. This condition was very well marked in many of the arterioles, and the muscular nuclei were very distinct and clearly defined in some, and a good deal wasted in others.

The relative width of vessels.		Of channel.		Of walls.
18	...	6	...	12
36	...	13	...	23
36	...	14	...	22
42	...	15	...	27
31	...	9	...	22
36	...	14	...	22
35	...	11	...	24
30	...	8	...	22
26	...	8	...	18

CASE 18.—James B—, æt. 50, died of acute nephritis; the kidneys were large and their surfaces smooth; were very much congested and mottled; some gray material was scattered amongst the highly congested tissue. The cortical portions had a similar appearance, and they were increased in size. The kidneys had the morbid appearances commonly seen in an early stage of acute nephritis. *Heart*.—The left ventricle was much dilated and hypertrophied, and its valves were healthy. Many of the arterioles of the pia mater, especially the smaller ones, were very much thickened, and their outer coats increased in size. Some of the vessels were not uniformly thickened, but in parts only. The muscular nuclei were in some of the arterioles very indistinct and in other parts absent; they were replaced by a granular substance; outside the muscular layer there was a quantity of hyalin-granular or hyalin-fibroid substance which caused the thickening. The walls of the larger capillaries were infiltrated with a similar hyalin-granular substance; many capillaries were in this manner diseased.

Relative width of vessel.	Of channel.	Of walls.
19 ...	6 ...	13
20 ...	7 ...	13
32 ...	11 ...	22
21 ...	8 ...	13
27 ...	9 ...	18
40 ...	13 ...	27

CASE 19.—Samuel C—, æt. 62, died suddenly in a comatose condition. The brain was considerably wasted. The cerebral arteries were very atheromatous. Spleen weighed 4 oz., liver 2 lb. 11 oz. The kidneys weighed 8 oz., and with the exception of the capsule being adherent they were normal. These organs were very carefully examined by Dr. Hughlings Jackson and Dr. Sutton, and they both agreed that they presented no other signs of disease. The heart weighed 15 oz., it was much increased in size, owing to dilatation and hypertrophy of the left ventricle. Its valves were

healthy. Lungs were emphysematous. Many of the arterioles of the pia mater were very much thickened, the layer outside their muscular tissue was much increased in size. This was due to a fine granular transparent substance. This new material was seen between the muscular nuclei and the coarser fibres of the tunica adventitia. The larger capillaries were also much thickened, and their walls had a granular or homogeneous appearance.

Relative width of vessel.		Of channel.		Of walls.
40	...	16	...	24
42	...	14	...	28
42	...	14	...	28
60	...	28	...	32
54	...	24	...	30
54	...	22	...	32
50	...	20	...	30
28	...	10	...	18
30	...	10	...	20
30	...	12	...	18
34	...	14	...	20
40	...	16	...	24
40	...	16	...	24
26	...	8	...	18
28	...	12	...	16
30	...	10	...	20
32	...	12	...	20
22	...	8	...	14
22	...	8	...	14
24	...	8	...	16

CASE 20.—William H—, æt. 51. There was softening of the cerebellum. The kidneys weighed $11\frac{3}{4}$ oz. They were granular, and they contained the remains of the two embolic blocks, otherwise they were healthy. The heart weighed 1 lb. 9 oz. All its cavities were dilated and the ventricular walls much hypertrophied; some vegetations on the aortic valves.

Many arterioles were very much thickened, and the layer outside the muscular nuclei was much increased in size. In this part a quantity of hyalin-granular and finely marked fibroid substance was seen. The walls of some of the arterioles were unequally thickened. The tunica intima was also

thicker than natural, owing to the formation of a fine fibroid tissue. The muscle of the arterioles was much wasted in some spots, and replaced by bodies which looked like large fat-granules. The larger capillaries were very much thickened, and their walls had a fine granular appearance.

The relative width of vessel.	Of channel.	Of walls.
24 ...	8 ...	16
28 ...	10 ...	18
36 ...	16 ...	20
32 ...	12 ...	20
72 ...	18 ...	54
72 ...	16 ...	56
20 ...	8 ...	12
20 ...	8 ...	12
24 ...	8 ...	14
38 ...	16 ...	22
40 ...	16 ...	24
44 ...	18 ...	26
44 ...	16 ...	28
32 ...	12 ...	20
30 ...	12 ...	20
30 ...	10 ...	20

CASE 21.—William S—, æt. 56, died on Nov. 24th, 1871.

Autopsy showed that the heart was enlarged; it weighed 19 oz. The right ventricle was much dilated and the left ventricle was also dilated and hypertrophied. The lungs were emphysematous. Liver was in an early condition of cirrhosis. Kidneys weighed 11 oz., their surfaces were granular. One kidney was much contracted, the other rather larger than natural. There were indications also of recent acute nephritis.

Microscopical examination of the pia mater showed that many of the arterioles were healthy, excepting that their outline was much more sharply defined than natural. Some of the arterioles were much thickened, and this was due to the increased size of their outer coat. The new formation in this part had a very fine hyalin-fibroid appearance. Some

of the larger capillaries were very much thickened and their walls were granular and translucent. A quantity of fat-globules were aggregated together in the coats of the minute vessels.

Diameter of the vessel.		Of the channel.		Of the walls.
16	...	5	...	11
22	...	7	...	16
40	...	15	...	25
23	...	10	...	13
32	...	17	...	15
36	...	20	...	16
26	...	13	...	13

CASE 22, a patient under the care of Dr. Hughlings Jackson, died in the Epileptic Hospital in Nov. 1871.

We are indebted to Dr. Jackson for kindly affording us the opportunity of examining the pia mater and kidneys. The left ventricle of the heart was much hypertrophied. The kidneys were very granular and their cortical parts were very much contracted.

Many of the arterioles of the pia mater and kidneys were very much thickened. The layer outside the muscular nuclei was much thicker than natural, and this was due to the formation of hyalin-fibroid substance. The muscular tissue was not appreciably increased, but it was wasted in parts, and in some spots the muscular nuclei were entirely absent. The outer layer of the larger vessels was also thickened by a similar fibroid change.

Diameter of the vessel.		Of the channel.		Of the walls.
32	...	12	...	20
28	...	11	...	17
22	...	8	...	14
26	...	9	...	17
35	...	14	...	19
48	...	25	...	23
52	...	25	...	27
33	...	10	...	23
27	...	10	...	17
24	...	9	...	15
22	...	8	...	14

CASE 23.—Edward S—, æt. 46. Autopsy, February 22nd, 1872 (p. 133 post-mortem register for 1872). The brain was healthy; lungs were œdematous. Heart weighed 1 lb. 15 $\frac{3}{4}$ oz.; its left ventricle was very much dilated, and its wall much hypertrophied; valves and orifices healthy. Right ventricle and auricle were also dilated. Liver and spleen presented no noticeable change. Kidneys—their surfaces were granular and mottled. A quantity of grayish material was seen amidst highly congested tissue. The appearances were such as are usually considered to indicate acute nephritis. The cortical parts were very much contracted, so that the tubes in part almost reached the surface of the kidney. The cortex also mottled like the surface. The arterioles of the pia mater were healthy. They were very carefully examined, and they presented none of the hyalin-fibroid changes seen in other cases, nor were there any indications of thickening. The arterioles of the skin, stomach, and kidneys, were greatly thickened by the formation of hyalin-fibroid material.

CASE 24.—John J—, æt. 37 (see post-mortem register for 1872, p. 237). Autopsy, April 22nd, 1872. The lungs were congested and œdematous. There was evidence of recent pericarditis. The heart weighed 1 lb. 2 $\frac{1}{4}$ oz.; its left ventricle was much hypertrophied; its valves and orifices were healthy. The right ventricle was also greatly dilated. The capsule of the liver was slightly thickened; this organ was otherwise healthy. Spleen—its capsule was also slightly thickened, but its substance apparently healthy. Stomach was healthy. Kidneys were very granular, mottled on their surface, and grayish material scattered here and there; the latter apparently the product of acute nephritis. The cortical parts were much contracted. The arterioles of the skin were very much thickened by fibroid material. This new formation was seen outside the muscular layer. The fibroid formation had a coarse fibre-like appearance. The arterioles of the retina

were also thickened by the formation of a hyalin-fibroid material.

CASE 25.—Elizabeth L—, æt. 42. Autopsy, April 2nd, 1872 (see post-mortem register for 1872, p. 201). A very large blood-clot was found in the left hemisphere, outside the optic thalamus. There was a small clot in the pons varolii, and a cyst in the right hemisphere. The left ventricle of the heart was considerably hypertrophied. Heart's valves and orifices healthy. The lungs, liver, and spleen were natural. The kidneys were very granular, small, and their cortex was much contracted. Many of the minute arterioles were thickened, some of them very much so by hyalin-fibroid substance outside the muscular layers. In a great many of the arterioles and capillaries a quantity of fat-granules were aggregated together.

Besides the above cases in which the arterioles were very much thickened we examined ten other cases in which some of the arterioles of the pia mater were thickened, though to a much less extent. In these cases the morbid condition appeared to be in a much earlier stage, but the histological changes were similar in kind though less in extent to those observed in the very thick vessels.

CASE 26.—Elizabeth R—, æt. 44. The kidneys weighed 8 oz.; they were slightly granular and contained cysts. The lungs were extremely emphysematous. The brain was atrophied. The heart weighed 10½ oz.; its left ventricle appeared to be slightly dilated, and there was atheromatous disease of the aortic valves. The outer layers of a few of the arterioles were thicker than natural owing to the formation of a hyalin-fibroid tissue, and this new formation was seen outside the muscular nuclei.

The width of the arterioles.		Of its channel.		Of the sides or walls.
30	...	18	...	12
30	...	19	...	11
45	...	28	...	15
22	...	10	...	12
A 25	...	10	...	15
B 22	...	9	...	14
24	...	16	...	8
14	...	8	...	6

CASE 27.—John S—, æt. 74, died of broncho-pleuro-pneumonia. The lungs were emphysematous. The liver atrophied. The spleen was also atrophied; it weighed only $4\frac{1}{2}$ oz. The kidneys were slightly granular, and not contracted; they weighed 15 oz. The increased weight was apparently due to venous congestion. The heart weighed 15 oz. There was no valvular disease, and the left ventricle was dilated and somewhat hypertrophied. A few of the arterioles were markedly thickened.

The width of the arterioles.		Of the channel.		Of the sides or walls.
A 10	...	3	...	7
B 13	...	5	...	8
C 57	...	24	...	33
D 32	...	10	...	22
12	...	6	...	6
14	...	6	...	8
10	...	4	...	6

The increased size of the arterial walls was due to the formation of a hyalin-fibroid tissue in the outer coat of the vessels. There was no evidence of muscular hypertrophy.

CASE 28.—A man, æt. 60, died in the London Hospital August 28th 1871, of epithelioma of the œsophagus. The heart was moderately dilated. Some of the arterioles of the pia mater were thickened by the formation of fine fibroid-looking tissue in their outer coats. There was no muscular hypertrophy; the walls of some of these thickened vessels were very irregular in size. The measurements are given, and the letters A B C D E F indicate the thickened ones.

By far the greater number of the arterioles were not thickened. A large number of fat-granules were aggregated in the walls of the minute vessels.

The width of the arterioles.	Of its channel.	Of the sides or walls.
A 31 ...	11 ...	20
B 32 ...	11 ...	23
C 26 ...	7 ...	19
D 25 ...	15 ...	20
E 20 ...	8 ...	12
F 28 ...	10 ...	18
35 ...	18 ...	17
19 ...	18 ...	11
19 ...	18 ...	11
40 ...	21 ...	19
22 ...	12 ...	10
30 ...	14 ...	16

CASE 29.—James C—, æt. 74, died in the London Hospital with disease of the knee-joint November 25th, 1871. The autopsy showed that the heart's muscle had undergone fatty degeneration. The condition of its cavities is not mentioned in the post-mortem record. The lungs were emphysematous; the arteries were very atheromatous; the renal capsules were adherent and the kidneys contained cysts; the outer coats of some of the arterioles were thickened by the formation of some coarse-looking fibroid tissue. The measurements of the thickened ones were as follows:

Width of the arteriole.	Of the channel.	Of the sides or walls.
19 ...	7 ...	12
13 ...	8 ...	8
17 ...	7 ...	10

CASE 30.—George G—, æt. 57, died of acute peritonitis following hernia. Autopsy July 12th, 1871. The spleen was atrophied, it weighed $3\frac{1}{2}$ oz. only. Liver atrophied and weighed 2 lb. 11 oz.; the kidneys weighed 11 oz.; they were slightly granular, otherwise natural; the heart weighed 12 oz. A few of the arterioles were slightly thickened in their outer coats by the formation of a fine hyalin-fibroid

tissue; by far the majority of the arterioles were not thickened. The measurements and letters A B show the proportions of the slightly thickened arterioles, the remaining measurements show no evidence of thickening.

Width of the arteriole.		Of its channel.		Of its sides or walls.	
A	39	...	16	...	23
B	46	...	21	...	25
	31	...	15	...	16
	47	...	27	...	20
	38	...	22	...	16
	51	...	28	...	23
	38	...	27	...	11
	18	...	9	...	9
	10	...	5	...	5
	14	...	7	...	7
	11	...	6	...	5

CASE 31.—A man, aged about 45, died of acute pneumonia. The kidneys were healthy, and the heart weighed 14 oz. Its left ventricle was slightly dilated. None of the arterioles were greatly thickened, but the walls of some of them were more sharply defined than natural, and the outer layers of a few arterioles were somewhat thickened and had a fine fibroid appearance. The muscular nuclei were indistinct in many of the vessels. The measurements given show the amount of thickening:

Width of the vessel.		Of its channel.		Of its sides or walls.
42	...	19	...	23
42	...	20	...	22

CASE 32.—A female, died after removal of cancer in the breast January 20th, 1872. The kidneys were granular, but not markedly wasted. Pericardium was adherent. The left ventricle of the heart appeared somewhat dilated; its valves and orifices were healthy; its muscle was very fatty. The arterioles of the pia mater were very sharply defined, and their outer layers seemed thickened by the formation of a fine fibroid tissue.

CASE 33.—Robert B—, died December 2nd, 1871, The autopsy showed vesicular emphysema. The heart was normal. The kidneys were large and mottled, they presented the appearances seen in a somewhat early stage of acute Bright's disease. The vessels were, for the most part, healthy; the only noticeable change was that the outlines of the arterioles were more sharply and clearly defined than natural. Only one arteriole was found with its outer layer thicker than natural, and it had a fine hyalin-fibroid appearance, and only one capillary arteriole was observed much thickened; its wall had a granular homogeneous appearance. The measurement indicated by the letter A show the proportions of the thickened vessel; the remaining measurements do not indicate any thickening.

Width of the vessel.		Of its channel.		Of the sides or walls.
A 22	...	8	...	16
32	...	10	...	12
34	...	20	...	14
42	...	25	...	17
44	...	26	...	18

CASE 34. — William W—, æt. 63, died of cerebral hæmorrhage November 11th, 1871. The heart weighed 13 oz.; its left ventricle was somewhat dilated. There was no valvular disease. The kidneys weighed 10½ oz., and they were healthy. The spleen was wasted. The outer layers of some of the arterioles were slightly thickened by the formation of a fine fibroid tissue. This was most distinctly seen outside the muscular nuclei. In no place were the arterioles greatly thickened. The measurements given show the thickening was not great.

Width of the arteriole.		Of the channel.		Of the sides or walls.
24	...	10	...	14
16	...	8	...	8
28	...	12	...	16
36	...	14	...	22

CASE 35.—Chas. M—, æt. 49. Autopsy December 9th, 1871; cause of death contusion of brain, the resul of injury.

Heart weighed 14 oz. Vegetations were seen on the aortic valves. Liver was congested, and weighed 3 lbs. 2½ oz. Kidneys weighed 9 oz., their surfaces were slightly granular and cystic; cortical substance small. Some of the arterioles were thickened in their outer layers by the formation of a fibroid substance. The measurements given show the degree of thickening.

Width of the vessel.		Of its channel.		Of its sides or walls.
46	...	16	...	30
37	...	18	...	19
72	...	26	...	50

Cases in which the minute arteries and capillaries were healthy, but there were fat-granules, so called, in the walls of some of the vessels.

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles, made from a scale divided into $\frac{1}{100}$ of an inch.			
							Diameter of arterioles.	Of channel.	Of walls.	Of lumen.
36	July 26th, 1871	Sarah N—	F.	...	So-called catarrhal pneumonic phthisis, or broncho-pneumonic phthisis; lungs very much diseased; other viscera healthy, excepting intestines	The muscular nuclei were very distinct and stained very well; in the minute arterioles they almost touched the outer edge of the tunica adventitia; not the least evidence of thickening	$\frac{9}{100}$ 14 18 29 17 33 31 36 17 13 21	$\frac{1}{100}$ 8 10 13 8 21 19 22 8 7 13	$\frac{1}{100}$ 6 8 16 9 12 12 14 9 6 8	$\frac{1}{100}$ 6 8 16 9 12 12 14 9 6 8
37	Sept. 19th, 1871	Sarah S—	F.	51	Acute tuberculosis, capillary bronchitis, kidneys healthy, excepting tubercle in them; heart healthy	The muscular nuclei were distinct, not well stained; in the minute arterioles they almost touched the outer edge of the tunica adventitia; and the vessels were not thickened	34 30 34 25 36 44 26 23 19	16 15 15 10 18 27 14 12 10	18 15 19 15 18 17 12 11 9	18 15 19 15 18 17 12 11 9
38	Nov. 27th, 1871	John B—	M.	22	Acute tuberculosis; tubercular meningitis; lung studded with tubercle cavity at one apex; liver and kidneys contained tubercle	The arteriole appeared to be healthy; and the muscular nuclei appeared to extend, or almost, to the outer edge of the tunica adventitia; vessels were not thickened	24 22 26 20 20 18 16 ...	12 12 10 12 10 10 8 ...	12 12 6 8 10 8 8 ...	12 12 6 8 10 8 8 ...

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles made from a scale divided into $\frac{1}{100}$ of an inch.		
							Diameter of arterioles.	Of channel.	Of walls.
39	Nov. 28th, 1871; page 474 of p.-m. record for 1871	Julia P—	F.	32	A large blood-cyst in the spleen, which had burst, and some of its contents had escaped into the peritoneal cavity and set up acute peritonitis; kidneys were faintly granular, and slightly contracted; heart weighed eight ounces; muscular tissue, valves, and its cavities, were healthy; liver adherent to cyst, otherwise healthy; brain healthy	There was no evidence of thickening, and no appearance indicating a cavity apart from the tunica adventitia; the muscular nuclei in the arterioles almost touched the outer edge of the tunica adventitia, and they were very distinctly seen; a few aggregations of fat-granules were seen in the walls of the vessels	24 22 20 18 20 22 20	14 12 12 10 12 10 10	10 10 8 8 8 12 10
40	Nov. 22nd, 1871, page 470 p.-m. record	James F—	M.	22	Fractured cervical vertebrae; spinal cord crushed; lungs much congested; pericardium healthy; heart, liver, spleen, brain, kidneys natural	The arterioles appeared to be healthy; the muscular nuclei almost touched the outer edge of the tunica adventitia; the latter layers in some of the vessels had a very distinct fibrous appearance; there was no evidence to show that the vessels were thickened	28 28 26 22 26 32 28 30	14 16 12 14 14 16 14 16	14 12 14 9 12 16 14 14
41	Nov. 20th, 1871, page 454 p.-m. record	Fredrick S—	M.	26	Pericardium not adherent; heart weighed 1 lb. 3 oz.; left ventricle much dilated; aortic valves, when tested by pouring water in the aorta, were found to be incompetent; lungs were studded with apoplectic masses; liver slightly cirrhotic; spleen firm and congested; also intestines and kidneys were congested	The arterioles appeared to be healthy; the muscular nuclei almost touched the outer edge of the wall of the vessel; in other parts there was a thin layer of tunica adventitia intervening; the muscular layer was everywhere very distinct; the tunica adventitia was not increased; there were a few fat-granules aggregated in the walls of the vessels	32 32 32 32 30 36 36	20 18 18 18 14 24 22	12 14 12 14 16 12 14

42	Nov. 15th, 1871	...	M.	...	Fractured pelvis; lacerated liver, spleen, kidneys, and diaphragm	Arterioles appeared to be healthy; there were no indications of thickening; muscular nuclei distinct, and they almost reached the outer edge of the wall of the vessel	18 18 28 32 32 42 44	12 12 20 18 20 24 26	6 6 8 14 12 18 18
43	Sept. 21st, 1871	William S—	M.	27	Caries of the right ileum; kidneys and heart were healthy	The transverse muscular fibres of the arterioles had undergone fatty degeneration; they were studded with fat-granules; in other respects these vessels were healthy; no indication of thickening	38 39 18 50 29 35 34 22	24 20 9 29 20 16 12	14 19 9 21 13 18 10
44	Oct. 12th, 1871	Jane G—	F.	20	Gliomatous tumour in the cerebellum; the other viscera were healthy	These arterioles appeared to be healthy, and there were no indications of thickening, except in one capillary arteriole; the specimen was very carefully examined, and only one vessel was found in this condition; some fat-granules were aggregated in the walls of some of the vessels	56 29	36 15	20 14
45	March 30th, 1872	George W—	M.	52	Autopsy; London Hospital; there was medullary cancer of stomach, colon, peritoneum, pleura, and of abdominal and thoracic glands; heart's muscle soft, and apparently fatty, in other respects this organ was healthy; kidneys normal; liver contained cancer	The arterioles of the pia mater were healthy; the muscular nuclei were well stained, and were lying close to the outer edge of the arteriole wall; no hyalin-fibroid changes	...	...	...
46	...	...	...	...	Fractured vertebrae; spinal cord crushed	The arterioles of the pia mater were healthy; no indications of thickening; muscular nuclei were distinct; some of the arterioles were injected with Beale's blue injecting fluid	66 66 52 54 48 44 42 40 48	44 40 30 34 26 24 24 24 26	22 20 20 20 22 20 18 16 20

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles, made from a scale divided into strength of an inch.		
							Diameter of arterioles.	Of channel.	Of walls.
47	Nov. 18th, 1872	Thomas M—	M.	39	Syphilitic changes in liver; spleen, acute pleurisy; heart, left ventricle natural, vegetations on aortic valves, the remaining valves and the orifices were natural; kidneys and brain were natural	The vessels were apparently healthy, with the exception of fat-granules being aggregated here and there; no indications of thickening; no hyalin-fibroid change	39 38 28 24 24 26 14 18	20 26 14 13 12 16 10 10	19 17 14 10 12 10 6 8
48	Nov. 18th, 1871	David E—	M.	32	Death due to the administration of chloroform; viscera healthy; muscle of heart soft, and its left ventricle slightly dilated; heart weighed 14½ oz.	Arterioles presented no abnormal appearances; no indications of hyalin-fibroid changes	22 38 26 19	12 17 14 11	10 15 12 8
49	March 7th, 1872	...	...	17	Autopsy: viscera healthy; death following injury to right hand and arm	Arterioles of pia mater were healthy; no indications of hyalin-fibroid changes	...	...	...
50	March 5th, 1872	William E—	M.	35	Death due to poisoning by corrosive sublimate; there was cedema of larynx and false membrane on the mucous surface of trachea; mucous surface of large intestine congested; rest of viscera healthy	Arterioles of pia mater were healthy; their muscular nuclei were well stained, and lying near the outer edge of the arteriole wall; no hyalin-fibroid changes	...	...	...
51	Jan. 27th, 1872	Ann C—	F.	25	Autopsy: death due to bronchitis; heart; right ventricle much hypertrophied and dilated; pericardium healthy	Arterioles of the pia mater were healthy, they presented no hyalin-fibroid appearances; muscular nuclei	...	...	...

52	Nov. 21st, 1871, page 466 p.-m.-re- cord	F.	51	dium thickened; lungs bound down by old adhesions; remaining viscera healthy	The arterioles were apparently and studded with yellow markings indicative of fatty degeneration, defined, and there was no evidence of valves and orifices natural; liver-thickening or of hyalin-fibroid capsule thickened; spleen normal; change; the muscular nuclei almost kidneys pale, otherwise they were touched the outer edge of the walls of normal; intestines and other vis- cera healthy	24 22 36 38 36 36	12 12 22 22 22 20	12 10 14 16 14 16	...	...	...
53	Feb. 21st, 1872	M.	56	Autopsy showed acute endocar- ditis; left ventricle and muscle of heart dilated; disease of the aortic valves; acute pneumonia and pleu- risy; morbid changes in the kid- neys, indicating acute nephritis	There were no indications of thickening in the vessels of the pia mater; the nuclei of the muscular fibres almost touched the outer edge of the arterioles, and there was in consequence very little appearance of tunica adventitia; there were no aggregations of fat-granules	38 44 27 31	23 25 15 16	16 19 12 15	...	...	...
54	Jan. 24th, 1872	F	43	Autopsy showed broncho-pneumo- nic phthisis	The arterioles were healthy; there was no evidence of thickening; the muscular nuclei were seen close to the outer edges of the walls of these vessels; there were no large aggrega- tions of fat-granules	29 44 16	17 28 9	12 16 7	...	...	...
55	Dec. 4th, 1871	M.	62	Stricture of the urethra; fibroid degeneration in the lungs; dilated right ventricle of heart; liver cir- rhosed; hypertrophied brain	The arterioles were healthy; the muscular nuclei were very well stained, and they were seen lying close to the outer edge of these vessels; there were no aggregations of fat-granules	50 42 40 40	28 23 22 22	22 19 18 18	...	...	...

The arterioles, in some cases, were free from thickening, and the heart was not hypertrophied; whilst there was chronic disease of the kidney, viz., large white kidney and so-called scrofulous pyelitis.

CASE 56.—This occurred in a patient named William E—, æt. 33. He was in the London Hospital many weeks with great and general œdema, and the urine contained a large quantity of albumen. While in the hospital he had attacks of great dyspnœa also of vomiting; and a week or two before he died he had convulsions alternating with coma.

The autopsy showed that the kidneys were larger and much heavier than natural. Their capsules separated readily; their surfaces were smooth and white, and very few vessels were seen. The kidneys had the peculiar, general, marbled-white appearances so well represented by Dr. Bright in one of his drawings of Bright's disease. The cortical parts were much increased in size and appear to be infiltrated with similar white material. Heart was normal. The arterioles of the pia mater were not thickened.

CASE 57.—In the case of a man, who died in the London Hospital under the care of Dr. Andrew Clark, there was general œdema; the urine contained a large quantity of albumen, and there were symptoms of uræmic poisoning. The kidneys were in the morbid condition known as "large, white, and mottled." Heart was normal. The arterioles of the pia mater were carefully examined, and there were no indications of thickening.

CASE 58.—In a third case a girl, aged 7 years, who died in the London Hospital, under the care of Dr. Down, February, 1872, there were large white kidneys and general œdema. Heart was natural. The arterioles were not thickened. The disease in this case was of considerable standing, for the patient had been under Dr. Down's observation with albuminuria for more than twelve months, and she was in the London Hospital with kidney disease on more than one occasion.

CASE 59.—A fourth case was that of George G—, æt. 18. He was in the London Hospital several weeks with general œdema and a large quantity of albumen in the urine. He died with symptoms of uræmic poisoning. The autopsy on May 6th, 1872, showed the kidneys weighed $14\frac{3}{4}$ oz. They were much larger than natural and their capsules separated readily; their surfaces were smooth and contained no cysts. They were of a pale fawn colour, and a large number of yellow spots were seen scattered in the cortical parts. The cortex was greatly increased in size and converted into similar fawn-coloured substance. These organs were good examples of large white kidneys in an advanced stage of the disease. Heart weighed 8 oz., there was no dilatation or hypertrophy; its valves and orifices were healthy. Liver, spleen, and lungs were healthy. The walls of the arterioles were not thickened. In the minuter ones the muscular nuclei were situated close to the outer edges of the vessel.

CASE 60.—A fifth case was that of William L—, æt. 27, who died in the London Hospital. It was stated that he had been under the care of Dr. Thorowgood, at the Victoria Park Hospital, for many months with symptoms of phthisis and strumous pyelitis. The capsules of the kidneys were very much thickened, and the right kidney was adherent by old tough fibrous bands to the liver and intestines. On section, this kidney seemed almost completely destroyed. It contained several cavities filled with thick puriform matter. There was very little kidney structure remaining. The left was similarly affected, but not to the same great extent. Heart healthy.

The arteries of the pia mater were injected with Beale's Prussian blue solution and afterwards carefully examined by aid of the microscope, and there were no indications of arterial thickening.

In other cases (see case No. 13, James P—, æt. 49) the kidneys were large, white, and mottled, the arterioles were thickened by hyalin-fibroid changes, and the left ventricle of the heart was dilated.

In the latter case we cannot conclude that the kidney disease induced the morbid changes in the arterioles and heart; for there is no evidence to show that the cardio-vascular changes did not precede the renal disease; and the case of James B—, æt. 50 (see Case 18), tends to show that the cardio-vascular changes may be the primary and antecedent disease, and the renal changes subsequent. In this case the kidneys were highly congested and mottled, and a quantity of grayish-looking material was lying amongst this highly congested tissue. These kidneys were large and their cortex was much increased in size, and their surfaces were smooth. The morbid appearances were such as are usually seen when death has occurred in an early stage of acute nephritis, and they appear to show that the kidney disease had probably set in a few weeks only before death. The left ventricle of the heart was much dilated and hypertrophied, and many of the arterioles and capillaries were greatly thickened by the hyalin-fibroid changes. The cardio-vascular disease had, therefore, evidently preceded the acute renal changes.

The arterioles are sometimes much thickened, whilst there is no kidney disease or very little morbid changes in the kidneys. In support of this statement we may first mention the case of John C—, æt. 34, who died of aneurism of the aorta. His kidneys were carefully examined, and they presented no signs of disease. The arterioles of the pia mater were much thickened by the formation of fibroid material external to the muscular nuclei. The kidneys were healthy and the arterioles thickened in the case of Sarah S—, æt. 63 (see Case 1); also in the case of Samuel C—, æt. 62 (see Case 19); also in the case of James D—, æt. 77 (see Case 3); also in Case 2.

In other cases there was little morbid change in the kidneys, no morbid change to indicate that their excretory power had been greatly diminished; whilst the heart was much hypertrophied and the vessels much thickened. This is shown in cases 7, 10, and 20.

DESCRIPTION OF PLATE V.

- Fig. 1.** The microscopical appearances seen in very granular contracted kidneys. (From a girl aged nine years.)

The letter **A** points to six Malpighian bodies which are lying clustered together, and surrounded by a quantity of fibroid material. These bodies contain numbers of fat and other granules. A convoluted tubule indicated by **D** is seen coming down from one of the Malpighian bodies, with its epithelial cells destroyed. The letters **B** mark tubules which are lined with epithelial cells for the most part healthy. **C** points to isolated wasted tubules containing shrivelled, ill-defined, epithelial cells, and in some, in place of epithelium, nothing but fat-granules remain.

- Fig. 2.** Arteriole much thickened by coarse fibroid changes outside muscular layer; tunica intima thickened also.
- Fig. 3.** Tunica adventitia and intima thickened by fibroid changes.
- Fig. 4.** Arteriole of the pia mater. Fibroid changes outside the muscular nuclei.

X 750.

DESCRIPTION OF PLATE VI.

- Fig. 1. A capillary of the pia mater much thickened by a homogeneous hyalin substance.
- Fig. 2. Capillary very much thickened by a granular substance.
- Fig. 3. Hyalin-fibroid thickening outside the wasted muscular layer.
- Fig. 4. The arterioles in the choroid greatly thickened by the formation of fibroid material outside the so-called muscular nuclei. The dark parts represent choroid pigment.
- Fig. 5. Arteriole of skin thickened by fibroid formation outside muscular layer.
- Fig. 6. Arteriole lying amongst muscular tissue of heart greatly thickened by the fibroid material.
- Fig. 7. Minute artery of the kidney greatly thickened by hyalin-fibroid changes in the outer layer of the vessels.