ON
SUB-ACUTE INFLAMMATION OF THE
KIDNEY.

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It is not my intention in the present paper to enter into unnecessary details respecting a disease, with the general features of which it may be presumed that all members of this learned Society are sufficiently conversant. Merely glancing at such portions of the subject as are already familiar to the Profession, I shall endeavour rather to fix attention on changes hitherto little observed; changes which commence in inflammation of the kidney, and tend completely to disorganise it, but which run a slow and insidious progress during life, and often leave in the dead body no such obvious traces as would strike the superficial observer.

The latent processes to which I refer are among the most interesting and curious in the whole compass of pathology; consisting (1) in the more or less complete obliteration of all that is characteristic in the ultimate structure of the kidney; and (2) in the attempt to compensate for this, by the organization of a new secretory apparatus.

An examination of this latter process,—by which (as I shall presently show) vesicles are substituted for the normal tubularity of the gland,—will lead me to explain the origin of

those large renal cysts, so well known in the dead-house ; but first, in order to elucidate that earlier stage of the disease, in which the tubes and Malpighian tufts are destroyed, I shall attempt to define more exactly the nature of nephritis, and shall describe those primary lesions of structure which attend it. Such observations as I have to offer on these topics will be rendered clearer, if I may be allowed to preface them by a few words on glandular inflammation generally.

Many pathologists have inclined to conceive inflammation in glands as in a great degree foreign to the true apparatus of secretion ; they have thought it essential that products of the inflammatory process should have been deposited interstitially to the secreting elements, and should have remained palpable in the rough structure of the organ ; and they would hesitate to use the name of "chronic inflammation" for any other disease than one, where, as in cirrhosis, they might find a hardness to handle, or might point to the supposed maturity of a previous inflammatory infiltration. This view of the case, though substantially correct in respect of the intenser forms of inflammatory action, is too limited and absolute ; for it would probably exclude the entire class of sub-acute glandular inflammations. Organs may, I conceive, rightly be said to suffer inflammation, whenever their natural functions are deranged by an excess of nutrition ; the test of that excess being, that their supply goes beyond their capacity of growth or of secretion. Thus, while we withhold the name of inflammation from the increased nutrition of the pregnant uterus, or of the lactating breast, or of the diabetic kidney, because such increase is commensurate with the necessity, and applied to the purposes, of growth or secretion, we *do* give that name to the case where a superfluous blastema becomes commingled with the secretion, as in active albuminuria, or undergoes interstitial development, as in cirrhosis of the liver ; and in both these instances with equal pathological accuracy.

Inflammations of simple mucous membrane mainly evince themselves in derangement of the secretory functions ; and,

especially if sub-acute, by no means necessarily imply any sub-mucous deposit of organisable material. The modified secretion of such a surface may be eliminated with a rapidity that renders accumulation impossible ; and then (as in gleet or in ophthalmia) how little, beyond a doubtful hyperæmia, would remain for the morbid anatomist !

All this is notoriously true in respect of mucous membranes : it is equally true, though less notorious, in respect of the true glands, which are but processes or involutions of the same structure. No interstitial effusion of lymph need exist in a gland to warrant us in accounting it inflamed ; its inflammation may consist simply in functional derangement, and, during life, may be recognised only by admixing its albuminous products with those of normal secretion.

There is, however, a momentous difference in the two cases. The simple mucous membrane sheds from its surface whatever inflammatory secretions may have formed there ; it is quit of them : they cannot re-act on it injuriously. But the glands have not the same facilities for eliminating morbid products : in proportion to their complexity of structure, and to the narrowness of their canals, they must be liable to embarrassment from changes in the physical qualities of their own secretions ; the delay, accumulation, and obstruent action of which would (it is obvious) suffice to derange the whole microcosm of the organ in which it might occur : and thus an amount of irritation, which on a simple mucous membrane would be utterly insignificant, may, if transferred to a gland, serve to originate its complete disorganisation.

These preliminary remarks will not, I hope, appear to the Society otherwise than appropriate. The influences just alluded to are precisely those which most of all illustrate my subject ; for it is under their operation in the kidney, that the lowest amount of inflammatory action, or even an excitement of function hardly rising to inflammation, may and does, by its secondary and reactive results, become a thoroughly irreparable disease.

Without dwelling on those excessively rare cases, where

idiopathic nephritis (independent of tubercles or of calculus) may, by its mere intensity, have ended in large suppuration or (almost uniquely) in gangrene, I may state that, in an infinite majority of instances, inflammation of the kidneys is sub-acute. It depends on some humoral derangement of the entire system, and commences as functional excitement manifested in an act of over-secretion. The morbid material which thus stimulates the kidney in its struggle for elimination will sometimes consist of products of faulty digestion—the lithates or the oxalates; sometimes of matters cast upon the kidney in consequence of suppressed function in other organs—the skin, or the liver; sometimes will be the mysterious ferment of a fever poison*—typhus, or scarlatina. In these several cases, whatever variety may exist in the detail of their causation, the essential symptoms during life and the essential anatomical changes are strictly identical in kind. They vary only in degree. The *materies morbi* seeks to effect its discharge by means of an increased activity in the secreting functions of the kidney; it stimulates it; and the result of the stimulation is not so much an increase of the watery secretion as it is an augmented cell-growth in the tubules of the gland.† This acceleration of function is incompatible with maturity of the secreted products; the epithelial cells undergo various arrests or modifications of development, and become more or less palpably imbued with evidences of inflammation.

If attention happen to be directed to the state of the urine, that fluid will be found to present manifest signs of derange-

* Perhaps it is not sufficiently known that scarlatina is by no means the only febrile blood-disease which affects the kidney. It is one of many. Becquerel and Franz Simon have frequently found the urine albuminous in cases of small-pox, typhus, and erysipelas. In the first two of these diseases I have repeatedly seen just the same condition of urine as one finds in scarlatina,—the same epithelial and fibrinous casts, &c.; and in the dead body I have noticed the same structural lesions.

† It was conjointly with Dr. Johnson, in the Autumn of 1845, that I made my earliest observations of this peculiar activity. Our joint examinations were referred to in his Paper of that date.

ment. Microscopical examination will show in it numerous nucleated cells, which, in the hurry of over-secretion, have descended from the urinary tubules. Many free cytoblasts will likewise generally present themselves, together with a variety of those indefinite shapes, which are known to the morphologist as abortions of cell-growth, and which constitute a series of connecting forms between the pus-globule and the healthy gland-cell. Mingled with these, in greater or less quantity, will be noticed also those remarkable fibrinous threads first described by Dr. Franz Simon in connection with renal disease. They are seen as exceedingly delicate, almost perfectly transparent and colourless cylinders, often containing in their mass some of the cell-forms just enumerated, or, not unusually, a few blood-discs, resulting from hæmorrhage into the tubules.

On several occasions, where the renal irritation has been gouty, I have seen crystals of lithic acid thus entangled in fibrin: in other cases, though far less frequently, I have distinguished crystals of oxalate of lime similarly enveloped. It is well known that these little cylinders are fibrinous moulds of the inflamed urinary tubules, some of the other contents of which they bring with them in their descent. They are thus quite as characteristic of the disease they attend as croupy expectoration is of tracheitis; and the cells or crystals included in them often afford the most valuable therapeutical indications.

Of course, under all the circumstances last mentioned, the urine contains albumen and will yield a precipitate with nitric acid; but, so much more delicate is the microscopical than the chemical test, that it has twice happened to me lately, in cases of scarlatina, to find groups of blood-corpuscles in the urine when nitric acid had given no precipitate visible to the naked eye.

While the urine is presenting these characters, there co-exists, with such signs of renal disturbance, a remarkable tendency to serous effusion in various parts of the body; and frequently the startling occurrence of inflammatory dropsy

will first draw attention to the primary mischief. The explanation of this concurrence is easy and unquestionable.

If patients chance to die while their urine is first furnishing the signs enumerated, it will often happen that the kidneys in their general appearance present no marked deviation from healthiness. Their cortical substance may indeed show the minute blood-dots of intra-tubular hæmorrhage; or, more rarely, may present here and there a pin-head abscess. But often, perhaps most often, a superficial observer would pronounce the kidneys healthy, and, unless previous knowledge of the albuminuria had existed, they would receive no further attention: or the case-book might contain that vaguest of all vague records—"slight congestion of the kidney."

On minuter analysis, however, the microscope will reveal a large amount of disease. The ultimate tubules are found, as one might anticipate, gorged with an uneliminable excess of crude and vitiated secretion. Blood, and amorphous matter, and an infinite range of cell-growth, from pus-globules to the healthy germination of the gland, present themselves in various combinations; and among them, shape or colour will sometimes enable us to discern the specific cause of the derangement—crystals of lithic acid or of oxalate of lime, or the ochreous tinting of bile. By products such as these the tubes are plugged, irregularly distended, and not unfrequently burst and annihilated. So close is the compaction of material, even in many of those tubes that have no shaped inflammatory products within them, that they are plainly impervious; and it is only by artificial means—by further tearing of the fragment, or by use of chemical agents, that we can satisfy ourselves that the dense plug in question consists but of agglomerated gland-cells.

I have never seen the disease, in the form under consideration, otherwise than double. It varies, of course, materially in respect of intensity, and sometimes will appear to affect with its utmost severity every portion of both kidneys. How gravely it must interfere with the secretion of urine (particu-

larly of its watery part) will be obvious on the slightest reflection, and will sufficiently explain its tendency, so soon as it has made some way, to induce serous effusions, and thus to originate the disease known as inflammatory dropsy.

It is impossible to doubt that many unexplained deaths must have depended on structural changes such as these: the morbid action un-recognised during life, and its traces overlooked afterwards. But, unless the inflammatory process has been universal in both kidneys, and of sufficient intensity to involve the almost complete obliteration of glandular substance, it more usually happens that the patient survives a first invasion of the disease, and his albuminuria becomes chronic, or, under the most favourable circumstances, vanishes.

It can very rarely happen that a patient will emerge from the perils of acute albuminuria, with his kidneys quite free from permanent injury: a certain proportion—perhaps a very small one, but almost certainly some—of the tubular structure will have perished. The former function of this obliterated part has now to be distributed, and discharged vicariously by the rest of the organ. If happily the spoiled tubules are few, their surviving fellows are of course abundantly competent to perform the slight increase of task divided amongst them, and the patient enjoys a complete recovery. But if, on the contrary, the minute structural lesions which I have described, have been diffused in an aggravated form throughout a large portion of both kidneys, so that a great destruction of tubular substance must have occurred,—then it is not so easy for the residual sound texture to compensate for what has perished: it is “working double tides” or something more: it can only do this at its own expense: it finds it a hardship, an inconvenience, an injury. It is under these circumstances, probably, that the symptom of albuminuria becomes chronic. Its persistence depends, no doubt, on the habitual super-stimulation entailed on the surviving structure. The continued over-work is in fact a chronic inflammation.

Under judicious treatment the usual event appears more

satisfactory, even in severe cases: with convalescence the albumen slowly ceases to be perceived in the urine,* and the disease is thought cured. But presently, on some slight provocation of cold, or irregular diet, the symptom re-appears and gives notice of increasing mischief. This recurrent albuminuria is a characteristic and alarming sign.

There are two practical points intimately connected with this part of my subject: first, that the portions of a kidney left uninjured by the acuter attack are, *ipso facto*, maintained in circumstances of abnormal excitement, and thus predisposed to undergo subsequent change; in other words, that the diseased condition tends to spontaneous increase: and, secondly, that renewed exposure to causes of the original disease,—to intemperance, cold, and the like, will enable these influences to tell with a fearful and accumulative advantage on kidneys already in functional embarrassment. I refrain from expatiating on the clinical details of these cases, which are so frequent in professional practice, and, in their severer forms, so well known. I shall merely remark that any difficulty in their diagnosis may be solved by the microscope. Whenever the disease is advancing, the urine will be found to present in a modified and mitigated degree the same inflammatory products as in the sub-acute attack—the same epithelial cells (more frequently in an immature form), the same occasional (but far less plentiful) presence of the blood corpuscles, and of the fibrinous casts of the urinary tubules.

Thus—with occasional great exacerbations, and with un-failing smaller steps of progress,—the disease may go on, even for years, before continued dropsy and the well-known signs of urinous poisoning give notice of its near termination. But those who are familiar with the course and complications of renal disease will easily believe that a large proportion of

* It is quite certain that the symptom of albuminuria may be absent for some time (probably while the disease is quiescent) even though the kidney be very extensively disorganised.

the patients, whose case I am considering, must die with obscure symptoms not referred to the urinary organs; often from double pneumonia, or from peritonitis, or from encephalic effusion, which is perhaps considered idiopathic, when in fact it has arisen in the non-elimination of urea; and then, on *post-mortem* inspection, the kidneys, often presenting no glaring morbid appearances, are wrongly accounted healthy, and the patient's death is ascribed to a disease only secondary on the renal one.

Now in the *post-mortem* examination of these chronic cases, we may or may not find the kidneys materially contracted and deformed. It happens, to say the least, *very* frequently that the organ has preserved its full size and presents the ordinary colours. Perhaps it may have a cyst or two on its surface. Between such kidneys, and those which are all knobbed and puckered and wrinkled,—there is not the essential difference which first sight would suggest. I shall first detail the changes which are latent in the healthier looking kidney, and subsequently shall consider the anatomy of the contracted specimens, and analyse the circumstances which determine that apparent atrophy of the gland.

In the first instance, then; In commencing the microscopical examination of the cortical substance, we partially find a similar state of tubes to that described in connection with the sub-acute attack—a state, namely, of unequal distention and of blocking up by their own accumulated products. In the cases which have lasted a long time, these products will often be found to have undergone material alterations, from the combined effects of pressure and absorption. The contents of the epithelial cells will have lost much of their natural fine granularity; so that the cells will appear, even when viewed singly, to have acquired a marked increase of solidity and substance. But—more than this; in many parts hardly a trace of tubularity will be found; the tubes have been burst; their contents have been interfused amid the matrix and blood-vessels; and their débris may be found

on opposite sides of a preparation,—here black and bloated, there pale and collapsed.

Betwixt these trophies of disease there is a new manifestation. The interspace is crowded with a profuse development of cysts, apparently foreign to the healthy structure of the part. They are of all sizes; some are visible to the naked eye; some are of the magnitude of normal gland-cells, $\frac{1500}{10000}$ inch; but the majority are of an intermediate bulk, $\frac{300}{800}$ inch. Even where smallest, they are distinguished by their sharp outline; and the larger ones are conspicuous by their roundness and transparency, for all above $\frac{1}{1000}$ inch have predominantly fluid contents.

To explain this very remarkable phenomenon I take leave to digress for a moment from the straightforward pursuit of the inflammatory changes. Any one who has made a dozen *post-mortem* examinations must have observed cysts in the kidney; and there can be few pathologists who have not speculated on the origin of these growths. Their connection with chronic obstructive diseases of the kidney being notorious, some observers have supposed them to originate in dilatation of the Malpighian capsules; while others have referred them to distention of the urinary tubules. They exhibit great variety in size; they are seen every day as small as mustard seeds; they have been seen as large as cocoa nuts. Thus, they obviously range from a very conspicuous largeness to a size at which the naked eye loses them. On microscopical examination of cysted kidneys, the same uninterrupted gradation of size is seen to repeat itself. The larger vesicles fill the field of the microscope; the smaller ones diminish progressively, so that scores of them may be in the field at the same time.

A section of cysted kidney, carefully examined with a sufficient magnifying power, may show an astonishing number of these minute vesicles; a number quite disproportionate to that of the larger cysts visible to the naked eye; so that sometimes, by a single one of the latter class seen on the surface of the kidney, I have found myself guided to a

disease which is substantially a vesicular transformation of the ultimate structure of the gland. The smallest cysts are simple nucleated cells, of the same size (or rather, within the same limits of size) as the common secretory, or epithelial cells of the gland. From these cells they seem to be distinguished by their very definite outlines, and by their transparent fluid contents: but a step further in microscopical analysis shows that the distinction ceases at this point. They show no signs of a specific origin; no germs can be found for them other than might equally belong to epithelial development; it seems as though from the same germs—according, no doubt, to varying influences—healthy gland-cells might grow, or these fluid-holding cysts.

Fuller investigation of the specimen reveals the following very suggestive fact. The copious formation of cells occupies the place of tubes, holding their relation to the vascular plexus of the gland; and as one gets to the periphery of the portion of gland thus transfigured, one finds the broken extremities of the original tubules,—some empty and collapsed, others obstructed and often dilated with morbid accumulation. In some cases this obstructive material contains a large proportion of fat, or consists of it almost entirely.

In short, in pursuing the minute anatomy of the cysted kidney, we are conducted back to that same structural change which we found in connection with sub-acute nephritis, and demonstrably dependent on inflammatory processes: or sometimes we are led to a change in some respects similar to this, associated with what is known as the mottled condition of the kidney.

The pathology of cysted kidney may accordingly be traced in either of two directions; from its first causation, or from its extreme phenomena. Following the latter course, we have ascended to a period in the history of cysts, in which they lie with numberless gland-germs amid the remnants of broken tubules. The unbroken tubules around show no growth of such cysts in their interior; many are distended, it is true, but not with cysts; their distention is of a kind that we have

already investigated,—inflammatory, or perhaps fatty. From the smallness attained by the cysts, it seems quite obvious to me, that they cannot commence in any transformation of the tubes themselves, or of the Malpighian capsules. Accordingly, I find the same theory suggested by this method of inquiry, as when the morbid change had been traced, descensively, from its causes: viz., that certain diseases of the kidney (whereof sub-acute inflammation is by far the most frequent) tend to produce a blocking of the tubes; that this obstruction, directly or indirectly, produces rupture of the limitary membrane; and that then, what should have been the intratubular cell-growth continues with certain modifications as a parenchymic development.*

The only theoretical alternative lies in the supposition that the cysts may have grown in the tubes (independently of their natural contents), and may, themselves, by their development, have caused the destruction of the limitary membrane. But to this theory there are, as seems to me, two insuperable objections; (1,) the absolute invisibility of any such growth within the tubes, and (2,) the still more convincing fact, that the cyst formation does not occur idiopathically, but only in conjunction with diseases which are in themselves fully adequate to account for the blocking and bursting of the tubes.

During the growth of the cysts, they frequently exhibit an endogenous formation of cells which line them as an epithelium: and in this condition (which commonly does not

* I take this opportunity of remarking that true cystic degeneration does not seem ever to result directly from obstruction in the pelvis of the kidney, or in the urethra, or ureters. I have often seen all the receptacular and excretory parts of the apparatus largely distended, without any material injury to the cortical part of the gland: and even in those cases where long-continued distention of the pelvis and calyces has caused expansion and atrophy of the cortex, the formation of interstitial cysts has not presented itself to me. Rayet has judiciously distinguished these latter cysts from the mere dilatation which attends retention of urine in the excretory parts of the kidney; giving to the latter disease the name of *hydro-nephrosis*.

continue if they become very large) they present a curious similarity to the vesicles of the thyroid gland, and are, no doubt, like these, organs of secretion.

If I am right in my statement of facts, and if my theory of the cyst-growth is sound, then the early stages of the process are certainly points of great interest. For no one accustomed to the interpretation of nature can doubt the reparative tendency of these acts. The effused gland-germs are the last phenomena of the original disease, and the first of the attempted compensation. The transparent nucleated cysts, with their clear sharp outlines, are not mere dropsical epithelia; but are organised for secretion into their own cavities, so as at least to withdraw from the blood, if they cannot eliminate from the body, the materials which fill them.* Nor will it be without interest to the physiologist to remark, as the proximate cause of this peculiar development of gland-germs in the kidney, a modification of structure so slight as the rupture of that limitary membrane, which had heretofore invaginated the cells and had qualified their access of nourishment.

Returning now to the traces of inflammation in an uncontracted kidney, we have yet to ascertain the condition of its blood-vessels. Numbers of the Malpighian bodies are extinct for all purposes of secretion; their vessels obliterated, their capsules wrinkled round them; they are dwindled, opaque, and bloodless. Sometimes the contraction of the Malpighian bodies is secondary on that rupture of their capillaries which Mr. Bowman has indicated as the source of intra-tubular hæmorrhage; which rupture, of course, may have arisen either in an augmented impulse of the arterial stream which fills them, or in an impeded circulation through the venous plexus into which they discharge themselves. But rupture of the capillaries is not the only cause of atrophy, to which these bodies are liable in the disease under consideration. The vascular tufts may be exposed to injurious pressure from

* I may take this opportunity of mentioning that on two occasions I have found the Xanthic Oxide forming a considerable proportion of the contents of a cyst.

materials accumulated in their capsules. Thus, I have seen them flattened into a fourth of their natural compass, while the remaining larger portion of the capsule (probably continuous with an obstructed tubule) has been distended with a colourless and transparent fluid.

Such is the minute anatomy of a kidney which, having suffered from sub-acute inflammation, has undergone in consequence no noticeable alteration of volume, although having in its interior a very considerable new development. Now—if that new development could be suddenly subtracted from the volume of the gland, if the cysts could be withdrawn from the parenchyma, what a collapse there would be! how the meshes of matrix would drop together round the atrophied Malpighian tufts, and round the empty tubules of the cortical substance! The kidney would at once be reduced to half or a third of its previous dimensions. The condition thus suggested to the fancy is not without reality; it is one with which every member of the Society is well acquainted, as the extreme stage of renal degeneration,—the stage in which nearly all the efficient elements of the organ have vanished, and have left no new development in their stead.

If the pathology of the uncontracted kidney be rightly understood, that of the contracted specimen will follow it naturally. It seems to me that, in the mere destruction and absorption of tissues, there is abundant explanation of the shrunken dimensions of a kidney which has passed through inflammatory changes. The tubes have burst, and a great portion of their contents has been removed by absorption; the Malpighian bodies have dwindled to a few: what then remains to make bulk? In the uncontracted specimen a false appearance of size is maintained by the adventitious cyst-growth, which I have described as filling the interstices of the organ. But the cysts are so much over and above the real kidney-structure; and if that succulent surplus could be removed, the result, as I have suggested, would be the falling-together of wasted textures into a comparatively small compass. The cause of shrinking in the gland is the gradual absorption of spoiled material. This cause operates equally

in all chronic cases, and its effects are to be traced in the uncontracted, as in the contracted specimen. The main difference between these two lies in the *more or less* of interstitial cyst-development; the most dwindled are those in which least of the new growth has arisen or has survived.

I see no reason for believing that the interstitial effusion of lymph effects much towards the final contraction of the kidney. There are not wanting, I know, some pathologists who will assert it to be the great agent in the change; and who conceive they have seen the whole process of fibre-formation according to the most approved foreign cell-theories. But I suspect that the observers of new fibre will often have confounded cause and effect. Coincidentally with atrophy of the kidney there occurs a contraction of the reticular matrix; but *that* contraction is probably consequent on a prior absorption of the intervening tissue. The meshes of the matrix come nearer together, and in a given space there is an excess of fibrous tissue, only because the material is withdrawn which originally expanded that matrix through three times the space it now occupies.

I am by no means prepared to affirm that interstitial effusion of lymph and its subsequent organization may not *possibly* occur in the kidney; or that, if it occur, it may not *in any degree* conduce to the resulting atrophy. Indeed, I have seen what I have thought to be interstitial new matter assuming a certain fibrous arrangement; but this has been very rarely, perhaps somewhat doubtfully; and it has always shown itself in a degree far subordinate to the other causes of contraction. Destruction of tubes and Malpighian bodies, with more or less extensive vascular obliteration, is *always* to be observed in specimens of contracted kidney; and I have never seen a degree of contraction in the gland which these changes might not sufficiently explain. I therefore find no reason for assuming the superfluous operancy of an unemonstrated agent.

In respect of kidneys contracted after the inflammatory process, a very interesting question occurs to me;—Have they passed through an uncontracted cystogenous stage?

and, if so, what has become of so many cysts? I am unwilling to speak very positively on this point, but I suspect that the following observations may explain in some degree the relations of the two forms.

In the uncontracted cystogenous kidney, the disease has been of rapid process; for it has usually invaded the greater portion of both glands, and its course has been short in proportion to its intensity. These are the cases in which the phenomena of the disease are best studied: cases in which an infinite number of cysts occur, ranging upwards from epithelium-size to a magnitude visible to the naked eye, and seldom much larger.

If, on the contrary, the original inflammatory attack and the cystic degeneration have been restricted to smaller portions of the gland, and if (as no doubt very often happens in connection with gout or rheumatism) various portions of the gland have been successively invaded, a different result occurs. The vesicles, if placed advantageously near the surface of the organ, may undergo an ulterior and almost indefinite growth, presenting us with the well-known phenomenon of large renal cysts. But, if placed in the interior of the cortical substance, I am disposed to believe that they must in some measure promote its ultimate demolition. For, their pressure on the capillary plexus which surrounds them and which was originally not adapted for such an arrangement, must, of necessity, tend to impede and ultimately to obliterate it; which vascular obliteration would of course entail their own subsequent atrophy and disappearance. I believe that in contracted kidneys something of this sort must have occurred, and that the cyst-growth, except very partially and under very peculiar circumstances, cannot advance beyond the low degree of development witnessed in the uncontracted specimens. This belief is partly founded on, and would partly explain, the fact, that there is a certain inverse proportion* observed be-

* This fact is not to be explained by the supposition of confluence of several smaller cysts to make a larger one; a method of development which I have never noticed, or even seen satisfactory traces of, and which—if it ever occur, must be of the greatest rarity.

tween the *size* of the cysts and their *number* : as if the same length of time which suffices to give bulk and extreme development to a portion of the crop, determines also the abortion of the rest. I may observe too, in further illustration of the same point, that the contracted kidneys—although they present in their interior an infinitely scantier formation of cysts than the uncontracted ones—do yet, even when most atrophied, rarely, if ever, fail to afford some traces of the cystogenetic process.

Accordingly I am inclined to view this process as inseparable from any such sub-acute inflammation of the gland as may terminate in its contraction. But of cysts thus generated few can attain maturity. They are girt with tissues that impede their free expansion, and are fed by a vascular plexus ill-suited to their nourishment. Their growth soon arrests itself by compressing the blood-vessels that supply it ; and thus, while in the slower cases, a few may attain considerable size, the numerical majority will have participated in the general atrophy of the gland.

Up to the present point I have studiously avoided introducing the ambiguous and controversial name of “Bright’s disease.” And now it will probably be asked, what relation to Bright’s disease is borne by the malady I have treated of. Is it the same thing under another name? This question can be answered in a word, only when it shall have been settled what Bright’s disease really is. The history of the complaint, or complaints, included under that title was perhaps originally systematised with too much haste. Starting from dropsy with albuminuria, and noticing that two chief forms of morbid appearance corresponded to that symptom (one, namely, where the kidney was large and mottled,—the other where it was contracted and knobbed, or irregularly granular), pathologists have considered these two forms as representing the extreme stages of one and the same disease.

I must venture to express a doubt as to the justice of this generalisation. After investigating both classes extensively, I am convinced that the *mottled* and the *contracted* kidney do,

in almost every instance, belong to different morbid actions, not to different stages of the same. This opinion I shall very briefly defend.

Last session, in one of the soundest and most ingenious papers ever read before this Society, Dr. Johnson contended that Bright's disease is a fatty degeneration of the kidney. He started from *the mottled kidney*, taking it as, *κατ' ἐξοχὴν*, Bright's disease. Far more fully and conclusively than had hitherto been done, he illustrated the pathological affinities of that disease, and demonstrated its essential connection with the scrofulous diathesis. This view was corroborated by some experiments of my own (which Dr. Johnson did me the honour to quote), in which I had artificially produced a fatty degeneration of the kidney by measures adapted to develop the strumous cachexia. I will not absolutely affirm that mottled kidneys may never undergo atrophy. In certain very few cases where the scrofulous deposit has been made in consecutive small instalments, slowly disorganising the gland—in such, *perhaps*, contraction may have ensued in the portions first and separately invaded. But I am persuaded that this occurrence must be quite exceptional. Under ordinary circumstances the fatty deposit occurs with uniformity throughout the gland, the function of which is so totally disturbed, that death anticipates the leisurely process of its contraction. The mottled kidney, in an infinitely *large* proportion of cases, remains *large* and mottled to the end.

On the other hand, if we start from what has been considered the final stage of Bright's disease, *the contracted and atrophied kidney*, we mount, step by step, not to fatty degeneration of the gland (or, if ever, with peculiar infrequency), but, through the series of changes enumerated in the present paper, to epithelial engorgement, to sub-acute inflammation: we mount to a new circle of pathological affinities, not to the strumous degenerations—not to phthisis and its congeners, but to the irritative blood-diseases, to fever, gout, rheumatism, and the like.

It seems almost indispensable for correct nomenclature

that the name of "Bright's disease" should be discontinued; for I do not see how it can be restricted to either of the two diseases which modern pathologists recognise as included by it. *Stant nominis umbrâ*. One class of persons may persist in giving the name to contracted kidneys which never have been mottled; others to mottled kidneys never about to be contracted. Both parties, with equal right or equal wrong, may appeal to the admirable pages of the "Medical Reports," and find their ambiguity apparently justified. Each of the two diseases is "Bright's disease;" for both are described with equal originality and truthfulness by the respected physician whose name has been identified with them.

I would suggest that, for the one class of cases which I believe to be far most numerous—the class, namely, treated of in the present paper—it will be convenient to employ the familiar and appropriate name of INFLAMMATION. I have chosen to call it *sub-acute*, rather than *chronic*, because its peculiarities have more to do with low intensity than with long duration. Cold and intemperance, the various fever-poisons, the irritation of gout, of rheumatism, of the oxalates,—these are its habitual causes. Its tendency is to the obliteration of tubes, to the development of parenchymic cysts, and finally to the contraction of the kidney.

Secondly,—There will be the degeneration which Dr. Johnson so successfully illustrated last session, commencing, as he demonstrated, in a fatty engorgement of the epithelium, and therein distinguished from the purely inflammatory affections. In its progress this disease has invariably appeared to me to have the sub-acute inflammation grafted on it; but *its origin is sui-generis*, is non-inflammatory; and it would be convenient, I think, if it were separated from the other under the name of SCROFULOUS DEGENERATION. This disease, as I have stated, leads in very few cases to contraction of the gland: at least, such is my own experience, and I have obtained from others some very strong evidence confirmatory of my opinion.

I may add that I have often seen the interstitial cyst-development in connection with the scrofulous degeneration, but not so frequently or regularly as in the sub-acute inflammation of the kidney. I am unable at present to decide whether the cyst formation belongs in these cases to the fatty disease, as of *its* creation ; or whether it merely arises when the inflammatory process has been superinduced, and depends for its birth (just as all the other fatal and irreparable changes in the gland depend for *their* immediate causation) rather on that secondary influence than on the original accumulation of fat.

I have now little further to add. With respect to the symptoms of sub-acute inflammation of the kidney, I will make one observation in addition to those already embodied in my paper. The descent of epithelium and its germs with the urine ; the presence of albumen there, and sometimes of blood ; the little casts of the tubules—sometimes wrought of fibrin, sometimes of compressed epithelium ;—these signs belong equally to the sub-acute inflammation and to the scrofulous disease. They are signals simply of renal irritation, whether from one cause or the other, and I suspect they only attend the scrofulous disease at that stage of its progress in which sub-acute inflammatory action is superadded to the primary fatty degeneration. Dr. Johnson's accurate observation has enabled us, under most circumstances, to diagnose the two classes from each other ; for, in the scrofulous disease there will be always seen, as he describes, more or less oil entangled in the fibrinous casts, or gorging the cells which descend in the urine ; a phenomenon which does not belong to the pure sub-acute inflammation.

In regard of treatment ; I have no new drug to recommend for the removal of cysts or the restoration of tubules. Here, as with so many other maladies, vigilance with respect to the first premonitions of the disease, and an exacter knowledge of its pathology, will enable every intelligent practitioner to apply old remedies with improved success.

From the moment in which albumen exists in the urine, I believe the exhibition of diuretics to be perilous and unwise in the extreme. From that moment everything must be done to spare and economise the injured organ.

Weight in the loins, with increased disturbance of the secretion, will often suggest the expediency of abstracting blood, locally, by cupping. Where the necessity of this measure is indicated, and where it can be borne, it never fails to give relief. In cases which seem unable to bear abstraction of blood, dry cupping is often attended with advantage.

In no remedy am I disposed to place so much confidence as in the sweating-bath; whether by dry air or watery vapour. I believe that its habitual use tends, more than any system of treatment with which I am acquainted, to counteract the chronic causes of the disease, to retard its progress and palliate its effects.

That cold must be most carefully guarded against; that every gastric source of renal irritation must be studiously obviated by measured diet and appropriate medicine; that dropsy and other secondary affections will require their own specific treatment;—all this is so obvious, that it would be worse than superfluous if I should detain the Society by dwelling on it. The treatment is a plain, common-sense inference from the pathology.

To conclude; a disease is practically important, in proportion as it is frequent or fatal. That on which I have so long detained the Society has both these claims: it is peculiarly frequent, and, in its confirmed form, of necessity shortens life.

When I remind the Society of its causation; how exposure to cold, how gout, rheumatism, scarlatina, cause it; how, too, not only scarlatina, but other blood-poisons almost as frequently;—it will be obvious that few can arrogate to themselves an entire immunity from the causes of the disease. That these causes need act with no great intensity in order to their destructive result, will be evident from the observations

I have made on epithelial engorgement of the kidney as a means of its frequent disorganization.

Sub-acute nephritis has appeared to me to exist quite as frequently as pulmonary consumption; and I shall not, I think, exaggerate in saying, that in two-thirds of its cases it is latent: its symptoms are overlooked during life, partly from their unobtrusiveness, partly because they are often merged in those of some striking constitutional disorder; while in like manner its organic traces in the dead body have been neglected, as rarely impressing on the kidney those grosser changes of aspect which would arrest the casual observer. I repeat that it has happened to me again and again, after *post-mortem* examinations, to find, in kidneys roughly estimated as healthy, the most thorough abolition of natural structure.

And I shall be satisfied with the result of this paper, if the Society hereafter be convinced, that a disease of immense frequency and of the greatest seriousness has hitherto, in a majority of its circumstances, escaped the observation it deserves.

For assistance in obtaining materials for the present paper, I am much indebted to my Hospital-colleagues; to Dr. Liddell, Inspector of Hospitals; to Dr. Cohen, of St. Thomas's; and to Messrs. Nunn and Brinton, my colleagues at King's College. I am anxious peculiarly to express my obligation to Mr. Prescott Hewett, not only for his remarkable kindness in furnishing me with many important illustrations for my paper, but likewise for the access allowed me to that excellent collection of reports which he has organised at St. George's Hospital, and which I believe to be unequalled in this country.

Note.—The doubt I have ventured to express as to the so-called *stages of Bright's disease*, and especially as to the

alleged *contraction of the mottled kidney*, first suggested itself to me in the form of the following argument.

The mottled, or fatty, kidney is essentially scrofulous: it is constantly found as a complication of phthisis. If its tendency be to produce contraction of the gland, or rather if it be but the first stage of a disease habitually advancing to that result; then, unquestionably, contracted kidneys ought to be found in the necropsies of phthisical patients. Are they so found?

In referring to statistics on this subject, I may remark that there is less room for fallacy than in many such numerical quotations. The diseased condition is very obvious. Mottled kidneys may mistakenly be written healthy, and the observer who has erred in respect of one specimen will probably err again with many. But no moderately careful person can possibly call a contracted kidney a healthy one.

Accordingly, I expect to learn, at least approximately, the disposition of the scrofulous degeneration to induce contraction of the gland, when I ask the following question:—In how many instances of fatal phthisis has a contracted kidney been seen?

1. Monsieur Louis refers to 214 examinations of the kidney in phthisis. In no one of them does he report any diminution of size to have been noticed.

2. In the invaluable Museum Book of St. George's Hospital, among 116 cases, carefully and circumstantially registered, I find only two that require notice; one, where in a woman of 42, dead with vomicae in the lungs, both kidneys were found "small, particularly the right one which had a granular surface:" and another of a man (age not mentioned) dead of phthisis, whose kidneys were "much reduced and granular, with a large cyst in the left one." Unfortunately there were no particulars to be obtained of these patients' histories, nor had any microscopical examination of the specimens been made. I mention these two, though they prove nothing one way or the other, as being the only two out of 116 phthisical cases to which the slightest suspicion of im-

portance could attach. I may add, too, that Mr. Hewett, who assisted me with these references, and to whose opinion on such a subject the utmost deference is due, tells me that he has long entertained doubts as to the contraction of kidneys affected with scrofulous degeneration; that he believes (as I do) that the *mottled* and *contracted* kidneys are of different pathological families.

EXPLANATION OF PLATE V.

This plate illustrates Mr. Simon's paper on Sub-acute
• Inflammation of the Kidney.

- 1, 2, 3, 4, 5, 6.—Different appearances of the cyst formation.
- 1, 2, 8.—Unnatural distention and plugging of the tubules.
- 7.—The vascular tuft of a Malpighian body squeezed into small compass by fluid pressure from within the tube.
- 1 and 3 are seen with an eighth object-glass; and the smaller cysts, here represented, are not larger than ordinary epithelial cells; the others are seen with a fourth-inch object-glass.