AN ADDRESS

ON THE

DEPOSITION OF THE CRYSTALLINE
URATES IN THE TISSUES;CONSIDERED AS A SEPARATE PATHOLOGICAL INCIDENT,
WITH A SUGGESTION FOR A DISTINCTIVE NAME.*Delivered before the Medical Society of London.*

By SIR WILLIAM ROBERTS, M.D., F.R.S.

OF all the phenomena associated with gout, by far the most definite and characteristic is the deposition of the crystalline urates in the joints and elsewhere. The occurrence of such deposits is usually regarded as pathognomonic of the gouty state; but the converse of this proposition is not accepted. Clinical observers have always recognised the existence of a pathological state, to which the names of suppressed, latent, or larval gout have been applied, in which no uratic deposits can be discovered. On the other hand, the collateral conditions associated with uratic deposits are extremely diverse. These conditions sometimes differ so much from each other, that they certainly would never have been classed together as varying forms of one and the same disease had there not been this single connecting link of uratic deposits.

Again, although the arthritic troubles of gout may be reasonably attributed to the direct effect of the mechanical irritation produced by the deposition of the crystalline urates, there are other troubles assumed to be truly gouty which cannot, with our present knowledge, be thus explained, namely, the visceral and nervous disturbances, which constitute an important part of the symptomatology of that nosological complex or totality which we call gout.

Further, uric acid exists in the body of the gouty in two very different states, namely, in a state of solution in the blood, and precipitated in the solid form in the tissues. To the former of these conditions the term lithæmia has been applied, and it is invoked to explain the occurrence of the visceral and neurotic disturbances just mentioned.

Moreover, we cannot ignore that there are pathologists among us who are unable to limit their view of gout to the effects of uric acid. They look upon the disorder as a complex constitutional state, having wide bearings in all sorts of directions. To them the obstruction of uric acid, whether dissolved in the circulating fluids or deposited in the solid form in the tissues, only constitutes a single manifestation of a multiform deviation from health. The most diverse views have been enunciated as to the nature of the initial error in gout. Some regard gout as primarily a disturbance of innervation, others as an altered type of nutrition. In some cases gout presents itself to us as if it were a form of plethora; in other cases as if it were a mode of senescence or growing old, analogous with atheromatous or mineral degeneration or with fibrosis. Some consider gout as, initially, a defect of the excretory powers of the kidneys for uric acid, others as primarily due to a diminished alkalinescence of the blood; and one observer contends that gout is essentially a necrotic process in the tissues induced by uric acid in solution, and that the deposition of the crystalline urates is secondary to the necrotic process.

Amid these conflicting views there is one fixed point in the controversies on gout around which all arguments revolve, namely, the deposition of the crystalline urates in the tissue. No speculation can get rid of this plain fact of morbid anatomy, nor can there be any doubt that these deposits, quite apart from their mode of origin, are directly answerable for a well-marked group of clinical and pathological manifestations.

All these considerations seem to lead up to the idea that it might be well, in the interest of clear conceptions respecting gout, and as likely to contribute to the promotion of definite lines of further investigation, if we were to isolate in our minds the precipitation of the crystalline urates as a detached and independent pathological occurrence, separate from all antecedent and collateral incidents, and erect it into a precisely definable symptom or physical sign apart from everything else.

This is a proposal quite in accordance with what has been done in other departments of medicine, and has greatly conduced to the progress of pathology and clinical research. Time was when the presence of sugar in the urine was regarded as synonymous with diabetes, and the presence of albumen as synonymous with Bright's disease. But by isolating these conditions under the names of glycosuria and albuminuria we have been enabled to attain to a greater precision in our ways of thinking and speaking about these two diseases. Glycosuria is now regarded in the light of a symptom, and we must add something thereto to complete our conception of classical diabetes, and we recognise that glycosuria may exist as an element in other morbid conditions besides diabetes. So with albuminuria. We know that this symptom may coincide with a variety of disorders. Another example in point is the separation of clotting in the veins from phlebitis, with which it was formerly confounded. It is now recognised that this incident may be due to other causes than phlebitis; and it has accordingly been isolated under the distinctive name of thrombosis.

Following these precedents, and others that might be cited, I venture to suggest that uratic precipitation should be separated from its associated connections and dignified with a name of its own. For, trivial as the question of name may appear, it is quite certain that the distinctness of our comprehension of any notion is immensely favoured by designating it with a name. By this means vague notions are condensed into definite conceptions. I have puzzled myself not a little over this problem of nomenclature. The thing to be designated is the precipitation of the crystalline urates in the tissues or fluids of the body. The name I have finally decided to recommend to you is *uratosi*, compounded of the term *urate* with the Greek termination *osis*. I am afraid the word is something of a barbarism, but a highly competent classical friend of mine assures me that it does not offend very far in this direction. It resembles in construction and in force of meaning such well-known words as tuberculosis, thrombosis, adiposis, and melanosis. The chief merit of a name, however, is that it is adapted to express clearly and precisely the meaning intended to be conveyed, and is handy and convenient for service. This merit may, I think, be fairly claimed for the word *uratosi*, and in order to illustrate its use I shall venture to employ it in the course of the further remarks I have to make. We should, I think, gain several advantages besides that of avoiding a certain amount of circumlocution from the adoption of this proposal.

In the first place, we should bring into clearer relief the distinction between uric acid dissolved in the blood and uric acid precipitated as crystalline urate—or, to use our new phraseology, the distinction between lithæmia and uratosi. The differences between these two conditions are, I think, important, and deserve, perhaps, more attention than they have hitherto received. In lithæmia the exact state of the uric acid is not precisely known. We are sure that it is not in the free state, because it is chemically impossible for uric acid to subsist in the alkaline blood serum without entering into combination with the bases contained in it; but whether that combination be a quadrurate or a biurate, or a mixture of the two, or sometimes one and sometimes the other, we do not know. These two combinations are strongly contrasted in several respects; the quadrurates are highly soluble in blood serum, and cannot be thrown down therein in the crystalline state, while the biurates are very sparingly soluble in that medium, and are readily thrown down in it in the crystalline form. On the other hand, in uratosi we know exactly what we have to deal with; uratic deposits are composed of crystalline sodium biurate—that, and nothing else. Again, it is an open question whether uric acid dissolved in the blood exercises any injurious effects at all. It appears not improbable that uric acid is as innocuous when dissolved in the blood as we know it to be when dissolved in the urine. But we do know of a certainty that when uric acid is precipitated in the tissues as crystalline urate it does produce injurious effects. Another distinction between lithæmia and uratosi is this: Lithæmia is only an exaggeration of a normal condition, for traces of uric acid always exist in healthy blood; but uratosi, even in the minutest degree, is pathological. There is yet another point of interest in this connection, though at present only of speculative interest, to which I desire to draw attention. It is quite possible that precipitation of the crystalline urates may occur not only in the form of the coarse masses with which we are familiar in and about gouty joints, but also in the form of minute stars or detached crystals, which can only be detected with the microscope; and it may turn out that the visceral neuroses,

the thrombosis and embolism, and the occasional sudden death which are encountered in gout, are not the result of any diffuse lithæmia, but are the consequences of actual uratoses—that is to say, are due to the precipitation of minute crystals of sodium biurate (microscopic uratoses) either in the substance of the organs or in the blood itself. I hazard this conjecture on the ground of the two following observations: Sir Alfred Garrod found that the blood serum of one of his patients contained uric acid to the extent of one part in 5,714. On the other hand, I found by direct experiment that when blood serum was artificially impregnated with uric acid to the extent of 1 part in 5,000, there could be detected in it after a certain lapse of time minute stars and needles of sodium biurate. It would thus appear undoubted that in some cases of gout the blood may, though probably very rarely, become so laden with uric acid as to be within striking distance of a general uratoses throughout its entire mass. The establishment of the possibility of such a condition would obviously enable us to explain the occurrence of some very obscure incidents in the history of gout.

Secondly, the adoption of the present proposal would enable us to discuss more freely and with greater precision of language the true relation of uratic deposits to the ætiological factors which are concerned in their production. According to current views these deposits are regarded as an infallible sign of the existence of the gouty diathesis, however diverse the associated conditions may be. Hence, we are in the habit of speaking of *ordinary* gout, and of *saturnine* gout. Would it not be a more accurate expression of the facts to speak of *gouty uratoses* and *saturnine uratoses*? It is not easy to believe that lead poisoning produces really and truly the same constitutional diathesis as that which exists in true gout. It seems easier to believe that the gouty diathesis and lead poisoning, while differing in all other respects, have one tendency or vice in common, namely, the tendency to uratoses. The possession of this one vice would necessarily give to two otherwise radically distinct primary disorders a very close resemblance in regard to their symptoms and physical signs, because these latter depend almost entirely on the direct local effects of the uratoses which is common to both. This line of argument might be illustrated and strengthened by drawing an analogy between uratic deposits and urinary concretions. A large group of cases of uric acid, gravel, and calculus are the undoubted offspring of gout; but there is also another large group of cases, occurring chiefly among the poor and among rice eating populations, which have no kinship with gout, and are the result of a totally different set of ætiological factors. Nevertheless, the clinical and pathological phenomena in the two groups, depending as they do on the mechanical irritation induced by the concretions, are practically indistinguishable. Here then we have a closely analogous example in which two essentially distinct constitutional states produce identically the same morbid precipitation and the same sequential morbid effects.

On the other side, it might be contended that this line of argument is inconclusive, and is directly rebutted by established facts relating to the hereditary transmission of gout, for, as is well known, *sthenic* gout in the grandfather may reappear in the grandchild as *atonic* or “*poor*” gout. These two conditions differ from each other quite as much as does ordinary gout from *saturnine* gout. Almost the sole point of resemblance between them is the occurrence in both of uratic deposits, and yet they are undeniably of common constitutional origin. To this objection it may be answered that gout is not in reality a homogeneous nosological unity, but a loose bundle of morbid tendencies. And it is quite in accordance with what we know of the laws of heredity that these several tendencies should be transmitted in unequal degrees, or that some of them should not be transmitted at all, while others are transmitted with increased intensity. In this way it might come to pass that a grandchild might inherit from his plethoric gouty grandfather his tendency to uratoses, but not his tendency to plethora—just as he often does inherit the characteristics of his grandfather's eyes and nose, but not those of his forehead and chin.

Then, again, it is admitted that there are other factors which have considerable ætiological force in the production of uratic deposits besides the gouty diathesis and lead poisoning, namely, senescence, nervous shocks, local injury, and trophic changes. Two or more of these may act in combination in determining the occurrence of uratic precipitation. Adopting the nomenclature suggested, we could speak of a uratoses of purely gouty origin, of a uratoses in which a gouty tendency was reinforced by lead poisoning,

by senescence, by shock, or by local injury; or we might speak of a purely saturnine uratoses, or of a uratoses in which saturnine poisoning was reinforced by a previously existing gouty tendency—and so forth. By this mode of expressing ourselves we could conveniently indicate the concurrence of several factors in producing uratic precipitations, none of which alone were adequate to determine their occurrence.

Lastly, we should gain something in regard to a clearer apprehension of therapeutical indications. We could, on the one hand, aim at minimising the incidence of the predisposing cause—be it the gouty diathesis, or lead poisoning, or whatever else might eventually come to be regarded as the most important predisposing factor. Or, on the other hand, failing in that direction, we could aim directly at the uratoses itself. In this latter way we might succeed in preventing the more serious consequences of an irremovable predisposition; just as in uric acid gravel we are able, by alkalisating the urine, to guard our patient against calculous accidents in circumstances where we cannot remove the causes predisposing thereto. Uratoses is, like gravel, of and for itself a disastrous pathological accident; and it may induce consequences in the joints or in the kidneys of a permanent character, which far exceed in gravity, and far outlast, the ætiological factors which originally gave rise to it.

And should it turn out at last, after further inquiry and investigation, that there does subsist an absolute and invariable solidarity between uratic deposits and the gouty diathesis—and I wish to express no positive opinion on this point—we should lose nothing by adopting these proposals. On the contrary, we should distinctly gain in facility of description and in clearness of conception in regard to the main incidents of the gouty state.

I have, I think, said enough to bring the subject fairly before the Society, and I wish to elicit the opinion of the Fellows on two points, namely, first, whether it be desirable to erect the precipitation of the crystalline urates within the body into an independent symptom or physical sign, with a distinctive name; and secondly, whether the name suggested is worthy of approval.

AN ADDRESS ON ANEURYSM:

ITS CURE BY INDUCING THE FORMATION OF
WHITE THROMBI WITHIN THE SAC.

Delivered to the Midland Medical Society.

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[Concluded from page 1109.]

Let us now turn our attention to some clinical evidence of the value of this mode of treatment in curing aneurysms. In doing so the cases presented are not arranged chronologically, but in pathological order.

Case of Aortic Aneurysm seen at an Advanced Period, when threatened by Impending Death from Dyspnoea, treated by Induction of White Thrombi within the Sac, the Treatment being cut short within the Month by Fatal Dyspnoea; Post-Mortem Examination showing the Interior of the Aneurysm to be filled to Two-Thirds of its Extent by Firm White Thrombi.—S. W., aged 32, an engineer, was seen by me in private, in consultation with Drs. Hunter and Middleton, early in November, 1887, suffering from an aneurysm, which projected from the chest above the clavicle on the right side of the root of the neck, and was supposed to be either purely aortic or an aortic aneurysm involving the innominate. For a year previously he had been affected with symptoms pointing more or less directly to aortic aneurysm. As these symptoms increased he had to desist from work. Subsequently he consulted many surgeons and physicians, principally in England, who all advised him against operative interference. On a number of occasions he had had attacks of breathlessness, so severe as to threaten a fatal termination. There was no syphilitic history.

On examination an eccentrically pulsating swelling was seen at the inner extremity of the right clavicle, occupying the episternal notch, which seemed to cause the sternal end of the clavicle to project forward. The trachea was distinctly pressed to the left,