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Wm D.D.

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OBSERVATIONS ON
RHEUMATOID ARTHRITIS, HEBERDEN'S NODES,
DUPUYTREN'S CONTRACTION, AND SOME
OTHER ARTHRITIC CONDITIONS.

BY

SIR DYCE DUCKWORTH, M.D., LL.D.

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It cannot be denied that while we have an accurate knowledge of the morbid anatomy of the disorder known as rheumatoid arthritis, we are still in ignorance of its exact pathogeny. I suppose that as many theories have been suggested for the latter as names have been affixed to the malady. The condition is so common, and its characters so striking, and at times so urgent, that it has not failed to enlist careful study at the hands of many able physicians and surgeons. When generalised and well marked, there is seldom difficulty in making the diagnosis of the disease, but the case is very different in respect of its lesser degrees and limited manifestations. To take but one instance, the condition recognised as Heberden's nodes, where we find no consensus of opinion as to its pathogenic significance, some regarding it as truly gouty, despite the teaching of Heberden himself, others claiming it as rheumatoid—"end-joint rheumatism." The same may be affirmed of the palmar fascial sclerosis known as Dupuytren's contraction, which is relegated to rheumatic or gouty influences according to the particular views of observers. It is surely high time that we had a little more certainty respecting these

conditions, for that they have specific indications of their own cannot be doubted.

We have to recognise first, that a large number of people are, and remain, free from all manifestations of articular diseases. They are not, in the language of the French school, arthritically disposed. Exposure to the conditions which would provoke rheumatism or rheumatoid disease in certain individuals, is powerless to establish these in others. There is surely something significant in this fact, affording a basis for the belief that there exists in some persons a predisposition to diseases of this class, alighting specifically upon joints and parts composed of similar tissues. The same remark equally applies to the manifestations of scrofula, a term which some pathologists now appear to discard, believing that Koch's discoveries have shown that what was formerly known as scrofula is but a phase of tuberculosis—a doctrine I utterly deny. It is quite certain that many individuals are not, and never will become, scrofulous. While, then, these two habits of body are widely spread and commonly met with, they are not universal. It is now generally held by pathologists that rheumatic fever depends upon a specific infection by some peccant matter implanted in persons whose textures present a suitable soil for its manifestations. This infective agent must be spread largely and widely, and yet great numbers of persons freely exposed to it entirely fail to suffer from its effects. Although the joints are usually much involved in the disorder, it is found that, on the subsidence of it, these structures soon recover their natural condition, and afford no evidence of any permanent structural damage during life, or even after death. Repeated attacks of the disease may likewise entail no permanent damage to joints. I forbear to allude to other possible disablements caused by acute rheumatism, because we are now only concerned with the articular structures as affected by it.

In too many instances we have to recall the fact that pains in joints, muscles, fasciæ, and other parts, are styled, not only by the patients themselves, but by their medical attendants, as "rheumatic," "pains," and "rheumatism," being too often regarded, carelessly, as synonymous. With respect to the condition of osteo- or rheumatoid arthritis, we do not observe that it is a common sequel of rheumatic fever. In a definite proportion of cases there is, however, to be gathered presumptive evidence of a past attack, or of attacks of this disease, and in any case presenting signs of cardiac or pericardial lesions we may be fairly sure of this, since these form no part of the

features of the disorder under consideration. This fact goes in support of the individual diathetic tendency which is present, and which is significant of a textural predisposition to disease of this class. In rheumatoid arthritis there may be acute exacerbations of pain with polyarthritis, and a moderate degree of pyrexia, which may be, and have often been, mistaken for attacks of rheumatic fever, but they lack several important features of that disorder, and certainly resist the treatment which commonly affords relief from it.

The conditions which predispose to rheumatoid arthritis are well recognised, and chiefly relate to depressed health, exhaustion from loss of blood, hyperlactation, poor living, and prolonged anxiety, together with exposure to damp and rapid alternations of temperature. They are also such as would un-failingly excite any scrofulous tendency into activity.

With respect to the pathological changes induced by the disease, we have to note that they may be partial and limited, or very widely diffused over the joints. The intensity of the process clearly varies much, so that it is a matter of degree in each case. And here we have to face the fact that the ultimate results in many instances of chronic deforming arthritis tell us but little of the actual factors that have been concerned in bringing them about. To be quite certain in a particular case as to its exact pathogeny, we must have knowledge of the clinical history and conditions which determined and accompanied the phenomena of it. A study of museum specimens, devoid of such histories, tells very inadequately their true significance, for it is certain that other forms of arthritis may closely simulate some of the changes witnessed in rheumatoid arthritis. To my mind, it is clear that gouty arthritis induces many of the characters of the latter, and I have come to regard it as an error to hold that uratic deposits are an invariable, or at least an enduring, evidence of accompaniment of true gout. If joints encrusted with urates, and damaged in most of their component tissues by gouty changes, be submitted to any solvent that will remove the deposit, it becomes a matter of impossibility to assert with confidence, in the absence of a clinical record, that such changes are the unequivocal results of gout. It is proved that joints which have been recognised as gouty during life on trustworthy evidence, with definitely associated concomitants of that disease, may after death show no uratic deposition, such having been removed in the course of time, especially in vigorous patients.

The lesions in Charcot's disease, again, often recall those of some forms of rheumatoid arthritis, the process having been,

no doubt, sudden in onset, and riotous, so to say, in their rapid development.

There are changes also in strumous arthritis, conditioned, no doubt, by tuberculous invasion, which resemble some of those witnessed in rheumatoid arthritis.

A consideration of these facts has doubtless led many careful students of this disease to the belief that it owns no unity of pathogeny, but that, as "many roads lead to Rome," so many and various pathogenic factors may possibly be concerned in evolving forms of arthritis which we find it convenient to label rheumatoid arthritis. I am in accord with those who so regard the matter. The clinical histories of the cases compel one to take such a view, for the contrasts are many and strange. Thus we meet with deforming arthritis in young children, and with *malum coxæ senile*. We find the characteristic lesions in the joints of those who have simply been exposed to extreme hard work, and have never been seriously incommoded by them. As a consequence of menorrhagia, hyperlactation, misery, poverty, ill-nutrition, and exposure to extreme changes of temperature, as in the occupations of baking and laundry-work, we find all degrees of the malady. Again, where night-watching, with loss of sleep, anxiety, and grief, have broken down the health, the disease may set in; and even in the well-to-do, leading prudent lives, without undue provocation, so far as can be learned, we may meet with it as an evolutionary outcome or as a form of textural senility. A trifling injury may be the starting-point of many involved joints, plainly indicating irritation conveyed to and from certain nervous centres, and in this connection we have to bear in mind the whole class of articular dystrophies known as spinal arthropathy and neurarthritis. How are we to compose all these conditions under one pathogeny? The articular system, as a whole, reacts to many varieties of specific irritation. The poisons of rheumatism, of gout, gonorrhœa, pyæmia, dysentery, scarlet fever, influenza, enteric fever, and tuberculosis, have all sometimes an elective affinity, sooner or later, for the component textures of the joints, and no less may traumatism, as we have seen, set up not only monarthritis, but seemingly, by reflex action, involve in succession many joints. In all these instances, however, with the exception of pyæmia, we may note the special predisposition to articular affections; for the non-disposed do not, as a rule, suffer from rheumatism, gout, or tuberculosis. The so-called scarlatinal rheumatism is not seldom met with in those who are apt to suffer from true rheumatism, and some examples of it are clearly of a pyæmic nature. Gonorrhœal poison is especially

prone to alight on the joints of those of gouty habit or inheritance, and injuries to joints commonly heal quietly in persons not predisposed to scrofula, gout, or rheumatism. In the case of syphilitic arthritis, which is a rare condition, it may be suspected that there is the same tendency, or possibly a strumous one. Injured joints, as *loci minoris resistentiæ*, are naturally sites for specific manifestations of any existing dyscrasiæ.

With respect to a recently promulgated view that rheumatoid arthritis is due to microbic influence, or a toxin produced by this, it may be affirmed that no satisfactory evidence is as yet forthcoming in support of it; and if it were, it could but add one more factor to the many that appear to be engaged in the pathogeny of the disorder.

It must be granted that while, as I have remarked, the joints react to varieties of irritation, either toxic or traumatic, there remain cases, happily the majority, in which the articulations resist the provocation to undergo changes leading to, or resulting in, the lesions of rheumatoid arthritis. Such resistance, I believe, resides in a sound condition of the system, one void of rheumatic, gouty, or strumous proclivity, and this probably consists largely in such a measure of integrity of the nervous system, including stability of nerve centres and vigour of neuro-trophic action, as proves efficient to avert the degenerating and destructive influences which threaten those who, in default of such constitutions, are feeble and predisposed to break down in this textural line. This view of the matter brings us in face of the dystrophic theory of rheumatoid arthritis, one which I have long been disposed to adopt as affording the best explanation of the many difficulties surrounding the subject. It may be formulated concisely as follows:—

Predisposing Causes.

Primar (inherited) weakness, irritability or defect of nervous centres, especially of those which are assumed to preside over the nutrition of the joints.

Determining Causes.

- A. Depressing conditions which directly aggravate this debility of neuro-trophic centres.
- B. Toxic or traumatic peripheral irritation acting reflexly through these unstable centres so as to involve one or many joints.

There is one factor which to my mind clearly plays a frequent, and often a large, part in the pathogenic relations of rheumatoid arthritis. It has not escaped the attention of careful observers, but it has not been adequately appreciated. I allude to the strumous element which is often to be noted in the ancestral, family, or personal history of patients. It is not

strange that this habit of body, which is as widespread as that of the arthritic diathesis, should often be blended in various degrees with the latter, and we meet daily with instances which illustrate it, in which the manifestations of struma and gout, as well as those of struma and true rheumatism appear, one condition materially modifying the other. It is of great importance to recognise such influences, for they materially alter the clinical features of cases as witnessed in practice. It is certainly common to find a history of phthisis in the families or near relations of those who become the subject of rheumatoid arthritis, and I conceive that this occurrence is of no ordinary significance. It is well ascertained that the arthritic habit of body exercises an inhibitory influence over strumous and tuberculous processes from the earliest periods of life, so that any manifestations of the latter are either kept in abeyance or greatly retarded in their progress; yet, according to the degree of the strumous proclivity in any given case, we find evidence of its influence in modifying the effects of rheumatic or gouty poison, and no less in cases where other specific irritants come in to disturb the system, as witnessed in the graver results of syphilis, gonorrhœa, exanthematic or septicæmic toxins. The strumous proclivity provides a bad soil for all of these to alight upon, and thus is a common cause for the untoward outcome of many of such cases. I think we may thus find explanation for some of the varied forms which gout, rheumatoid, and gonorrhœal arthritis assume. I am disposed to think that cases presenting more than usual effusion in the joints, manifesting a tendency to linger long, and proving rebellious to ordinary treatment, owe their characters to a strumous element, which thus modifies the nature and course of the disorder. The same depressing conditions which evoke the trophic changes in the joints no less induce the strumous tendency, which may have been hitherto latent or but little manifested, to assert itself and play its part in the process. I think we have hitherto failed to realise the influences of this kind which possibly determine some of the characters and varieties presented by rheumatoid arthritis as a whole. Other changes, notably in the kidneys in the case of gout, may not improbably be due to the same modifying influence.

The peculiar form of rheumatoid arthritis in association with lymphatic irritation, enlarged spleen, and lympharia met with in children, has as yet no well-defined pathogeny. I venture to offer the suggestion that this condition may possibly be determined by the existence of a strumous cachexia, which modifies the ordinary phenomena of the disorder.

M. Lancereaux has directed attention to a slowly progressive variety of arthritis, which he connects with chronic interstitial nephritis, arising from, or occurring as part of, the condition of arterio-capillary fibrosis. He notes the absence of uratic deposit, the destruction of articular cartilage, and the formation of ecchondroses and bony outgrowths. The joints chiefly involved are the metacarpo-phalangeal of the thumb and the knees. He regards the disorder as independent of gout and saturnine impregnation, and presenting the characters of rheumatoid arthritis, and due to a neuro-trophic defect.¹ I do not think these observations have been much noticed or confirmed by other observers, and I am disposed to regard these changes, which I have myself observed, as dependent upon a gouty habit, and to represent some of the incomplete forms of gouty arthritis. I may allude here to Dr. Archibald Garrod's views respecting what he regards as examples of secondary rheumatoid arthritis. He believes that joints which have been involved by rheumatism, gonorrhœal arthritis, or gout may sometimes undergo the peculiar dystrophic change of rheumatoid arthritis, the original specific irritant having in each case determined the onset of the new form of degeneration.

From the point of morbid anatomy, regard should be had to the specific differences which are to be found in the outgrowths respectively of gouty and rheumatoid arthritis. Those of the former are of the nature of exostoses, and those of the latter are ecchondroses, as shown by Dr. Wynne.² Lancereaux's variety of dry arthritis is perhaps somewhat allied to the condition of Heberden's nodes, of which I will next treat. As the result of many observations, I have come to regard these as more often significant of a truly gouty tendency than a result of any rheumatic condition. They are met with in certain families, many members of which may be overtly gouty, and they are distinctly hereditary. They appear sometimes to be little more than a senile change, and are regarded by some authorities as indications whereon to prognosticate longevity. A node may be the result of a local injury to a solitary joint in a gouty subject. In some persons these nodes are rendered active and painful by any gout-provoking food or conditions. Charcot noted the frequency of cancer in women who manifested them. Heberden's remarks about them have been, I think, somewhat misunderstood. What he meant to infer was that they were not ordinary examples of gouty arthritis, and that they occurred in persons in whom gout was unknown. But we have

¹ *Trans. Internat. Med. Congress*; vol. ii. p. 193. London, 1881.

² *Treatise on Gout*, Duckworth, p. 77, 1889.

learned since his time to recognise many phases of non-classical, irregular, and incomplete gout. He also declared that these nodes were "void of all pain," which is certainly not the case in many instances. I have known typical Heberden's nodes to be the seat of uratic deposits quite unsuspected during life,¹ and I believe that a careful inquiry into the ancestral and family histories of the subjects of them will generally afford evidence of gouty taint. When the small "crab's-eye" or pearly nodules are formed on the enlarged tubercles of Heberden's nodes, they are significant of gout, and not of rheumatoid arthritis. They are not, however, due to uratic deposit, and are probably the result of inflamed adventitious bursal sacs, or else they depend on protrusions of synovia through some small openings in the capsule of the joint.

In some cases, not of gouty nature, we may regard the change as a form of dry arthritis—(*arthrite sèche* of the French school).

Respecting the scleroses of the palmar fascia recognised as Dupuytren's contraction, I have to declare my opinion that this is a condition almost entirely confined to persons of gouty inheritance or proclivity. It is not frequent in persons who have overt gout, but it is clearly an appanage of those who suffer by inheritance or acquirement from the irregular or incomplete phases of gout. Rheumatism has nothing to do with it. There are to be met with cases somewhat allied in character in which a gouty thickening occurs in the thecæ of tendons, often of the flexors of the fingers, accompanied by much pain, contraction, and inability. It comes on insidiously, sometimes after exposure to severe cold, and is apt to last for many months before it yields. It is usually followed by complete recovery. It is, however, a most intractable condition, and no local or constitutional measures appear to influence it. Happily it passes away gradually, and leaves behind it no inability of the parts affected. The great object in most of the incomplete phases of gout is to recognise the importance of securing for the patient the highest general level of health that is attainable, and not to treat him *secundum artem* for gout, which treatment commonly lowers the bodily powers and does harm. Lowered vitality distinctly disposes such subjects to multiform ailments which depend on their special predisposition. Rigid dietary and courses of water-drinking commonly do harm in such cases, and render the patients miserable.

The diagnosis is not seldom difficult in cases which present in no marked form either the characters of chronic gouty

¹ Treatise on Gout, Duckworth, p. 71, 1889.

arthritis or of rheumatoid arthritis. Even when all the past history and the present concomitants are fully secured, difficulties sometimes occur. Many of such cases, however, have been cleared up in recent times, and with careful study the difficulties should diminish in the near future. In cases of what is often termed "muscular rheumatism," I am convinced that we have generally to deal with gout whose manifestations are mostly in the fibrous nerve sheaths, or in the lymphatic spaces of the parts affected; and I believe that the same is the case in most examples of lumbago and sciatica, true rheumatic poison rarely, if ever, being an exciting factor in these ailments.

In conclusion, I will venture to hope that, in setting forth these opinions and in stating my experience, I may have helped to throw a little light upon these difficult problems, or at least to offer suggestions for further consideration respecting them.

