Chronic Arthritis: Classification and Principles of Treatment

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Remarks to Open a Discussion at the British Medical Association Congress, New Zealand Branch at Auckland, 1924, *by* D. W. CARMALT JONES, M.D., F.R.C.P., *Professor of Systematic Medicine, University of Otago.*

I am reminded that this is not the first time that this subject has been discussed at Medical Congresses. I believe that archæological research in Egypt and elsewhere has discovered specimens of osteo-arthritis in human subjects which are at least 5000 years old, and in this year of grace we are met to find the place of that condition among the various forms of joint disease—for the point is not yet settled.

The inference, I think, is fairly obvious. There is no natural and indisputable classification of arthritis, and the reason for this is equally obvious. Joints are passive structures, they consist of articular surfaces, synovial membranes, ligaments and bones, and these are liable to damage or destruction from a variety of causes. Whatever the cause, and whatever the structure injured, the net result to the patient is much the same. The function of a joint is to permit movement of levers through a certain limited range, and when the joint is injured either too much movement is permitted, or not enough, generally the latter, and with the disturbance in function there is usually considerable subjective disability manifested by pain. Thus there is little difference to the patient whether his trouble is due to tubercular infection, hæmophilia or fibrositis, and the physical signs present in each of these forms of arthritis might present little difference from one another.

However, the prognosis and treatment of cases of arthritis of varying origin differ profoundly from each other, and some classification is required, but it should, I think, be remembered that any classification is arbitrary, and only to be adopted for convenience.

At the best of times many cases must be dubious, anomalous specimens are frequent, watertight compartments are not to be looked for, and the best classification we can arrive at will only be applicable to the common run of cases.

The classification which I am in the habit of adopting in teaching is etiological, and it can be applied to arthritis as to the diseases of any other organ or system.

The most easily recognisable causes of disease are, I think, the following:—1, Senility; 2, overwork; 3, malnutrition; 4, extraneous poisons; 5, metabolic poisons; 6, bacterial toxins; 7, trauma; 8, new growths. Each of these, of course, makes a heading with many subdivisions.

Of these, the first three, senility, overwork and malnutrition are probably contributory causes of osteo-arthritis, though I think infection is more important. Gout is recognised as due to metabolic poisoning, and bacterial toxins, in one way or another, are the causes of the recognised infective arthritides, tubercular, gonococcal, streptococcal and so forth, and I shall attempt to give my reasons for thinking them at least important contributory causes of muscular rheumatism, fibrositis, whether affecting joints or not, osteo-arthritis and rheumatoid arthritis. Trauma, used in a wide sense, includes direct inquiry to joints, and also hæmophilic and allied lesions, and other conditions, comparatively unimportant, such as pulmonary hypertrophic osteo-arthropathy, which one believes to be due to a physical cause, chronic venous congestion.

I suggest, then, that the chronic forms of arthritis which we may profitably discuss are gout, a metabolic toxæmia, direct infections of joints with recognisable organisms, traumatic lesions, including those due to hæmorrhage; about these there is not much dispute, the difficulty in classification lies among those which I have somewhat vaguely classed as infective, fibrositis, rheumatoid arthritis and osteo-arthritis.

Gout is perhaps a disease of decreasing importance at the present time. I, at any rate, have rarely, if ever, made the diagnosis in this country. Modern work on blood chemistry, which has illuminated so many problems in medicine, has so far taught us little with regard to gout. One view is that in gout there is a deficiency in the body of a ferment called “oxidace,” in consequence of which the “purin” bodies which have

the formula C5 H4 N4 are imperfectly oxidised, uric acid being formed instead of urea. This is not the whole story, because in pneumonia and other diseases, uric acid is present in the blood in excess without producing the symptoms of gout. In gout, however, sodium bi-urate is formed in the blood and deposited in the tissues, and an attack of acute gout is associated with a sudden deposit of the kind. We need not, I think, now discuss the subject at length. Gout is generally considered a metabolic toxæmia, it is recognised clinically by its hereditary character, its usual incidence in middle-aged males, its peculiar onset, at night in the metatarso-phalangeal joint of the great toe, by the presence of tophi in the ears and other sites, and by its response to colchicum.