"GOUT."*

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[Abridged.]

(*Concluded from page* iii.)

Now, chronic rheumatism and rheumatoid arthritis very often begin like rheumatic fibrositis, *i.e.*, the toxin first affects the fibrous tissues in the tendons, ligaments and faxia around a joint. There is frequently a history of some injury, even a slight injury to the joint, which within a week or two is followed by pain and tenderness around the joint with loss of power in movement. With the pain there may also be some local heat (an inflammation) and tightness due to exudations around the joint capsule.

Very frequently such a case is diagnosed as gout, and the patient is put on a strict anti-gout régime, with lowering treatment by iodides, colchicum, and alkaline waters. Also, unfortunately, the case is aggravated by the patient being ordered massage and electricity.

I firmly believe that such cases as these and so-called fibrositis are muscular. Rheumatism, or the beginning of chronic rheumatoid arthritis, *i.e.*—eventually, if not treated the inflammatory process will bury itself into the joint, causing degeneration of the synovial membrane and cartilage, and in the end erosion of the joint bones.

The modern tendency for lay people to read and study advertisements of "cures" for all ailments leads many patients to disaster with reference to rheumatic diseases, for advertisements purporting to cure these ailments by some nostrum or other are invariably headed "Cures for rheumatism and gout"; they will go on to describe how their wonderful elixirs will dissolve and eliminate the uric acid from rheumatic joints. Hence, many people suffering from rheumatism in different forms have wasted their money and their strength long before they get into the hands of the physician. They are greatly surprised when they are told that their trouble is not rheumatic gout, that it is not due to uric acid.

Not so many years ago an enormous number of ailments were all attributable to a uric acid diathesis. I am personally convinced that diseases due to uric acid excess, including gout, are daily becoming more rare, while diseases due to toxins of microbic origin are increasing. This is chiefly due, I am sure, to the fact that our knowledge, thanks to the pathologist bacteriologist, is improving.

To return, however, to our consideration of gout in both the typical and atypical forms, the real difficulty is in making a correct diagnosis. In the acute typical forms it is easy enough, as described, but in the atypical and chronic forms it is one of great difficulty. There are, however, many cases of real gout of the atypical type, *i.e.*, where the big toe is not the offending member, but where other joints are affected such as the knee joint, for instance.

If careful consideration is given, however, to the history of the case, the age of the patient, and the clinical symptoms a diagnosis can be made, especially if the urine is found to contain an excess of uric acid. As a general rule, moreover, when gout attacks a joint or joints, it remains in these joints until the attack subsides, whereas in acute articular rheumatism the trouble frequently jumps from joint to joint.

It is the chronic afebrile cases of rheumatism, however, which are so frequently misjudged to be gout—by these I mean rheumatoid arthritis and osteo-arthritis, or arthritis deformans. The errors in diagnosis are more frequent still where the phalanges of the fingers are deformed—if the changes are limited to the capsule edges of the finger joints, causing small elevations around the joint—these are known as "Heberdens Nodes," and are typical of rheumatoid arthritis—these nodes contain no urates or deposits caused by uric acid.

It is very rarely, however, that we now see the true gouty finger joints so well known by our ancestors. These finger joints were swollen hard, stiffened and deformed, and frequently the urates ulcerated through the skin forming actual chalk like deposits in and around the knuckles. Probably you have heard of your own ancestors with gouty fingers being able to use these joints to write on a slate, the score at a game of whist. These were extreme cases and are seldom, if ever, seen nowadays.

We are compelled to believe that the reason is that we, of the present generation, are more temperate both in eating and drinking—by drinking of course I mean alcoholic liquors, especially rich wines. Port wine appears to generate a gouty diathesis quicker than any other wine or spirit.

Now, the only concise way to diagnose gout in all forms is by examination of the urine.

For the purpose of explanation we distinguish two kinds of uric acid: (1) Endogenous, and (2) Exogenous.

The first, *i.e.*, endogenous uric acid, is the uric acid that an individual can store in his blood, and excrete in his urine, by the wasting of his own muscles, and some individuals have the power, unfortunately, even when on a strict carbohydrate diet, of making a large amount of uric acid in the blood from their own tissues. This is probably the true hereditary gout tendency. Such an individual will excrete the same amount of uric acid daily no matter how strictly his diet is formulated for him, and if he should take much proteid and alcohol—say a good dinner of beef steak with port wine—the uric acid in his blood may, you can readily understand, assume a large proportion. Hence such an individual will either suffer from severe gout attacks, or, if he persists in diet indiscretions, will eventually suffer from chronic

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