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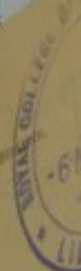
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GOUT: ITS ETIOLOGY

By ANTHONY R.
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Gout is common with most of the people, has passed through a mass of literature, its true etiology has received but so little of the subject, the true gouty attack is the small joints and the local and constant for many years. After this, and was ordered on uric acid as the cause of the disease were found deposited. The English and American physicians with literature "Uric acid disease," "Uric acid disease" in manufacturing houses of iron and steel. Some of the drug firms are publishing special journals on the disease being only with the cause of the etiology of the disease. It seems as justified in looking upon the past as we have a patient with an arthritic joint of the big toe, by placing and absorbent cotton dressing over the joint; the milk and linseed minimize the local irritation and the necessity for their use, increase the elimination. True as we are treated only one of its

A case report study of 8 cases of the kind present a chronic excretion of uric acid in the stools were observed in profusion, and hence shown as almost devoid of bacterial



Tracts
1638 ①

GOUT: ITS ETIOLOGY AND TREATMENT.

By ANTHONY BASSLER, M.D.,
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Gout, in common with most of the katabolic conditions of the human body, has passed through a mass of literature in which the important matter of its true etiology has received but scant attention. In the early considerations of the subject, the true gouty manifestation of acute inflammation of the small joints and the local and constitutional treatments therefor predominated for many years. After this, and up to the present time, the attention was centered on uric acid as the cause, simply because crystals of sodium biurate were found deposited. The outcome was a flooding of the American and English physicians with literature in which the terms "Uric acid disease," "Uric acid dyscrasia," "Uric acid diathesis," figured largely, and the vaunting by manufacturing houses of innumerable saline eliminants for the cure of the condition. Some of the drug firms even went to the trouble and expense of publishing special journals on the subject, and still, throughout it all, we were dealing only with the cause of the clinical manifestations and not the true etiology of the disease. It seems to us that the time has come when we are justified in looking upon the past of this subject about as follows: Assuming that we have a patient with an acute gouty inflammation of the metatarsophalangeal joint of the big toe, by placing him in bed we rest the joint; the flannel and absorbent cotton dressings of the foot increase the warmth and thus relieve the local pain; the milk and reduced diet lower the food intake and therefore minimize the loading of the circulation with toxins from the intestine and the necessity for their elimination by the emunctories, and the taking of large amounts of fluid internally, the saline purgatives, hot baths, etc., increase the elimination. Treated along these lines the patient finally leaves the bed and is returned to his life activities; but the gout still remains, for we have treated only one of its manifestations. What is the real etiology of gout?

A close personal study of 8 cases of *bona fide* gout showed that every one of them had present a chronic excessive putrefaction in the intestines. In all of these cases the stools were highly acid from butyric acid formation, a product of putrefaction, and hence bacterial and dietetic in its origin. All showed an almost doubled bacterial content, in which the anaërobic organisms,

Gram-positive diplococci and the *B. aërogenes capsulatus* were predominant. The aldehyde test, suggesting parenchymic change in the liver cells, was always present, and a low bile output from the liver, further suggesting a deficiency of the liver functions, was also generally noted. In 2 cases the daily output of urea was normal; in the rest it was subnormal, but in every one the output of uric acid exceeded the normally present 10 grains per diem. While urorosein was at times met with, the ethereal sulphate partitions did not run high as a feature in the cases, and an increased amount of indican was not present in any of them. The arterial pressures were high during the acute attacks, subsiding when the acute manifestations subsided, but never coming down to normal unless the intestinal condition was corrected afterward. During some of the acute attacks the liver, stomach, and intestinal secretions fell off markedly, and then large amounts of indican might be noted in the urine for two or three days.

My inferences from the observation of these cases led me to believe that the cause of gout was a chronic excessive putrefaction in the intestine of the saccharobutyric form, in which the acute attacks came on from a sudden overloading of the general system with toxins from the intestine, but that this intoxication was always more or less present in the intervals. They suggested to me that in these acute attacks the power of the liver to oxidize the purin bases of the body from uric acid into urea was curtailed, and that an output of uric acid salts, instead of urea, was the result. We are all aware that urea is a very soluble substance, capable of going into solution in any of the body-fluids, while the uric acid salts, being most insoluble and therefore difficult of elimination, accumulate in the body and become deposited where the circulation is at its lowest ebb, viz.: in the cartilages of the small joints and the fasciæ.

It must not be understood that gastrointestinal symptoms should be present in all cases of saccharobutyric types of putrefaction in order to make a diagnosis, for the fact is that a study of over 1000 cases shows this not to be so, and, indeed, these persons, clinically speaking, have a most perfect subjective status of digestion. The diagnosis can only be made by most minute and careful examinations of the feces and urine, and then only when the individual has been on a carefully selected diet of weighed quantities of food and fluid for several days.

Believing, as I do, that the cause of gout is to be found in the form of intestinal putrefaction mentioned, I have treated these cases along the lines indicated with the most gratifying results. A diet is selected and made up for each individual in which the proteid content is high, and the sugars and starch-bearing substances low. The entire quantity of food and nourishing fluids is held down to the caloric necessity demanded for the work, age, and weight of the person under treatment, and the articles of diet are taken in amounts weighed and measured each day. In some of the cases the weight of the body was worked down; in others it was maintained stationary, and in one it was worked up fifteen pounds. The daily output of uric acid was measured

every four days, and the diet changed according to the results noted. A definite amount of exercise was prescribed, equivalent to walking seven miles a day, for the purpose of increasing the general oxidation of the body and that of the liver in particular. At least five glasses of water were taken daily, and a twenty-minute immersion bath in warm water every other evening before retiring. Carlsbad salts were used when constipation existed, but no other medicines or alkalies. The patients were all treated by the method of bacterial instillations which the author has advanced,¹ until the bacteriology of the stools was again normal, this latter procedure, in his opinion, being the most valuable therapeutic measure used.

