of the circum-macular region, may be considered as almost pathognomonic. Often low grades of neuritis which seemingly extend some distance back into the nerve-substance can be recognized, these ordinarily appearing monolaterally and generally found in middle aged subjects.

As a rule, uncontemporaneously binocular, generally first appearing in the visual field, and associated with defective vision; the retinal and optic nerve-groupings are not difficult of recognition. Prognosis is never good and incomplete blindness is almost certain.

From this clinical study, it will be seen, as before hinted, that the eyeball, with its accessories, is peculiarly prone to disturbance during the course of general disease; in fact, the organ in its every detail of structure, is liable to changes that are dependent upon gout.

GASTRO-INTESTINAL AND HEPATIC RELATIONS OF GOUT.

Presented in the Section on Practice of Medicine at the Forty-eighth Annual Meeting of the American Medical Association, held at Philadelphia, Pa., June 1-4, 1897.

BY CHARLES G. STOCKTON, M.D.

Professor of the Principles and Practice of Medicine and Clinical Medicine, Medical Department, University of Buffalo; Attending Physician to the Buffalo General Hospital.

BUFFALO, N. Y.

Murchison and Sir Dyce Duckworth, among other students of the subject, have associated the excessive formation of uric acid and the development of gouty attacks with disturbances of the liver. Under the name of lithemia or latent gout, Murchinson described a set of symptoms very commonly met with in this country; among the conspicuous symptoms is the appearance of uric acid, urates and calcium oxalate crystals in the urine in abnormal amount.

These cases of lithemia show evidence of disturbed primary digestion, congestion of the liver, headache, lassitude, malaise, but rarely evidences of deposits, arthritic or otherwise, that are characteristic of true gout.

The question has been raised, and I think justly, is lithemia gout or is it the expression of a toxemia resulting from habitual disorder of the digestive organs including the liver? Before answering the question it may be well to turn to some later views regarding the relation of uric acid to gout, and the origin of uric acid in general.

It would appear to have been conclusively shown by Sir Alfred Garrod, Sir William Roberts, and later by Arthur P. Luff in his recent Goulstonian lectures, first, that "uric acid is not normally present in the blood of man or other mammals nor in the blood of birds; second, that uric acid is normally produced in the kidneys only, and is formed from urea, probably by the conjunction of that substance with the glycocin in the kidneys."

Since the glycocin has its origin in the liver, we can readily understand how hepatic disorder might lead to disturbances in the amount and quality of the glycocin formed. We may therefore understand how hepatic diseases, either with or without gout, may be competent to derange urinary secretions. So it will be seen that an excess of uric acid in the urine does not necessarily mean a gouty diathesis. In fact, it has been shown by Pfeiffer that the uric acid output, instead of being increased in gout, is in point of fact diminished, and the kidneys seem to have lost in part their power of elimination; and although we find in the blood of the gouty individual uric acid in the form of quadurates, and also find the deposits in the joints and other parts not in the form of uric acid, but in that of the biurate of soda, this takes place not because there is uric acid in the blood, but because of some reason that we do not yet understand.

This proposition, namely, that gouty deposits do not follow merely because there is uric acid in the blood, may be proven by the fact that in leukemia, and several other affections in which there is rapid growth and destruction of leucocytes, there is formed a relatively enormous amount of uric acid from nucleinic acid, and yet in these cases we find none of the ordinary symptoms of gout, no deposits of the biurate of soda in the tissues, but we do find the uric acid passing from the body, partly unchanged and partly in the form of urea.

It will thus be seen, 1, that gout does not depend upon the continued presence of the excess of uric acid in the blood; 2, that the formation of uric acid in the kidneys is disturbed in case of gout, probably as a result of some disorder of the liver; 3, that disorders of the liver unaccompanied by gout are capable of deranging the normal uric acid output.

Let us now return to the question already propounded. Is lithemia gout, or is it the expression of a toxemia resulting from habitual disorder of the digestive organs, including the liver? From experience gained in the study of functional disorders of the stomach the conclusion has been forced upon me that the great majority of instances of so-called lithemia are in fact cases of toxemia, in no true sense gouty in nature. As there are many causes of functional disturbance in digestion, so there are many causes of lithemia. It is readily admitted that gout is the occasional cause of the disorder of primary digestion, but the proportion of cases in which it is shown as an actual probability is extremely small.

Perhaps the full import of this position will be more clearly shown when it is pointed out that permanent relief of the condition does not follow the treatment directed toward gout, but that it does follow the right ordering of diet and the right management of the disturbed digestion in other ways. Undoubtedly it is true that digestive diseases aggravate gout, and I think that Luff has shown why it is that indiscretion in diet, particularly as regards certain kinds of food, are capable of exciting paroxysms of gout in gouty subjects. I have carefully and repeatedly studied the stomach contents in a few cases of gout, and find that quite uniformly there occur periods of marked hyperchlorhydria with delayed starch digestion and flatulence, followed by enlargement of the liver, which conditions, if not relieved, are likely to be succeeded by characteristic arthritic attacks.

At other times such indiscretions in gouty subjects excite paroxysms of gastralgia or angina pectoris, the latter usually accompanied by high arterial tension, and sometimes by the discharge of a large amount of pale-colored urine. Again, an acute eczema makes its appearance. I have found that the hyperchlorhydria occurring in the gouty is very intractable to treatment, and those remedies (acting through the nervous system) that generally prove useful in hyperchlorhydria depending upon reflex nervous causes, in gouty patients are of little value. Large and repeated doses of alkalies and potassium iodid give the best result.

Now, such patients may not observe a strict dietary

and may habitually lead sedentary lives. Such indiscretions may provoke attacks of auto-intoxication closely resembling those attacks seen in the non-gouty, yet it can not be too strongly insisted upon that gout is not the most common, but is rather an infrequent cause of such attacks of toxemia. In other words, it is shown that the condition in the gouty patient is distinct and requires a different management from the condition in the non-gouty. This brings us to the consideration of diet in the two classes of cases in question.

To those who have had the widest opportunity for studying gout there is a remarkable uniformity in the belief that all forms of proteids, particularly the dark meats, are objectionable in the paroxysms, and that a diet rich in such substance is likely to precipitate an onset of gout. Fruit juices and the fermented liquors are especially objectionable; on the other hand, farinaceous foods are well borne. In the case of the non-gouty lithemic, albuminoid foods are often the most suitable. Such a patient will find relief from a diet of lean beef, and will suffer if the starchy foods are taken in considerable quantity. It must be acknowledged that individual cases require individual diet and management, and to assume that all cases in which gout is not a factor, will do best upon a nitrogenous diet, is to make a careless generalization.

The careful study of the stomach contents and the adjusting of the diet according to the knowledge thus obtained, together with the frequent examination of the urine, meantime noting the increase or decrease of the body weight, the muscular activity, the state of the nervous system and the feelings of the patient, should be our guides in reaching a knowledge of the correct dietary. It may be set down as a rule that in all cases of lithemia, whether gouty or non-gouty, the fermented liquors and fruit juices are objectionable and sometimes act as a real poison.

An active life out-of-doors has a most beneficial effect on all cases, and this has been used as an argument to prove the identity of lithemia and gout. A little thought will suffice to answer this argument, for it will be remembered that such habits of life are most favorable for good digestion and proper behavior of the stomach, intestine and liver. This rule applies not only to lithemia, but to all digestive disorders.

These remarks are intended to prepare the way for the following conclusions: 1. That gout is a definite disease to which certain individuals are predisposed, but which depends for its development upon causes largely unknown. 1. Laziness and full nitrogenous diet and the use of fermented liquors predispose to the disease. 3. So-called lithemia, as the term is popularly applied, is not gout, but is an auto-intoxication depending upon gastro-intestinal and hepatic derangements. 4. The diet in gout should be largely free from nitrogenous substances. 5. The diet in lithemia must be ascertained by a careful study of the primary digestion, the urine and the general health of the patient, but a nitrogenous diet is often the most satisfactory one.

TREATMENT OF GOUT.

Presented to the Section on Practice of Medicine, at the Forty-eighth Annual Meeting of the American Medical Association, at Philadelphia, Pa., June 1-4, 1897.

BY H. C. WOOD, M.D.

PHILADELPHIA.

I am expected to epitomize in fifteen minutes the wisdom of the ages with regard to the most frequent of all conditions, probably, of the better class of the human race. I want in the first place, however, to clearly develop before you what I myself believe, that all our scientific knowledge of gout at the present amounts to little more than a mass of trundling expectation upon which hereafter shall be built some true knowledge. And I think that in the successful treatment of gout the understanding of this is the basis. There are three great manifestations of the same thing which is universally allied to itself. We have rheumatoid arthritis as one type; we have podagra or true gout, as the second type; and we have acute articular rheumatism as a third type. Let me give you just one illustration from family history, that of my own case, which represents the family history of all the better families in this city which have endured here for generations: A great grandfather leaving his descendants the results of high drinking and living in England, a few dollars and much gout, the one disappearing, the other continuing; a second generation whose history I do not know much of; a third generation, nearly the whole of which dying of gouty degeneration of the cerebral arteries or heart; a fourth generation, some of them developing attack after attack of acute rheumatism, half a dozen, eight, nine or ten in the life history of a single individual; one of them having true podagra; all of them plagued with the various manifestations that we know as nervous gout. There is a relation between these things, not the same thing, but they have the same basis, and this basis absolutely eludes our grasp scientifically.

Now, when we come to treat gout, if we purge ourselves of the false idea which we think we possess, we can recognize the importance of this great principle, not to attempt to treat gout at all, but attempt to treat the individual who comes before us. Let me take simply the question of diet. You know that we inherited from Sydenham the belief that gout was made worse by red meats and that they should not be used. I have seen gouty patients in whom a single piece of ordinary red roast beef would precipitate a furious attack. I have also seen many gouty patients who would not get well until they were put upon red meat. What is the diet for gout? There is no diet for gout. It is diet for the individual. I have seen gouty patients who, if they took starch or sugars, went right down; and I have seen gouty patients who had to take starch and sugars to be built up. Therefore the first principle in the diet of gout is to adapt it to the individual before us. You judge of the case by the effects of experiment. In a large majority of cases sugars and starches have to be cut off. In spare gouty patients starches often do good; farinacious diet may be essential. You have to order your diet according to the individual. A milk diet is one which probably suits the large majority of patients. But that which suits the individual, the stomach, the digestion, will suit the gout or kill the gout.

When we come to the treatment of gout by exercise we find the one thing which does more good than anything else in almost every case, provided we direct the right amount of exercise. If we try to put into an ounce bottle, three gallons of exercise, we crack the bottle. Massage is a form of exercise, and it may be all that your patient can endure; fifteen feet of walking may bring on weariness or it may require some Alpine height. The same story, study your case. Begin with the slightest amount of exercise, but do not let