tremens the mortality is very high. The prognosis is unfavourable when such diseases as diabetes, chronic nephritis, advanced heart disease, and cirrhosis are pre-existent.

Apical pneumonia is generally looked upon as more serious than basal, and as a rule the greater the amount of lung tissue involved the more unfavourable the case. Herpes labialis is often looked upon as a favourable omen, but some of the worst cases I have seen have had this sign. When there is evidence of a thrombus in the heart it is quite the exception to get a recovery, but I have such a case at present under my care in this infirmary.

REFERENCES.

¹ *Twentieth Century Practice of Medicine*. ² *BRITISH MEDICAL JOURNAL*, 1898, vol. 1. ³ *Von Ziemssen's Cyclopædia of Practical Medicine*. ⁴ *Twentieth Century Practice of Medicine*. ⁵ Quoted by Fitz in the *System of Medicine* by American Authors.

NOTES ON THE TREATMENT OF CHRONIC GOUT.

BASED ON EXPERIMENTAL AND CLINICAL INVESTIGATION.

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It is not my intention to discuss the treatment of chronic gout at any length, but to review certain points which seem to me worthy of consideration. Before inviting attention to these points it is advisable to glance at the pathology of this affection.

There is an excess of uric acid in the blood of gouty subjects, and biurate is deposited in their tissues. It is probable that the excretion of uric acid is slightly less in gouty than in healthy subjects, and that there is a marked nitrogen retention, which cannot be accounted for by the slight diminution in the excretion of uric acid. Upon these the only known facts regarding the chemical pathology of gout a number of theories have been constructed. The most plausible is that gout is due to defective elimination by the kidneys, which, owing to some functional or organic derangement, are unable to excrete the whole of the uric acid formed, so that a portion is retained and accumulates in the blood and tissues. It would be more in agreement with clinical experience if the advocates of this hypothesis were to regard the retention as periodic rather than continuous. While this theory is a useful guide to treatment, many facts tend to show that uric acid is not the predominant factor in the causation of gout. The safest course is to regard gout as an expression of perverted metabolism; in other words, the outcome of excessive formation or periodic retention in the organism of one or more of the intermediary or end products of nutrition.

Since uric acid at least plays an important incidental part in the pathology of gout, it is necessary to allude to its supposed antecedents and place of formation. There are three ways in which it is supposed to be produced:

1. By the liver conjugating the ammonium salts and lactic acid.
2. By the kidney synthesising urea and glycine.
3. By the decomposition of nuclein.

There is very little evidence in favour of the theory of the formation of uric acid by the kidney. Its supporters relied to a large extent upon the fact that hitherto uric acid has not been found in the blood of normal mammals; but as the methods employed for separating uric acid from blood are unreliable, this argument is scarcely valid. They have further argued that the fact of various observers having failed to detect uric acid in bird's blood lent considerable support to their hypothesis, but Dr. Chalmers Watson has recently demonstrated its presence in the blood of different birds. Schroeder maintains that uric acid cannot be formed in the kidney. He extirpated the kidneys in hens and snakes, the hens surviving the operation from six to twenty hours, and the snakes from five to ten days, and in both cases he demonstrated an excessive accumulation of uric acid in the tissues. The researches of Minkowski, on the other hand, show that in birds the greater portion of the uric acid is formed by the liver. He removed the livers of geese, and observed that whereas in normal geese the uric acid formed from 60 to 70 per cent. of the total waste nitrogen, in those experimented with the proportion fell to from 3 to 6 per cent. The waste nitrogen in

these birds appeared as ammonia, and was accompanied by a large quantity of lactic acid sufficient to render the urine strongly acid. The experimental results in regard to mammals are not sufficiently conclusive as to the organ chiefly concerned in the production of uric acid.

The evidence as to the genesis of uric acid from nuclein or nucleo-proteid since Horbaczewski's classical researches has been gradually accumulating, and, although contradictory, points to the fact that nuclein is the mother substance of uric acid and alloxur bases; and as there are grounds for supposing that one or more of the bases may be a factor in the production of gout, their origin must have an important bearing on the dietetics of gout.

In treating chronic gout our aim should be to prevent the formation of abnormal proteid decomposition substance, and the accumulation of these and other waste products in the system; and to promote their elimination. *Diet.*—Unfortunately, there is considerable diversity of opinion concerning the most suitable articles of diet for gouty subjects. While I do not believe in any hard-and-fast rules for all cases, there is one which should not be forgotten, namely, moderation. In certain cases this and that may be forbidden, but until the total quantity is restricted no progress is made. In the gouty the very greatest importance should be attached to digestive troubles, and the diet should be carefully adjusted to the capabilities of the patient's digestive power. Gout being in all probability due to some error in metabolism, it is essential that any disorder of the alimentary tract and its annexa should be met by appropriate medicinal and dietetic treatment. The amount of exercise the patient habitually takes must be considered when determining the diet. As it has been shown that a diet consisting largely of peas and beans is capable of producing more alloxur bodies than one composed almost exclusively of animal food, gouty patients should be advised to partake sparingly of leguminous food, and foods rich in nuclein. Those who advocate a purely vegetarian diet in gout do not take a sufficiently broad and comprehensive view of its pathology and treatment. Proteids whether they be of vegetable or animal origin are absolutely necessary, and the essential point is to give the kind of proteid most easily digested, and just that amount which will be digested with probable certainty in the normal time. Carbohydrates have no influence on the uric acid excretion, but their liability to produce acid fermentation, and to retard proteid digestion is to be remembered. Fats should be curtailed, as they are prone to produce indigestion. A mixed diet is the most desirable, providing the digestion admits of it. If regular action of the bowels cannot be obtained without artificial means drugs having a cholagogue action should be used.

Regarding the deleterious influence of alcoholic beverages there is absolute unanimity; but the *rationale* of this pernicious effect is still conjectural. Although it is advisable as a rule for gouty patients to abstain from alcohol, there are a certain number of cases, chiefly of the asthenic type, where it is necessary to order a stimulant with one or more meals. The alcoholic beverages which are the least injurious are probably well-matured whisky and good sound claret, but individual idiosyncrasy has to be considered. Chittenden, Cameron, and Levison found that a moderate quantity decidedly increased the excretion of uric acid. My own experimental observations do not support that statement. During one series of observations whisky and port wine were tried, and on another occasion claret and port, without any effect on the uric acid output.

Exercise out of doors is of undoubted value in chronic gout, and during inclement weather indoor exercise is not to be despised. Levison's assertion that the uric acid excretion is markedly increased by exercise requires modification. If a man of sedentary habits takes a moderate amount of exercise the excretion of uric acid is increased for the first day or two, but if the exercise be continued daily the increase is not maintained. The stimulating effect of regulated exercise upon metabolism is an agent of considerable power in preventing the various manifestations of gout.

Sodium Salicylate.—If the retention theory be accepted, our object should be to diminish the production of uric acid in order to relieve the kidneys. It seems very inconsistent for some of the supporters of this theory to advocate the use of

sodium salicylate in gout, a drug which undoubtedly increases the excretion of uric acid both in health and in gout. Various theories have been propounded to explain this increase. Lecorché, Haig, and a number of other observers say that the drug eliminates from the blood and tissues the excess of sodium urate. If this explanation be correct, there must be an excess of urates circulating in the blood of healthy people, and that cannot be admitted. Further, the P_2O_5 and alloxur base should not show a corresponding increase if this view were correct, but as a matter of fact they are also increased. Luff thinks the drug increases the uric acid by transforming glycine into salicyluric acid, and that this in its turn combines with urea to form uric acid; therefore he disapproves of its use in gout. I have grounds for believing that it increases the uric acid at the expense of the leucocytes and other tissue cells, and if there is evidence of renal disease the drug should not be given. Of course this opinion is based on the assumption that the kidneys, when organically deranged, are unable to eliminate the whole of the uric acid formed in the body. Some think that so long as the excretion of uric acid is increased the patient must benefit, but though the increase may be beneficial, it may not. Pilocarpin increases the uric acid excretion, but no one, so far as I know, advises its administration in gout. It is true that sodium salicylate relieves the joint pains in gout—how we do not know—but as it increases the formation of uric acid we ought to consider whether its advantages outweigh its disadvantages before recommending it.

Colchicum does not seem to have an invariable effect on the excretion of uric acid, yet few doubt its beneficial action in acute gout, and it is also useful in some cases of chronic gout.

The Harrogate Old Sulphur Spring diminishes the excretion of uric acid, and this diminution is not due to retention, as evidenced by the increase in the leucocytes and the gradual rise to the original level after cessation of the waters. Whether this is the explanation of the salutary effect in chronic gout, or whether it is owing to its cholagogue action, I am not prepared to say, although I am inclined to think that it is due to its influence in accelerating hepatic metabolism. When given in conjunction with the massage douche, gouty patients experience considerable relief from their troubles. Doubtless there are some who may attribute the improvement to the douching with hot water and massage; but, having prescribed the sulphur water without baths or other adjuvants, I can testify to its beneficial influence in such cases. It may be remarked here that if the same routine form of treatment be meted out to all cases of chronic gout, disappointments are certain to occur—in gout the individual should be considered just as much as the ailment.

Potassium iodide is a drug of peculiar value in chronic gout. It appears to retard the development of cardio-vascular and renal changes, and in cases associated with albuminuria there is a diminution in the amount of albumen during its administration. Although it has no influence on the solvency of biurate, it is useful in reducing the inflammatory thickening around enlarged joints. It should be given cautiously, as some patients are very susceptible to it, and it is contra-indicated in advanced renal cases.

Quinine is supposed to diminish the excretion of uric acid, but in an experimental investigation I found a slight increase. Apart from any influence it may possess on the nitrogen excretion, it is an exceedingly useful tonic.

Guaiacum.—It is rather remarkable that Sir W. Roberts in his articles on gout does not mention guaiacum in connection with the treatment of this disease, though years ago Sir Alfred Garrod expressed a decided opinion regarding its prophylactic power, and other observers have since then testified to its efficacy in preventing acute paroxysms. It is not an uncommon experience to meet with patients who periodically take guaiacum on their own account with the happiest results so far as their arthritic attacks are concerned. As an eliminator of uric acid there is probably no drug to equal it. Guaiac resin should be taken in compressed form, in doses of 20 grs. twice a day for about a fortnight. After an interval it may be repeated if necessary. It is not advisable to continue its administration uninterruptedly for a prolonged period.

Alkalies.—A good deal of misconception has existed concern-

ing the part played by alkalies in the treatment of gout. That they are of considerable value in the gastric and hepatic complications of gout must be admitted, but that they have any particular influence on the gouty diathesis, or that they increase the excretion of uric acid or the solvency of biurate must be denied. They may possibly have some indirect effect on gouty processes by their action on metabolism, but this is difficult or impossible to prove by experimental investigation. These remarks refer to alkalies in general, and not to particular alkaline salts. It was believed for some time that the alkalinity of the blood was markedly diminished in gout and alkalies were given in order to correct this, but we now know that the alkalinity of the blood is either normal or slightly increased. Hence the treatment of gout by alkalies is practically restricted to disorders of the alimentary tract. The deductions Sir William Roberts and Dr. Luff drew from their experiments regarding the alleged superiority of potassium over sodium salts in gout cannot be accepted without clinical corroboration. These deductions are based on the supposition that the administration of alkaline salts will alter the alkalinity of the blood, and, therefore, that the sodium salts will hasten the conversion of the so-called quadriurate into the gelatinous or crystalline biurate. I have used the words so-called quadriurate advisedly, because it has not yet been proved that uric acid exists in the blood in this unstable form. It is possible that uric acid may enter into combination with some other constituent of the blood than the alkaline salts. On a dozen occasions I have prepared the sodium quadriurate according to Roberts's instructions, and no two samples reacted alike with his standard solution at the body temperature—in some the precipitation occurred in a few minutes—in others it varied from a quarter of an hour to an hour; therefore, experiments performed with such an unstable compound must be more or less unsatisfactory.

One of the most characteristic properties possessed by the blood is its uniformity of composition, and in no particular is this more marked than in the maintenance of its normal alkalinity. Sir Michael Foster states that the alkalinity cannot be increased by the administration of alkalies. If a large quantity of alkali is given to animals it is quickly excreted by the kidneys. Until it has been demonstrated that the alkalinity can be increased by medicinal doses of alkaline salts, it would be safer so far as particular salts are concerned to be guided by clinical experience, and it appears to me that the following sentence by Sir William Roberts supports that contention:

I have repeatedly administered the bicarbonate and citrate of potash continuously for three or four years, and in sufficient doses to maintain the urine persistently alkaline, yet I have seen the arthritic attacks recur with unabated vigour.

Regarding biurate solvents I do not know of any one superior to pure water, therefore I strongly approve of gouty patients taking a moderately large quantity of water between meals. Dr. Tunnicliffe found from laboratory experiments that tartrate of piperidine had a marked solvent effect on sodium biurate, but I do not think it has been shown clinically to exhibit this effect.

In ascertaining the efficacy of a supposed biurate solvent it should be given both to a healthy and to a gouty individual, while on a fixed diet. If it has no effect on the uric acid in the healthy, but increases the excretion of uric acid in the other case, we are justified in assuming that the drug either eliminates the excess of urates in the blood or increases the solvency of biurate, and subsequently effects its elimination.

MUNICIPAL SANATORIA IN CANADA.—According to the *Canadian Journal of Medicine and Surgery*, an Act has been passed by the Ontario Legislature to provide provincial aid for the establishment of sanatoria for consumptives. It enables the councils of counties, cities, or towns separated from counties, to establish sanatoria for the treatment of persons suffering from consumption; provides for the management of the sanatoria by a Board of not more than five trustees; enables municipalities to combine for the purpose of establishing sanatoria, and authorises the Lieutenant-Governor in Council to make a grant not to exceed one-fifth of the sum expended on the site, and not more than 4,000 dollars altogether, for the purpose; the site and plans to be approved by the Provincial Board of Health.