can therefore easily realize that, given circumstances such as I have narrated in the foregoing cases, the occurrence of glycosuria is just what we should expect. Sugar in the urine of young persons, even without the presence of any symptoms of diabetes, such as wasting, thirst, etc., is a much more important matter than when occurring in people past middle life, and in some cases, I fear, could we follow out the clinical history, would be found to end in diabetes.

We have at present no definite means of arriving at a sound prognosis, but if there has been some distinct breach of the laws of healthy living—a breach that can be overcome—and if the response on the part of the system is ready and complete when the cause has been removed, then I think we can take a hopeful view.

I have recently seen a rather delicate boy of one of our public schools whose urine showed a trace of sugar but no albumen. He had worked very hard during the term before Christmas, and when at home for the holidays seemed tired, thin, and not up to the mark. Examination showed a trace of sugar in the urine. I felt that the only thing was to advise his removal from school for a term, to diet him carefully, and to have the urine looked to periodically for some time to come. I confess that I am somewhat uneasy as to the future history in this lad's case, although at present he keeps well.

Another case I may mention, because the patient is doing well, and it is now eight years since I first saw him.

He was then 22, out of health, and the urine, of moderate density, contained a trace of albumen and of sugar. Last year the patient's general condition was satisfactory, and the urine was free from albumen and from sugar.

The occurrence of sugar in the urine in young subjects must always occasion us some anxiety, even if it does not persist, for the reason that we feel that there is a risk of its passing on to diabetes, a disease so often rapidly fatal in the young.

CONCLUSIONS.

1. I think it may be said that the presence of albumen in any appreciable amount in the urine is not normal or physiological, but that it does not necessarily imply coarse pathological change, and that it does not necessarily mean that the existing disturbance of function is permanent or progressive. At the same time, it cannot be denied that a certain number of cases which at first are rightly placed in the above category do later on show signs of definite organic kidney disease.

2. That the presence of sugar in the urine in any appreciable quantity is abnormal, that in the young it is of serious import, and that if persistent, it is likely to lead on to diabetes; that in people past middle life, and especially in those of gouty type, it is of less consequence and usually yields to treatment more or less speedily, to recur, however, in some cases, under conditions similar to those under which it first appeared.

3. That the presence of both albumen and of sugar in the urine indicates serious disturbance in the metabolic processes, calling for relief to the nervous strain which the patient may have been undergoing, and an adjustment as far as can be of his environment, but that under favourable conditions these patients may continue in at least fair average health for many years.

CLINICAL ESTIMATIONS OF PURIN BODIES IN GOUTY URINES, ETC.

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DESPITE the numerous publications upon the metabolism in gout and the allied conditions, the advance towards the real etiology of the disease is very slow. For practical purposes it still is necessary to confine our attention to the eliminated products, although, from the theoretical standpoint, the intermediary changes of metabolism now form the chief centre of interest. The substances resulting from the decomposition of proteids and nucleins by bacteria and ferments are at present being carefully investigated, and already it has been demonstrated that certain micro-organisms and ferments possess the power of breaking down amino-purins into simpler substances and of oxidizing the latter into uric acid. Evidence has

also been adduced to show that in extracts of the spleen, lung, liver, intestine, muscle, and kidney, there exist hydrolytic ferments which, in the presence of a sufficiency of oxygen, are able to transform the amino-purin to an oxy-purin, and finally to uric acid. In kidney, muscle, and liver extracts ferments which are able to break up the newly-formed uric acid are thought to be present. Whether such conditions obtain in the living organs is not yet determined. Ferments are able, we know, to act in a reverse direction, and therefore the effects of their action *intra vitam* may be quite different from that ascertained from extracts of organic tissues. The relation of the ferments to the large quantities of hypoxanthin produced in the muscle tissues during ordinary muscular contraction is also at present undetermined.

During the period necessary for the accumulation of facts ascertained by experimental investigation and of the records of the elimination of endogenous purins, we may do something towards the betterment of the gouty individual by an endeavour to introduce an element of exactness into the measures for dietetic treatment. If, in the meantime, we agree to practically neglect uric acid in regard to the etiology of gout, we cannot yet overlook its importance in regard to treatment, since it expresses the degree of hepatic sufficiency and thus indicates the amount of tissue reaction to the general toxæmia. There are numberless theories from which our choice of causation may be made, but the only, or almost only, well-grounded reason for prophylactic measures is the presence of an excess of uric acid in the tissues. So far as we know to-day, we are unable to prescribe any medicinal treatment which directly affects the deposited uric acid. We are, therefore, driven to regard dietetic treatment as the at present only available resource, and to recognize its great possibilities. The determination of the individual capabilities for certain foods has already mitigated the severity and recurrence of the attacks in many cases of gout. The value of the exact estimation of the individual factor, as opposed to the haphazard construction of dietaries, lies in the fact that it is of supreme importance to the gouty individual that he should be sufficiently nourished, and that his daily food should approach, as nearly as possible, the régime to which he has been heretofore accustomed. A knowledge of the "personal powers" often permits a greater choice of foods than one may dare to allow when giving empirical directions. When the patient is able, within certain limits, to follow his fancies and usual habits, his mental condition is less constrained than is the case with a patient who is placed under a rigid or a severely-restricted dietary. This recognition of the mental factor is of much comfort and importance to the gouty patient, and possibly tends to sustain the nervous control of the liver functions.

Since 1902,¹ when I first suggested the determination of the individual powers of assimilation for purin substances, I have had the opportunity of examining many gouty cases for this purpose, and have had the satisfaction of observing a retardation of the course of the disease and a diminution in the number of the acuter manifestations. I append an example, additional to those recorded in 1903.² The Table I indicates the procedures followed and precautions observed.

TABLE I.

Days.	Dietary.	Urinary Purin Nitrogen.
1	Diet of milk, bread, cheese, eggs, milk, oats, etc.	0.2010
2	" " " " " " " "	0.1560
3	" " " " " " " "	0.1600
4	Plus ½ lb. beefsteak=0.25 gram purin nitrogen	0.2560
5	Purin-free diet again	0.1760
6	" " " " " " " "	0.1650
7	" " " " " " " "	0.1560
8	Plus ½ lb. beefsteak about=0.50 gram purin nitrogen	0.2463
9	Purin-free diet	0.1560
10	" " " " " " " "	0.1800
11	Plus ½ lb. beefsteak=0.28 gram purin nitrogen	0.2445

In this case it will be observed that, while the addition of 4 oz. of best beefsteak (weighed raw) was followed by an immediate output of about 38 per cent. of the purin ingested, the addition of 8 oz. of beef resulted in an output of only 16 per cent., the remainder being retained in the system. Taking 40-55 per cent. as an

average normal output, it will be at once seen that this patient could well deal with 3 oz. of steak or its equivalent in other less purin rich meats, and that the intake of flesh meats need not be further restricted. As a matter of fact, some six months later another estimation showed that the tolerance capacity was increased; the allowance of flesh meat was accordingly raised, and the intestinal agencies continued adequately to discharge their functions.

It is, of course, a great advantage when these determinations can be made by exact laboratory methods. As such processes, however, are very tedious and entail the use of apparatus not always available, I suggested, in 1901,³ an instrument—the purinometer—by means of which the rougher clinical estimations could be made. An extended experience with this method of sedimentation has revealed certain drawbacks, but it has also shown that for ordinary purposes sufficient accuracy may be obtained by it. The following figures, additional to those already published,⁴ have been kindly supplied to me by independent workers:

TABLE II.

Total Purin Nitrogen (Purinometer).	Uric Acid Nitrogen (Hopkins's Method).	Purin Bases Nitrogen (Calculated by Difference).
0.2164	0.1735	0.0429
0.1776	0.1632	0.0144
0.1831	0.1702	0.0129
0.1716	0.1531	0.0185
0.1732	0.1410	0.0322

TABLE III.

Purin Nitrogen (Purinometer).	Uric Acid Nitrogen (Hopkins's Method).	Purin bases Nitrogen (Calculated by Difference).
0.1629	0.1365	0.0264
0.1423	0.1095	0.0328
0.1319	0.1064	0.0265
0.1284	0.1095	0.0189
0.1367	0.1140	0.0227
0.1673	0.1344	0.0328
0.1795	0.1435	0.0360
0.1556	0.1293	0.0263
0.1496	0.1381	0.0115
0.1468	0.0753	0.0715
0.1401	0.0812	0.0588
0.1903	0.0976	0.0929

These results, which accord with a vast number of similar estimations made by myself, show that the readings yielded by the purinometer fairly approximate to those obtained by Hopkins's method.

Rilalowski, however, in a St. Petersburg dissertation submitted in 1904, records a series of results obtained by the purinometer, by Hopkins's and by Salkowski's methods, and considers that the purinometer is only suitable for coarse estimations, since it yields an error of from - 50 to + 50 per cent. (For the interpretation of the Russian text I am indebted to Docent Dr. B. Slowtsoff, St. Petersburg.) Rilalowski also mentions several disadvantages, all of which were anticipated and allowed for when the instrument was graduated, and were included in the original description of it. He proposes to modify the instrument by substituting for the two-chambered tube a single graduated measure, into which the phosphate-free urine is placed for treatment with the silver solution. No values for his new graduations are appended. The purinometer was originally designed in order to obviate the use of two separate tubes and the necessary filtering, etc.

Rilalowski relies chiefly upon the following figures (see Table IV), taken from his Table III.

In this table the purinometer is compared with Salkowski's method for uric acid, Hopkins's method being used to control the estimations by Salkowski's method. For absolute comparison, Rilalowski determines the purin bases by Salkowski's method, and adds together the quantities of uric acid and purin bases in order to arrive at the total purin bodies.

If for a moment we leave the purinometer out of the question we may note the experiences of Rilalowski with the methods of Salkowski and Hopkins. In a few instances the results obtained are very similar, but in

TABLE IV.

Total Purins (Purinometer).	Uric Acid (Salkowski).	Uric Acid (Hopkins).	Percentage Difference between Hopkins and Salkowski.
0.2340	0.1480	0.1480	
0.1112	0.1425	0.1472	+ 3.5
0.0802	0.1426	0.1446	+ 1.4
0.0661	0.2974	0.3075	+ 3.4
0.5382	0.3066	0.3143	+ 2.3
0.2231	0.0920	0.0613	- 33.0
0.0720	0.0920	0.1680	+ 82.0
0.2004	0.1026	0.2030	+ 96.0
0.1125	0.1268	0.1066	- 16.0
0.2340	0.1266	0.1840	+ 45.0
0.1170	0.0590	0.1263	+ 11.0
0.1248	0.0746	0.1083	+ 45.0
0.0546	0.2304	0.3056	+ 32.0
0.1365	0.2634	0.1926	- 36.0
0.1768	0.2881	0.3380	+ 17.0

others the figures represent differences of - 33 to + 96.8 per cent. Yet these two methods are usually recognized as the best available methods for the estimation of urinary uric acid, and in skilled hands they yield almost identical results. If the findings of the purinometer are judged by the estimations of uric acid or of the total purins obtained with Salkowski's processes, the method of Hopkins is made to appear far less reliable than that used for the purinometer. On the other hand, if we accept the figures obtained with Hopkins's method, then Salkowski's are equally at fault.

It may be conceded that the principle of sedimentation from urine is not capable of yielding absolutely accurate results. The density and constitution of pathological urines are constantly changing, thus tending to alter the rate and extent of precipitation. By sedimentation it is not to be expected that results can be obtained that shall be comparable with those afforded by the usual laboratory methods. But although the changed conditions may be marked in different urines, they are more or less constant in individual cases, so that the principle of sedimentation permits relative, if not actual, estimations to be made with sufficient accuracy for clinical purposes.

If the following precautions are taken, the user of the instrument will obtain information sufficiently indicative for the prescription of purin-rich or purin-free dietaries.

The tale used in making up the solutions must be finely ground pure talc and not "prepared French chalk."

After adding each solution the apparatus should be vigorously shaken and the precipitate then allowed to settle.

The final sediment should be uniform. If small gaps appear or the precipitate does not uniformly fill the lumen of the tube, then the apparatus should be again shaken, and the precipitate be allowed again to settle.

The reading should always be taken the same number of hours after precipitation.

The urine should be taken from a mixed sample of the total daily urine.

If albumen is present it is better, although not absolutely necessary, for it to be removed by boiling in the usual manner.

The apparatus must always be kept away from the light during the estimation. It is well for the instrument to be cleaned immediately after use, in order to avoid the formation of a sediment upon the sides of the glass tube.

When not in use the stopper and the plug of the stopcock should be kept apart from the tube, or kept loosely in position by the interposition of a piece of thin paper.

REFERENCES.

- ¹ *Purin Bodies of Foodstuffs*, Sherratt and Hughes, 1902, and Second Edition, 1903, p. 124. ² *Practitioner*, 1903, vol. lxxi, No. 1, p. 79. ³ *BRITISH MEDICAL JOURNAL*, vol. ii, November 1st, 1902. ⁴ *Purin Bodies of Foodstuffs*, p. 147.

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ON CHEMIOTAXIS.

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HISTORICAL.

THE study of chemiotaxis—the taxis or arrangement of motile organisms induced by chemical substances—was first commenced about twenty years ago. Historically, it may be said to take its origin to a considerable extent out of investigations on the allied phenomenon of galvanotaxis exhibited by free-swimming organisms. It is, therefore, desirable to refer briefly to the researches which have