epithelial layers. When associated with remains of disintegrating nuclei their presence is detected with difficulty.

6. In smears from vesicles bodies the same as those discussed in paragraphs 4 and 5 or at least having like staining reactions are also found and in the sections of the skin taken in the papular stages of the eruption similar bodies are present in the lymph exuded among the epithelial cells. Bodies having the same characters are sometimes present in small numbers in the blood of patients suffering from haemorrhagic small-pox.

7. In smears taken from the pocks in the later stages of the eruption, within certain of the cells small clear bodies may be seen which stain very faintly with ordinary reagents. In smears of gradually-increasing age these clear bodies are seen to increase in size until they occupy the greater part of the cell, and in smears taken at still later date these bodies are found apparently in a free state. They still stain very faintly, and present no appearance of a nucleus.

8. The absence of pyogenic micro-organisms, both from smears and skin sections, is very striking in the haemorrhagic cases, and during the earlier stage of the rash in those cases in which the eruption ultimately became pustular. Out of seventeen haemorrhagic cases examined, only three showed pyogenic organisms in sections of the skin. In one of them cocci were present in the vessels in which they formed minute thrombi; in a second case a bipolar-staining bacillus was found in groups in the corium; and in a third case both cocci and bacillus just referred to were associated.

9. Towards the end of the vesicular and in the pustular stage pyogenic organisms are frequently found in sections, smears, and cultures.

10. Appearances similar to those described as being present in smears, vesicles, and pustules of the skin in variola occur also in varicella.

In haemorrhagic and in confluent cases of small-pox, pyogenic organisms are very commonly found in the blood; these, however, are probably to be regarded as a result of accidental contaminations of the blood stream by absorption of organisms, in haemorrhagic cases probably from the lungs, in sections of which cocci are found in immense numbers, and in confluent cases by absorption from the skin, and possibly from the lungs also.

AN IMPROVED METHOD OF TESTING FOR LEAD IN URINE,

WITH SOME NOTES ON THE CONTINUED PREVALENCE OF DIACHYLON POISONING IN THE MIDLANDS.

BY

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and

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THE object of this paper is threefold:

I. To remind members of our profession how common is the use of diachylon as an abortifacient in the midlands.

II. To criticize the magnesium wire deposition test for lead in urine.

III. To bring to notice a modification of the electrolytic test of great delicacy and simplicity, which has been used by Mr. S. R. Trotman for some time with much success.

I. THE PREVALENCE OF DIACHYLON POISONING.

This pernicious practice appears to have been growing since it was pointed out by Dr. Pope, of Leicester, in 1893. Dr. George Crooke showed its prevalence in Birmingham in 1898. Dr. Bell Taylor records a case in Nottingham in 1898. Dr. W. B. Ransom published notes of a number of cases in Nottingham in 1900. Dr. Wrangham records among others a case from Sheffield in 1901. Dr. W. Scott published in the *Sheffield Quarterly Medical Review* in 1901 notes of 27 cases occurring in the practice of the General Hospital, Nottingham.

I myself have seen 18 cases; these have presented most diverse symptoms, as shown in the following list:

(*a*) Previous diagnoses: Cerebral tumour, 1 case; intestinal obstruction, 3 cases; ruptured tubal pregnancy, 1 case; anaemia with gastritis, 1 case; renal calculus, 1 case.

(*b*) Most prominent features of other cases: Optic neuritis, 2 cases, one ended in atrophy with absolute blindness, the other completely recovered; acute mania, 2 cases, one commenced with a series of epileptiform convulsions—both recovered after about six months; severe abdominal pain and vomiting, 4 cases; severe anaemia with milder abdominal symptoms, 3 cases.

They all presented a severe anaemia with a characteristic faintly yellow complexion and the great majority a fine tremor of hands, lips and tongue.

In the great majority of these cases an examination of the gums decided the diagnosis with a finality from which there was no further appeal. There are, however, a certain number of cases in which the gums fail to give the sought-for proof. This arises from two causes.

1. The cause common to all lead poisoning, that scrupulous cleanliness of the gums prevents the formation of the sulphuretted hydrogen necessary to the formation and deposition of the lead sulphide. This occurs usually in the more youthful unmarried women.

2. I have seen one patient in whom the black punctate line was very faint and certainly not decisive (surely the term "blue line" is very misleading for it consists of a series of black dots absolutely distinct from the truly blue line of defective cleanliness), yet three weeks later—during which she had no opportunity of taking more pills—the line was beyond dispute. Mr. Chicken tells me that he has seen a case of diachylon poisoning in which the symptoms of lead poisoning preceded the appearance of the blue line by three weeks. It is therefore, as one might expect, a fact that the line may take some time in appearing and its absence may easily mislead one.

It is therefore of advantage to possess some other means for demonstrating the presence of lead.

II. THE MAGNESIUM WIRE DEPOSITION TEST.

In the *Lancet* of January 16th, 1899, Dr. Hill Abram published a modification of von Jaksch's test for lead in urine, which appeared almost as simple as testing for sugar; this I adopted some years ago, and was charmed with it, until I tried it with normal urine, with which it gave an identical result. Last year, under the kind tuition of Professor Kipping I endeavoured to obtain crystals of lead iodide from the yellow deposit obtained by this test from the urine of cases of severe plumbism and never once succeeded, yet I did obtain them from solutions of lead in water of 1 in 50,000 dilution as stated by Hill and Marsden.

The reasons for this apparent anomaly I believe to be as follows:

1. Dixon Mann states that the usual amount of lead excreted in the urine in cases of chronic lead poisoning is less than 1 milligram per diem, and assuming the quantity of urine to be 1 litre, lead is present in a dilution of less than 1 in 1,000,000. Mr. Trotman has confirmed this in two cases of chronic plumbism occurring in painters. The urines contained 0.032 and 0.006 milligram per litre respectively. It follows that a test having a limit of 1 in 50,000 will fail.

2. Recently in a case of diachylon poisoning Mr. Trotman has found in the urine 32 milligrams of lead per litre; this is equal to 1 in 26,000 solution, yet here he failed to obtain the magnesium wire test; and it was in similar cases that I failed to obtain the test.

In these cases some other explanation is needed. Now it is well known that lead is excreted in organic combination, and that on this account it fails to give the ordinary chemical tests; this no doubt is the true explanation of this failure.

III. THE ELECTROLYTIC TEST.

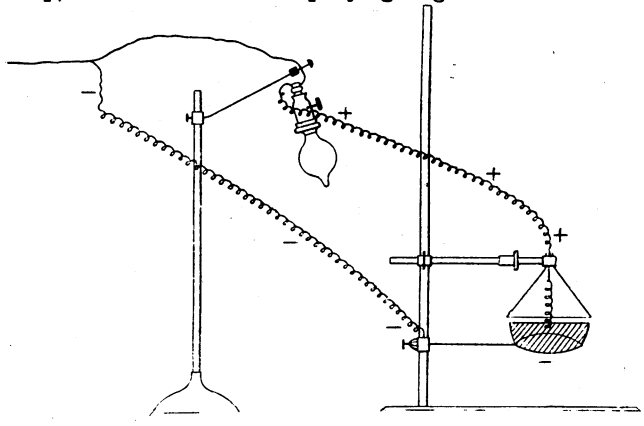
I have recently been fortunate in obtaining the aid of Mr. S. R. Trotman, our city analyst, who has not only confirmed my results with the magnesium wire test, but has tested a number of suspected urines by a modification of the electrolytic test which is both simple in manipulation and of extreme delicacy, and of which he gives the following account:

"It is unfortunate that the destruction of organic matter is essential, since the necessity for so doing often precludes the possibility of a rapid chemical diagnosis, such as could be carried out by a medical man unsupplied with laboratory apparatus. The need of rendering the combination of the lead less complex than it is in the albuminate of the urine is not, however, to be wondered at when we remember that delicate chemical, and particularly electro-chemical, reactions depend very largely upon the dissociation of a compound into its ions. Organic matter is commonly destroyed by means of hydrochloric acid and potassium chlorate, nitric acid, or other oxidizing mixtures. In the case of urine partial destruction only is required. The solution thus obtained is filtered and the lead separated by means of sulphuretted hydrogen or a double cell. Dixon Mann recommends that the liquid be placed in the inner portion of a

double cell, and dilute sulphuric acid in the outer. The cell is connected with the terminals of six Grove cells and the current allowed to run for six or eight hours. This method is perfectly satisfactory, but is capable of several minor improvements which greatly increase the simplicity of the test without altering its delicacy. The following are details of the exact method we have adopted with most satisfactory results.

"About 250 c.cm. of the suspected urine are placed in a porcelain dish (free from lead), and a few cubic centimetres of lead free sulphuric acid. A small flame is placed beneath the dish, and about 5 grams of potassium persulphate (KSO_4) are added little by little, with occasional stirring, the temperature of the liquid being gradually raised to 100° C. In about half an hour the contents of the dish will have become almost colourless, owing to the oxidation of the organic matter.

"Potassium persulphate is now a commercial product, and possesses many advantages over other oxidizing agents. Its substitution for hydrochloric acid and potassium chlorate does away with the evolution of noxious fumes and the prolonged boiling necessary to get rid of excess of the latter. When the liquid has concentrated to about 50 c.cm., it is introduced into a platinum dish, and an electric current passed through it for about eight hours. The current is obtained from the main supply by first running it through a lamp, as shown in the accompanying diagram:



"Before commencing the experiment the poles are tested by introducing the terminals into a solution of salt containing a few drops of phenol phthalein. After allowing the current to run for a sufficient time (conveniently over night) the liquid is poured away, and the dish well washed with distilled water. Hydrochloric or nitric acid is then added, and after digesting on the water bath or heating over a bunsen flame, a stream of sulphuretted hydrogen is passed through the liquid. The quantity of lead present may be accurately determined by matching the colour with a standard solution of lead nitrate. Great care must be taken to obtain lead-free sulphuric acid, and every fresh batch of materials should be tested by a blank experiment. As before mentioned, by employing the test in this way the most minute quantities of lead have been easily detected in specimens of urine when other tests have given negative results."

I have to thank the staff of the General Hospital for allowing me to make use of cases which I saw there while house-physician.

THE INFLUENCE OF SOME MODERN DRUGS ON METABOLISM IN GOUT.

BY WILLIAM BAIN, M.D., M.R.C.P.,
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THE increasing attention paid to variations in the constitution of the urine in gout has been partly directed to the relative proportions of the nitrogenous end products. From the therapeutic standpoint modifications induced by different substances in such urines have been studied by many observers. It is obvious that if drugs having a beneficial effect on the disease produce variations of a similar kind, a basis is provided from which inferences can be drawn in regard to the question of its causation. Among drugs employed in the treatment of this affection are a number which have been extensively recommended in recent times, and *a priori* might be expected to produce similar quantitative modifications of the urine. Such are tartrate of piperidin, benzoate of lithium, piperazin, lysidin, urotropin, sidonal, and colchi-sal. With the main object of ascertaining the influence of the drugs mentioned on metabolism they were administered to a case of chronic gout, the patient being on a fixed diet during the investigation. The general result was to indicate an absence of uniformity; a remark which applies with special force to the excretion of uric acid.

In an earlier case the investigation was rendered abortive through the development of a subacute attack of gout. This case is however included in the paper, owing to a special point of interest. It will be unnecessary to give more than the briefest clinical details of these two cases. As the methods of analysis and diet were similar in both cases they may be described now.

Methods of Analysis.—The urea was estimated by the hypobromite method, and the uric acid by Hopkins's process. For the estimation of the acidity titration by decinormal caustic soda was adopted, phenolpthalein being the indicator. For the P_2O_5 titration with uranium nitrate was employed.

The sulphates were estimated as follows: To 200 c.cm. of filtered urine 10 c.cm. of hydrochloric acid was added, followed by an excess of a saturated solution of barium chloride. The flask was shaken, and the precipitate allowed to settle. It was then separated by filtration, and washed chloride free with cold distilled water. The filtrate and washings were boiled for fifteen minutes and allowed to stand over night. In this case also the precipitate was washed chloride-free with hot distilled water. Both precipitates were further washed with alcohol and ether, dried and incinerated in a weighed capsule, the filter papers being of known ash.

Blood.—The films in Case I were fixed with formalin vapour, and stained with eosin and Loeffler's methylene blue. In Case II the films were fixed with heat (120° for about fifteen minutes) and stained, as in Case I.

Fixed Diet.—Breakfast: Tea, 1 pint; bread, 3 oz.; butter, 1½ oz.; and egg, 2 oz. Lunch: Milk, ½ pint and 2 biscuits. Dinner: Bread, 1½ oz.; meat, 4 oz.; potatoes, 3½ oz.; butter, ½ oz.; apples, 4 oz.; and lemonade, 4 oz. Tea Tea, 1 pint; bread, 3 oz.; butter, 1½ oz.; and egg, 2 oz. Supper: ½ pint beef-tea.

Case II was placed on the fixed diet with regulated exercise four days before the analysis of the urine was commenced, and after Christmas similar conditions were observed.

I take this opportunity of thanking Dr. Barrs for his courtesy in placing the two cases at my disposal, and also Dr. Telling for supervising the diet and treatment.

CASE I. *History.*—W. P., aged 54, a coach trimmer, was admitted on November 4th, 1900, into the Leeds Infirmary. His relatives were long-lived, and there was no family history of gout or rheumatism. Apart from gout he had always enjoyed good health. Twenty years previously he had an acute illness associated with severe pain in the great toe. This attack he attributed to sleeping in a damp bed. Subsequently other joints became affected. During the last five years subacute attacks had recurred every three or four months. He had always lived well, eating, as he said, more meat than bread, and drinking a moderately large quantity of beer.

State on Examination.—On admission there were many tophi in both ears, with small gouty deposits in the fingers and toes. The radial tension was markedly increased. On November 11th his temperature rose to 100° F., the left knee being painful and swollen. On the following day his temperature was 101° F. The urine at first contained a moderate amount of proteid, about one-twentieth by measurement, but with the advent of the febrile attack the proportion rose to about one-fourth.

An interesting point was the marked preponderance of serum globulin over serum albumen. As this is considered very unusual it is advisable to detail the method of estimation.

Quantitative Estimation of Serum Globulin.—On the addition of acetic acid in the cold to the filtered urine no precipitate formed. On boiling the filtered urine after the addition of a few drops of acetic acid there was a precipitate amounting to about $\frac{1}{10}$ by measurement. A sample of the urine was half saturated with ammonium sulphate. It was then filtered, and the filtrate boiled after the addition of a few drops of acetic acid; a mere cloudiness resulted which remained unchanged after the lapse of forty-eight hours. Full saturation with magnesium sulphate in neutral solution gave a similar result.

It was decided that the supervention of the febrile attack with marked albuminuria rendered this case unsuitable for the investigation of the action of drugs and another was selected.

The results obtained in Case I are given in Table I on p. 244.

The most notable and characteristic features of these results are the low excretion of uric acid preceding the subacute attack, and the increase after its development. Possibly the uric acid secretion was modified by the exhibition of colchi-sal, but, judging by its effect in the second case, this modification