Varying conditions of climate, diet—of hygiene generally—often affect in a degree so marked as to be obvious the grosser results of differing function of these organs and systems—for example, goitre, asthma. We cannot deny the possibility of a difference according to locality in the degree of frequency of excretion in the urine of a reducing substance in detectable clinical amounts.

This leads to the third consideration: Should such minor test results as those I am discussing be considered negligible and physiological or not? I think they should always be noted, however slight and however fleeting they be, and an explanation sought. It is possible that most, if not all, children from time to time may give detectable sugar reaction on examination of the urine, but even so this forms no foundation for an argument that the detection of such a reaction is of negligible significance.

If once found, examination of numerous specimens should be made—of the twenty-four hours' urine and also of morning and evening specimens. In a large proportion of cases the presence of a positive test corresponds with indiscretion in diet or digestive disturbance, and disappears on correction of such. In other children with indigestion and constipation the reaction is completely negative. This point is in itself important, as possibly affording evidence of individual idiosyncrasy in carbohydrate metabolism.

Another important line of consideration is demonstrated by the findings in the following case.

A girl, aged 6 years, has been under observation in hospital for several months suffering from what I suspected might be progeria. The urine was healthy. She developed a large abscess in the face with considerable sloughing, and the urine showed a distinct positive sugar reaction. The abscess is now healed and the urine free from any reducing agent.

Here was a well-marked case of "sapraemic glycosuria." The precise nature of the reducing substance or substances in the urine during the period of suppuration was not determined, but the fermentation test with yeast was positive and the urine after the abscess healed was free from sugar. I am not now concerned with the nature of "sapraemic glycosuria," but with evidence that slight positive urine reactions should lead to search for a septic focus.

The precise nature of the sugar present in a specimen of urine cannot be determined without expert laboratory assistance; and the examination of blood sugar is, in my experience, valueless except in the hands of a skilled worker in constant practice. The fermentation test with yeast is, however, simple, and in the presence of a positive fermentation test we are faced with the possibility that the reducing agent may be dextrose (glucose or grape sugar), or laevulose (fructose or fruit sugar), or both. (I am not here dealing with nursing infants suffering from gastro-enteritis, in whose urine lactose may be present.)

It has been proved that the presence of laevulose in the urine may be regarded as a sign of liver inadequacy. Laevulose often appears in the urine along with dextrose in marked clinical cases of diabetes. It may, however, appear alone and be derived from cane sugar, honey, vegetables, fruits, etc., in the diet. When it appears it is, as stated above, to be regarded as a sign of liver inadequacy. If, with this in mind, we now turn to the clinical aspect of cases in which we find traces of a sugar present in the urine, we find that in many there is definite evidence of attacks of acidosis; in many there is a history of recurrent attacks of "biliousness"; and in most there is what one may term a full fatty diet. In fact, clinical evidence goes to show that in most there is evidence of strain on liver function, and it seems reasonable to conclude that the small amount of sugar in the urine is probably laevulose. This is further borne out by the satisfactory result of treatment by reduction of fat intake and, if necessary, restriction in amount of other elements in the diet.

But in a considerable proportion of the cases in which only a small amount of sugar is present in the urine, and in the great majority of cases in which the sugar is present in large amount, the sugar is dextrose—that is, the patient has glycosuria. What is the significance of this fact? It is outside the scope of this paper to consider cases either of intermittent or of persistent glycosuria in which large quantities of sugar can be demonstrated in the urine. In all such cases we obtain expert laboratory assistance, determine the level of the blood sugar and the nature of its reaction to a dose of sugar, and also the leak-point for sugar. But whatever methods of research we employ, and however favourable the

results we obtain, our treatment must be that of diabetes mellitus.

It is with cases which show minor results on urinary examination that I wish to deal. In the majority of them there is no marked polyuria, and there may be no excessive thirst or wasting.

The presence of glucose in the urine is, we know, the chief sign of the existence of diabetes mellitus, but we also know that a small detectable amount of glucose in the urine may exist in the presence of sound health. Glycosuria is a term the interpretation of which in any individual case lies somewhere in a scale extending from sound health at the one extremity to diabetes mellitus at the other.

Cambridge, speaking of transitory, intermittent, and persistent glycosuria and diabetes, says: "It is important that it should be clearly recognized that each is but a phase of the same metabolic error, and that no hard-and-fast line can be drawn between them." He further speaks of a "preglycosuric stage." The obvious truth would seem to be: watch for the earliest beginning of abnormality and attempt to check it, as otherwise it may develop into uncontrollable disease.

The markedly outstanding lines along which to attempt to check development are dietetic—a well-balanced diet with the avoidance of constipation and intestinal putrefaction—and aseptic, the removal of all discoverable foci of infection.

My subject deals with small things and is mainly concerned with the beginnings of disease. For fuller development it requires blasts of clinical ozone to play around the figurative laboratory frog.

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BLOOD PRESSURE OBSERVATIONS IN FUNCTIONAL BRUITS IN CHILDREN AND YOUNG ADULTS.*

BY

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DURING convalescence from various mild infections children often go through a period in which they exhibit a sustained rise in blood pressure, the development of a systolic apical bruit presenting the usual functional signs, and accompanied by some cardiac dilatation. This phase of raised blood pressure is sometimes present during convalescence even from acute rheumatism, and its possibility must always be borne in mind when judging of the nature of a systolic cardiac bruit in children after pyrexia.

The child is far more unstable in its circulatory equilibrium than an adult, and the heart muscle just recovering from the damage of pyrexia is in no case able to sustain the increased work that a slight hyperpiesis involves. A rise of 10 or 20 mm. Hg, which is often to be observed, makes a far deeper impression than in an adult and can be the cause of the cardiac condition. My contention is proved by the fact that as soon as the blood pressure falls the cardiac sounds become normal, in the absence of an organic endocarditis.

The normal blood pressure in childhood has been very little studied in this country. I find a record of 3,000 repeated observations by Judson and Nicholson¹ in America. The standard of systolic pressure they lay down is as follows:

From 3 to 6 years	...	...	...	90-92 mm. Hg
" 7 to 9 "	...	...	...	up to 94 "
" 10 to 11 "	...	...	...	" 99 "
" 12 to 13 "	...	...	...	" 103 "
" 14 to 15 "	...	...	...	" 106 "

I have found that children are very susceptible to the action of the nitrites, and I have employed them in the following combination for reducing blood pressure in my cases: Sodium nitrite, liquor trinitrini, and spt. aetheris nitrosi in suitable dosage. In adults these are of no use. Appended are some notes of typical cases.

W. E., female, aged 9, had a mild faucial attack of diphtheria in February, 1916. Increasing tachycardia was observed as convalescence advanced, and she was kept in bed; ten days later, in spite of cardiac tonics, the heart had dilated beyond the nipple line, and a systolic bruit, not conducted, and heard in all postures,

* Being abstracts from a paper read before the Bradford Medical-Chirurgical Society, February, 1921.

had developed. Blood pressure 110. There was no albumin in the urine. Nitrites were given. Seven days later blood pressure was 95; the bruit and dilatation had gone. Six days later blood pressure was 96, and there was no bruit. The improvement in the heart did not occur until the blood pressure was lowered.

W. B., male, aged 5, had scarlet fever in June, 1919. There was cardiac dilatation on the eleventh day, with suspicion of an apical systolic bruit. On the twelfth day blood pressure was 100, and on the thirteenth 105. Nitrites were given; on the fifteenth day blood pressure 75, sounds clear, apex beat in the nipple line. On the twenty-fifth day blood pressure was 90, sounds clear; there was no albumin in the urine throughout. He was a highly strung child of a very neurotic mother.

S. D., male, aged 12, had fibrositis with pyrexia in February, 1916. He was the only child of neurotic parents. He had a sudden attack of severe pain in the back, with tenderness to the left of the spine in the lower dorsal region. His temperature was 100°, but was quickly controlled by salicylates. After seven days the heart was rapid and dilated considerably; the sounds were not clear, but he had no bruit. Seven days later there was no improvement, though he was kept in bed; a systolic apical bruit had developed; blood pressure 140. He was given the nitrite mixture, and after three days blood pressure was 120; bruit doubtful. For the next two days nitrites were not taken because of indigestion, and the boy was allowed up without my orders. I found the blood pressure again at 140, with dilatation and bruit more marked. After another three days' treatment the blood pressure was 115, with bruit much fainter. Ultimately he became quite well.

H. B., male, aged 7, had acute chorea in December, 1920. On the 26th the movements were very violent. Temperature 101°; blood pressure 103; there was a systolic bruit at the apex. The bruit and increased blood pressure persisted after the acute phase had passed. On February 1st he was able to walk, and I thought it safe to give nitrites. Blood pressure on February 1st was 105. The bruit was heard both standing and sitting, but was not transmitted to the back; it was intensified on lying down. On February 7th, after nitrites had been given for one week, blood pressure was 95; no bruit was heard in any position.

My conclusions, as a result of this experience, are as follows:

1. To take regular blood pressure observations in all cases of "impure" heart sounds after fever.
2. A rise in blood pressure, usually after, but sometimes during, the acute stage of an infection in children shows that a want of control of the arterial system is developing, and explains a functional bruit when it occurs.
3. If the heart condition in children is relieved by lowering the blood pressure, rest in bed can be dispensed with after sufficient rest has been taken to ensure that the heart muscle has repaired the temporary damage of the pyrexia.

To explain these cases is difficult. Three factors at least must be considered:

1. The patient's temperament. It is particularly the "nervy" children who suffer this disability. The neurotic element is a large one in modern etiology. Cannon² has proved experimentally that the suprarenal secretion is much increased in emotional stresses, such as fear and pain.
2. The action of the toxin upon—
 - (a) The arteries. Toxins are universally credited with a depressor action on the musculature of heart and arteries. I do not wish to question this. My cases are a fairly common exception to the rule that the blood pressure is lowered during an infection. It must not be forgotten, as pointed out by Albutt, that the same causes may operate to produce hyperpiesis in childhood as in adult life. These may be more active after a pyrexia. But this does not disprove my main contention, that the heart condition improves *pari passu* with lowering the blood pressure.
 - (b) The endocrine system. It is not unreasonable to assert that its delicate balance can be temporarily at fault during the toxæmia of childhood's illnesses, and that transient degenerative changes are present in these glands during a fever. It is quite possible, too, that the toxin may exhaust the sympathetic nervous system which controls them. The frequent presence of a compensated acidosis in these cases will, I hazard, be explained in the same way.

The condition thus described is on all-fours with the common "D.A.H." of the soldier. The very name is a glaring example of hasty and slipshod thinking, focusing the attention on the heart instead of on the circulation as a whole. Derangement of blood-pressure control is the correct diagnosis; the heart is only involved in so far as it is over-worked to maintain an efficient circulation. Earlier symptoms, suggesting hyperthyroidism, were common in these cases. In 1921 this phase has passed, and a raised blood pressure is often the only genuine sign of derangement that can be found, suggesting hyperadrenalism. The soldier was taken from sedentary life and drilled to dropping point for some months; he lived for some years on poisonous "dixie" tea and eternal stews, smoked endless cigarettes, suffered fear, lack of sleep, and usually several distinct pyrexias of known or unknown origin. It is no wonder that blood-pressure control broke down, and that when the vicious circle has been

confirmed it takes years to undo by reorganizing the endocrine system and building up the sympathetic nervous system which has become exhausted.

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A CLINICAL TEST FOR THE PRESENCE OF CELLULAR ELEMENTS IN THE URINE.

BY

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THE object of publishing this note is to put into the hands of the medical practitioner or the nurse a simple means of ascertaining the presence or absence of pus, blood, or bacteria in the urine. The principle of the test is not new, but I have not seen it in standard works on clinical pathology, and from extended observations it seems to me that it should have a useful place in clinical work. This method will frequently reveal pathological conditions in the urinary system where none had been suspected, since it has a wider scope and a much greater delicacy than the clinical methods ordinarily in use in general practice, where a centrifuge and microscope are often not available. If the presence of cellular elements in the urine is shown by this test a further specimen must then be submitted to microscopical examination to ascertain their nature. If the test is negative it can, with a few rare exceptions, be stated that in this respect no further examination is required.

Cellular elements are not normally present in a freshly passed urine, if we except an occasional epithelial cell. If cells are present they may be either bacteria, or cells derived from the body. These latter may be casts from the kidney tubules, or epithelium derived from the urinary tract; or they may be either red or white blood cells, the latter almost invariably polymorphonuclear leucocytes.

Nature of the Reaction.—The test is made to ascertain the presence of peroxide splitting enzymes in the urine. These enzymes have the power of setting free oxygen from hydrogen peroxide, and the reaction in a fluid medium gives rise to the ebullition of gas bubbles. In addition to enzymes certain other bodies have the power of setting free the oxygen, but for our present purpose haemoglobin is the only one that need concern us. If hydrogen peroxide is added to normal sterile urine no effervescence occurs even after a considerable time has elapsed. It follows that if the addition of the peroxide leads to effervescence we may take it that some abnormal element is present in the urine.

Source of the Enzymes.—These enzymes are present in pus cells or leucocytes in large amount, and these, therefore, give a very free reaction. The enzymes are said not to be present in lymphocytes, but in any case these cells are rarely present, alone at any rate, in urine. Epithelial squames give little reaction, unless heavily infected with bacteria, but the deeper epithelial cells react more freely. Tube casts may react vigorously. Red blood cells give the reaction on account of their haemoglobin. Of the albuminous fluids fresh blood serum gives an active reaction, probably on account of some blood cells remaining in suspension in it, since other albuminous fluids such as hydrocele and ascitic are inert. The slight reaction usually obtained from an albumin-containing urine is probably due to the presence of casts and a few blood and pus cells such as are usually present in such conditions.

Bacteria.—In an investigation of the various bacteria isolated in the course of routine laboratory work for a considerable period I found that practically all of the bacteria, with the exception of the streptococcal group (which includes the pneumococcus), gave active ebullition with hydrogen peroxide when the cultures were treated directly with it. At the same time it was noted, when bacteria were grown on sugar-containing media (which they fermented with the production of acid), that they were liable to give a negative reaction. This point has to be considered in dealing with very acid urines, since the peroxide-splitting enzymes only act well in neutral or alkaline media.

Minimal Amounts Giving the Test.—These were ascertained experimentally, using normal urine as a basis and adding the foreign substances in appropriate quantities. The