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.

REFERENCES.

- ¹ Nicholson's *Blood Pressure*, chap. ix. ² Cannon: *Bodily Changes in Hunger, Fear, Rage, and Pain*, 1920. ³ Sir Clifford Allbutt: *Dictionary of Treatment*, Hutchinson and Sherren.

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

BY

T. H. C. BENIANS, F.R.C.S.,

PATHOLOGIST TO THE PRINCE OF WALES'S GENERAL HOSPITAL,
TOTTENHAM, LONDON.

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

sensitivity of the peroxide test in the case of blood and pus was compared with the guaiacum reaction. It was found that the peroxide test was positive with about one-twentieth the amount of pus that would just give the guaiacum reaction (using ordinary tincture of guaiacum). The guaiacum test was positive only where there was sufficient pus in the urine to cause a definite turbidity, but the peroxide test gave a positive reaction where no trace of foreign matter could be seen.

With blood the peroxide test was positive with about 300,000 red corpuscles per cubic centimetre of urine, the guaiacum and ozonic ether failing at anything below 1,250,000. To the naked eye the former amount could not be seen in the urine. In the case of bacteria the presence of the staphylococcus was demonstrable in as small numbers as 200 million per cubic centimetre, which number was not obvious to the naked eye except by very careful comparison with a control.

Similar results were obtained with *B. coli* and *B. paratyphosus* B.

Technique of the Test.—If the urine is strongly acid or alkaline to litmus it should first be neutralized by the addition of a few drops of dilute alkali or acid. The peroxide should be fairly fresh, not less than 20 volumes in strength—40 volumes is better—and not too acid in reaction. I have obtained by far the best results with the neutral 100 per cent. fluid issued by Merck's under the name of "perhydrol," as with this concentrated solution only a few drops are needed for the test.

Take about a third of a test tube of urine and add about a quarter of the amount of the 20 volumes peroxide solution to it. Stand the tube in a rack, not in direct sunlight, or spontaneous evolution will take place. In a strongly positive reaction bubbles start to rise immediately and form a permanent layer of foam at the top of the fluid. If the reaction is only faintly positive bubbles will gradually rise and form a thin layer on the top; this may take from fifteen to twenty minutes or longer if the urine is acid. A few bubbles lying on the meniscus are made more evident by gently rocking the tube to throw them down into the fluid.

All degrees of effervescence may occur between the two described. In a normal urine one or two bubbles may rise immediately after the addition of the peroxide, especially if the urine is warm, but there is no steady evolution of gas or formation of a layer of foam.

Sources of Fallacy in the Test.—(1) Accidental contamination of the specimen with bacteria is a frequent source of error since the ordinary specimen glass is as a rule grossly contaminated with bacteria. Moreover, in non-catheter specimens, if several hours old, there is certain to be a good deal of bacterial growth which would give a positive reaction. It follows that the specimen must be collected in a quite clean vessel, and unless a carefully taken catheter specimen is available the test should be carried out shortly after the urine is passed. In the case of females a catheter specimen though not imperative is advisable on account of the cellular and bacterial debris that may be present. (2) The ordinary peroxide solutions are likely to be definitely acid, and this may lead to error in several ways. If an unnecessarily large amount of the reagent is added the action of the enzymes will be inhibited. More than this, if carbonates are present, the acid will act on them and lead to effervescence and so to a faulty diagnosis.

Summary.

Peroxide splitting enzymes are derived from most cellular elements. Hydrogen peroxide is also decomposed by haemoglobin from red blood cells. Cellular elements are not present in normal urine; in pathological urines those most often present are red blood cells, pus cells, and bacteria. In nearly all cases the presence of these bodies or their derivatives can be shown by this test, even when present in amount very much smaller than can be detected by the clinical tests commonly in use. The test, however, does not differentiate between one form of cellular element and another; it shows with a very small percentage of error whether abnormal bodies are or are not present, and whether or not further investigation is needed.

The reaction is negative with the various crystalline deposits (with the already noted exception of carbonates).

NOVEMBER 23RD was set apart by the municipal authorities of Berlin for the extermination of rats. The owners of property in the city were ordered under a penalty to distribute on that day in appropriate places upon their property effective rat poison to the value of fifty marks.

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

FRACTURE OF THE END PHALANX OF A FINGER, WITH RUPTURE OF THE COMMON EXTENSOR TENDON.

(With Special Plate.)

THERE is a device, known to many, for opening a clenched fist. It consists in forcibly approximating the tip of a finger to the first interphalangeal joint (by flexion). I have had a case recently of this kind. The *fons et origo malorum* was a coin in the closed hand of one person which another individual was desirous of releasing.

The aggressor in the struggle began by forcing the tip of the little finger towards the first interphalangeal joint (flexion). The result of this procedure was a swelling in the distal interphalangeal joint, which did not subside in a reasonable time. I accordingly made a radiogram of the joint. This showed that a portion of the bony substance of the dorsal proximal part of the distal phalanx was detached, and the result was incapacity to extend fully the end phalanx.

Treatment, which consisted of hyperextension for a few weeks, was quite successful, power to extend the phalanx fully being completely restored. The illustrations are radiograms taken before and after treatment. (See Special Plate.)

I have found an injury due to similar kinds of violence described under the name of "drop finger" or "mallet finger," but I think the interest in this case consists in the detachment of bone, which is shown in the radiogram, and which I am unable to find previously described.

I have to thank Mr. Hedley Marsh for helpful criticism.

Macclesfield.

JOHN N. LAIRD, M.D.

TORN SPLENIC ARTERY—SPLENECTOMY.

T. G. was admitted into the North Lonsdale Hospital in a state of collapse, as a result of an accident, on April 26th, 1921. He had fallen into the hold of a ship, first 30 feet on to a plank, then bouncing off the plank and falling another 10 feet.

On examination there was obviously acute abdominal trouble, with dullness in the left flank. I at once opened the abdomen and found blood in the peritoneal cavity, the splenic ligaments were torn away with the splenic artery, the artery was torn at the exact junction with the spleen as if shaved off, but the spleen itself was practically undamaged.

I removed the spleen, ligatured the splenic artery, closed the abdominal wound, and the patient left the hospital on June 20th.

The accident was remarkable inasmuch as there was no other bodily injury at all. I attribute the peculiar separation of the artery to the sudden break in the fall when at 30 feet he bounced off the plank and thus snapped the artery.

On November 23th I had a blood film examined at the Yorkshire Pathological Society, the report of which was that the red corpuscles showed no abnormality, and were almost uniform in size and shape, and that the leucocytes seemed to bear the normal proportion to the red corpuscles. On the same date the patient seemed to be in very good health, normal weight 9 st. 4 lb., height 5 ft. 4 in., and apparently not missing his spleen in any way.

JOHN ARTHUR REED, O.B.E., M.B., Ch.B.,
Honorary Surgeon, North Lonsdale Hospital,
Barrow-in-Furness.

AN OPERATION FOR THE RELIEF OF ASCITES, FOLLOWING CIRRHOSIS OF LIVER.

A MALE Hindu, aged 35, about twenty-five years ago had an attack of malaria lasting nearly one year. About ten months ago he suffered again from malaria, and since that time he noticed gradual swelling of the hypogastric region. This swelling increased, gradually extending to the whole abdomen. He had severe attacks of epistaxis; soon after his legs also commenced to swell. The patient is an inhabitant of a village about sixty miles from Kurduwadi, Deccan, India, the condition of which is insanitary.

When he consulted me he complained of pain after food, he had dyspnoea while walking, and suffered from slight insomnia. On examination the chest was emphysematous