matter for an organ which has to work the whole twenty-four hours, and I have always agreed with the late Sir W. H. Broadbent that we do more good by diminishing the heart's work than by driving it on with tonics.

Consider the condition of the heart, placed in a practically unyielding investment (the pericardium); every dilatation means pressure, and pressure means at least defective circulation in the cardiac veins and capillaries, to say nothing of the influence of the defective action of the left ventricle on the cardiac arterial supply. Thus every dilatation of the right side, if it increases intrapericardial pressure, must mean defective circulation in every muscle fibre of the heart. This risk is removed by lessening fluids, and in morbus cordis this also diminishes the strain on the valves, and so the tendency to recurrent endocarditis.

On the heart the effect is not only to diminish work and aid its mechanical efficiency but also to improve its nutrition enormously. The pressure in the whole arterial system is diminished, and the importance of this in advanced cases of arterial disease or impending cerebral haemorrhage needs no demonstration. The capillaries and venules are no longer over-distended and liable to rupture. The veins previously visible and prominent, especially in the lower limbs, shrink and disappear, and the very texture of the tissues becomes firmer and harder. This was well seen in the case of morbus cordis above mentioned, where fluid was taken chiefly in fruit. In place of the flabby limbs of a beer-drinking butcher this patient developed the iron-wire muscles of our nearest frugivorous relatives. The frugivora, I believe, take little fluid, and that mostly in the form of fruit.

Such changes as I describe profoundly affect the whole vascular system. Several times lately I have been called to see cases of aneurysm in which, I know not why, no attention had been paid to the quantity of fluid. One of these patients was so bad with dyspnoea that I scarcely dared to move him for examination. I found that he was taking 2½ pints of fluid a day. Four weeks later, as the result of reduced fluids, he was walking about, and only sat down for examination. The aneurysm was not cured, but life, which was threatened by the distension of high blood pressure, was at least prolonged.

In the dilated and splashing stomach commonly found with chronic collaemia there is often in addition an increase of heart area, and a capillary reflux of 7 to 8 with a blood pressure of 130 or less, and a quick, often irregular pulse, with palpitation on exertion. A capillary reflux of 7 with a strong heart should show a blood pressure of 140, and one of 8 a blood pressure of 160; thus we can measure the extent of the heart failure. These conditions are commonly seen together, and collaemia is the central factor. When the heart is already overweighted by high blood pressure it is most injurious to give excess of fluids in the false hope of washing out excess of uric acid. Excess of fluid is slowly absorbed from the stomach; while there it dilutes the digestive juices and favours fermentation; when absorbed it dilutes the digestive secretions, diminishing their power, and favouring the increase of microbic putrefaction, malnutrition, and debility. Its tendency is to dilate the stomach the muscles of which are, like those of the heart, in poor condition from defective circulation. As a result of such malnutrition and debility excess of fluid will eventually produce some oedema of the ankles, which is not infrequently accompanied by oedema of the cortical membranes, which lies at the root of brain-fag and debility, commonly called "nerves"; of bad temper, nervous excitement, hysteria, or of neurasthenia, depression, and lethargy up to the more fully-developed signs of uraemia—for example, twitching of muscles, headache and coma. Many of these conditions are often promptly relieved and may be cured by lessening fluids, strengthening the heart, and later on removing collaemia by a purin-free diet. I believe that no small part of the increase of such "nerve" troubles is due to increased consumption of fluids, especially where the increase of fluid is not accompanied by any measure to diminish the introduction of uric acid. As a rule collaemic sufferers dislike fluids. Their blood contains excess of fluid, and shows only small percentages of haemoglobin and cells, since the capillaries of all the organs of excretion (kidneys, skin, lungs) are blocked by collaemia. These patients are

easily persuaded to take less fluid, and generally have to be urged to take excess.

Albuminuria and Bright's disease are an additional indication for this treatment, for at least a part of the defective combustion, called Bright's disease, is due to cardiac weakness, and anything improving the condition of the central organ of the circulation improves the general combustion.

The treatment of collaemia with heart failure and its results is to cut down fluids but not to reduce the income of proteids—for no heart tonics will act if food is deficient; to rest the whole body as much as possible so as to lessen the work of the heart; to give heart tonics such as digitalis or nuxvomica in small doses long continued, till the pulse-rate is normal and the natural ratio between capillary reflux and blood pressure re-established. I used to be puzzled by cases of neurasthenia and depression in which there were obvious indications for heart tonics, and yet the administration of such tonics made the mental condition worse. In these cases there is always high blood pressure, and to raise the pressure in the skull still more by tonics will do harm so long as there is cerebral oedema; but remove the oedema by diminishing fluids, and the tonics can then be given with benefit. The same applies to the heart—take off the work and improve its mechanical power by diminishing fluids, and the tonics will do treble the good in half the time.

What harm may be done by diminishing fluids? Uric acid gravel, or the presence of large quantities of urate in the ears, joints, and fibrous tissues, forbid diminution of fluid over a long period, for the excretion of such urate store is almost certain to give trouble in the tubules and pelvis of the kidneys if sufficient fluid is not taken. Yet when a patient is obviously dying of heart failure on the one hand, or cerebral oedema on the other, we must immediately cut down fluids and consider the excess of urates afterwards. There might be increased tendency to thrombosis, but, as the ultimate effect is a general improvement of the circulation, this does not seem important, especially if a purin-free diet is adopted.

Constipation is another possible trouble, and sufferers on an ordinary diet will not be better on diminished fluids. But what is the inconvenience of a little constipation if weighed in the balance against heart disease, or uraemia, or cerebral oedema? The use of common laxatives will counteract it while matters are critical, and later there will not be much trouble from constipation if fruit is used daily.

In serious conditions I take thirst as a guide, and I find that most patients can reduce their fluids to 30 oz. a day, and, with the help of fruit, to 25 oz., 20 oz., or less, at least for a time. If salts, salines, and diuretics are given more fluid will be called for, and then we have to decide whether most good is done by withholding drugs and water, or by giving both. As a rule, I am in favour of the former course, though drugs can be used at first, as it takes several days to reduce the fluids of the body. I order that 30 oz. be aimed at, but that fruit or extra water shall be given whenever thirst imperatively demands it.

REFERENCE.

¹ *Canada Lancet*, August, 1903.

THE OCCURRENCE OF TUBERCLE BACILLI IN THE BLOOD IN TUBERCULOSIS.

BY

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It appears to have been little, if at all, recognized that tubercle bacilli may commonly be found in the blood of tuberculous patients.

Very recently Rosenberger¹ published a remarkable series of cases which must bring the question of blood infection in this disease into prominent notice. The cases he examined included many different forms of tuberculosis, and he was able to demonstrate the bacillus in the blood of every patient, whatever the type of the disease, and however early the disease appeared to be clinically. It is

clearly of great importance that his conclusion as to the occurrence of blood infection in every case of tuberculosis should be tested. To mention one matter—if it be completely proved that living tubercle bacilli occur in the blood of practically every tuberculous patient, no matter how early or circumscribed the lesion appears to be, it is without doubt that our ideas as to the use of vaccine-therapy—tuberculin—must be considerably modified.

Using to some extent his methods, I have examined the blood in 12 cases of pulmonary tuberculosis. The method of examination is simple, and is, shortly, as follows:

Method.

The skin of the front of the forearm is thoroughly cleansed with liquid soap, alcohol, and ether. A tourniquet is applied, and, with a sterile syringe fitted with a platino-iridium needle previously flamed, about 5 c.cm. of blood are drawn from any convenient vein. The blood is at once mixed with about an equal volume of sterile citrated salt solution. The mixture is placed in a swift-running centrifuge for twenty minutes, and some of the deposit taken up on a slide to form a thick film. This is dried in an oven at 60° C., and is then placed in sterile distilled water to lake the blood. Thereafter the film is fixed and stained in the usual way.

It is important to state that every precaution is taken against risk of contamination. A new pipette and new slides, kept in strong sulphuric acid, are used in each case. The centrifuge tubes and flasks are cleansed with caustic soda, pure sulphuric acid, and sterile distilled water, and similar care is taken with the syringe.

Out of the 12 cases, 10 had distinct physical signs and had tubercle bacilli in the sputum. Adopting Turban's classification, there were of Stage I 3 cases, of Stage II 6 cases, and of Stage III 1 case. This gives a total of 10, and all these 10 showed tubercle bacilli free in the blood.

The organisms were of the long or short forms, beaded or uniformly stained, and were acid-fast rods morphologically indistinguishable from the tubercle bacillus. There seems little possibility of doubting their identity with it; seen in the sputum they would be judged absolutely diagnostic.

In each case they were few in number; no groups were seen; usually the bacillus was alone in the field, or at most two appeared together. They were found with more or less difficulty. Often only one was seen after long and laborious search over a whole slide, and in a few cases two and three and four slides had to be examined before success. In one case phagocytosis was noticed, and only in one was there evidence of mixed infection in the blood. In this a diplococcus was seen in fair numbers, and it is being further investigated.

The result of cultures from the blood of these cases and the result of the inoculation of the infected blood into animals remain to be worked out.

One of the two cases in which no amount of search could show the bacillus was a patient with very indefinite symptoms and physical signs, and altogether of doubtful diagnosis. In the other case the physical signs were marked, but repeated examination had never revealed tubercle bacilli in the sputum.

Only 4 of the 10 cases giving a positive result had any fever at the time of examination of the blood. In the other 6 the temperature was normal, and had been so for many days or weeks, all of them getting about well, and showing, in short, the usual chronic course of the disease. I state this to draw attention to the fact that they were by no means rapidly progressing acute cases, or cases of general tuberculosis.

I have to express my thanks to Dr. Henry Clarke, Honorary Superintendent of the Research Laboratory, for kind advice and help, and to the other physicians of the Liverpool Hospital for Consumption and Diseases of the Chest, for the facilities they have given me in this matter.

REFERENCE.

¹ Rosenberger, *American Journal of Medical Sciences*, February, 1909.

THE fifth International Congress of Dental Surgery will be held at Berlin from August 23rd to 28th. The work of the Congress will be distributed among twelve sections. The General Secretary is Dr. Schaeffer-Stuckert, of Frankfurt-on-Main (Kettenhofweg, 29), to whom all communications relative to the congress should be addressed. In connexion with the congress there will be an exhibition.

CHRONIC NEPHRITIS TERMINATING WITH HAEMORRHAGE FROM THE BOWEL AND PERFORATION OF THE BOWEL.

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THE complications of chronic nephritis are numerous and for the most part well recognized, but the following case presents some unusual features, and calls attention to a dangerous complication about which very little appears to have been written:

A married woman, aged 27, was admitted into Queen's Hospital on account of profuse haemorrhage from the bowel.

She looked very pale, ill, and thin, and said she had not been really well for three years; and that since the birth of a stillborn child about a year before, she had suffered from vague pains in the epigastrium and in the lower part of the abdomen. On examination of the abdomen there was neither pain, tenderness, nor rigidity. The temperature was normal and remained within normal limits till the day of her death; the heart was hypertrophied and the second aortic sound accentuated; the pulse 112 and of high tension. The urine showed a cloud of albumen and one or two granular casts.

For the two days following admission the motions were fluid and dark with blood; after that they became light in colour, but on several occasions small dark red clots were found. The patient remained quite free from pain, the abdomen was soft and flat and nowhere tender. Feeding was limited to nutrient enemata. Bacteriological examination of the faeces revealed nothing unusual.

Before admission she had been treated for gastric ulcer, and after admission the diagnosis of gastric or duodenal ulcer was considered, and with it the advisability of operation; but the site of the haemorrhage was uncertain and the patient seemed to be improving, so it was decided to do nothing till she had been a little longer under observation.

On the seventh day after admission she became suddenly collapsed and complained of severe abdominal pain; she rapidly developed the signs of a perforative peritonitis and died on the following day.

Necropsy.

The peritoneal cavity contained a quantity of thin, blackish, offensive fluid, which had obviously escaped from the small intestine through three perforations, which could be seen in the lower end of the ileum. The lower 6 or 7 ft. of the small intestine and the caecum were blackened with numerous haemorrhages into the wall of the gut, and on opening the bowel I found more than 100 small ulcers in the lower portion of the small intestine. The ulcers were all small, the largest being about $\frac{1}{4}$ in. in diameter, and round or oval; each ulcer seemed to have originated in a patch of haemorrhage, though there were many haemorrhages which had not ulcerated; they varied in depth, many had reached the peritoneum, and three had perforated, while others were quite shallow and involved no more than the mucous membrane. The edges of the ulcers were not raised; Peyer's patches were not prominent. The remainder of the alimentary canal appeared to be healthy.

A careful examination of the mesenteric vessels was made and a microscopical examination of the wall of the intestine, but it threw no light on the immediate cause of the haemorrhages, though it seemed clear that the ulcers were due to invasion of the haemorrhages by micro-organisms from the bowel.

The kidneys were pale, granular, and contracted, and microscopically showed all the changes of a chronic interstitial nephritis and a great degree of endarteritis of the smaller vessels.

The heart was large and showed marked hypertrophy of the left ventricle.

Quite recently a similar case came under my notice:

A man aged 28, a lead worker, was admitted into the Queen's Hospital suffering from chronic nephritis and arterio-sclerosis, and died suddenly from a cerebral haemorrhage. The autopsy revealed a marked hypertrophy of the heart and degenerated arteries, a large cerebral haemorrhage, and an advanced interstitial nephritis, but in addition the lower 6 ft. of small intestine was blackened with numerous small haemorrhages into its wall, exactly as in the previous case; and on opening the bowel I found superficial ulcers forming in many of the haemorrhagic spots; but in this case there had been no haemorrhage into the lumen of the bowel, I take it because the ulceration was not sufficiently advanced.

Thus two very similar cases, differing only in degree, have been seen within a few months of each other in the post-mortem room of a comparatively small hospital, so it is not likely that the condition is one of very great rarity, though it must be one which it is important to recognize.

Whether these two cases must be regarded as cases of haemorrhage due to increased blood pressure, or whether we must look for some change in the mesenteric vessels, or for some toxin in the part of the bowel affected, I am