its specific gravity observed. The amounts of the following constituents were determined :—

Constituents.	Method.
1. Total nitrogen	Kjeldahl.
2. Total urea	Dupus.
3. Total uric acid	Hopkins.
4. Total chlorides	Moore.*

* By titration with AgNO₃, potassium chromate being used as an indicator.

The analyses were carried out over a period of three weeks. The curves obtained showed the following characteristics. The volume of urine fell to a low value on the fourth day after the operation and subsequently showed a gradual rise. The specific gravity was low to start with but rose towards the end of the period. (Fig. 1.) The total nitrogen showed little variation from the normal, the average daily excretion during the first week being 14 grammes, during the second week 13 grammes, and during the first five days of the third week 12 grammes. (Fig. 2.) The average urea excretion during the same periods corresponded to these figures. (Fig. 3.)

The uric acid was somewhat higher during the second week (Fig. 4) than during the first, but otherwise there was nothing particular to note about it. Until the fourth day after operation there were no chlorides present in the urine, but from this day they gradually (Fig. 5) rose to a normal value. This phenomenon may be explained as the result of excessive hæmorrhage before the operation in consequence of which the chlorides were retained. The urine was examined for the presence of sugar but no reducing substance was ever obtained. Albumin was detected in traces from the beginning of the observations until March 19th. With regard to diet, the patient ate on March 11th a poached egg, some fish and minced chicken, bread and butter, also beef-tea, milk, and barley-water. From March 18th beef or mutton was generally included in the diet.

These figures may be compared with those obtained by Foshay¹ who made similar estimations in two cases of nephrectomy. In the first case the operation was performed for calculus and floating kidney; in the second for sinus following nephrotomy. His results resemble ours in that the urea excretion shows little variation from the normal directly after the operation; he also found that the volume of urine was less than normal during the first few days following the operation and this is in accordance with our observations. The fact that in none of these cases did the urea excretion fall is of interest as illustrating the power of one kidney to do the work of two. There is, however, rather an important point of difference between Foshay's cases and the one recorded above; in the former the condition of the patients was pathological, in the latter practically physiological. This pathological condition must have especially affected the remaining kidney, for, as Foshay points out, the healthy kidney had been doing extra work for some months previously, and hence it is easy to understand why the removal of the diseased one caused no fall in urea excretion; in our case the excretion had been carried on by both kidneys alike, and yet the remaining kidney was found to be able to do the work of the two kidneys before any hypertrophy could have taken place.

SHELL-FISH TYPHOID FEVER.

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THERE were three well-marked outbreaks of shell-fish typhoid fever in Norwich during the year just concluded. These notes and charts relate to the two earlier outbreaks; the third and last outbreak attributed to the eating of mussels from Wells has already received notice in THE LANCET.² The first and most limited outbreak began in March and ended in April and was due chiefly, if not entirely, to mussels sent into the city from Breydon. Anyway, as the outcome of a warning issued to the public and of the seizure and destruction of the mussels, the

notifications fell to zero. The epidemiological evidence was to me so clear and sufficing that I did not trouble to have any bacteriological examination made. Graphically, the story of this outbreak is represented by Chart 1.

The dots signify weeks ending on Saturdays and the figures below the lines the number of notifications. "A" represents date of issue of warning; "B" date of seizure and destruction of mussels.

The second outbreak began in May and lasted until July. It proved to be a very much more troublesome affliction than the preceding eruption. Its cause was more obscure than its course. From clinical and epidemiological evidence I felt sure that we were dealing with a shell-fish typhoid fever and tried to obtain sanction for prohibiting the importation of shell-fish but was informed that there was no legal warranty for such a course. However, with an infinity of trouble I traced more than a dozen cases to cockles obtained from two shops. These shops happened to be kept by owners who were wholesale dealers in shell-fish as well as retailers of them, and these two dealers were, I found, the only importers of cockles from Ipswich, the source of supply being presumably the Orwell. Only a portion of their consignments from Ipswich were sold over their own counters, the remainder being sold to many and various retailers of shell-fish, who mixed these Ipswich cockles with others from many and various sources. I re-issued a warning to the public and advised that cockles be not eaten, and feeling satisfied as to, at any rate, the main source of the mischief directed that cockles coming from Ipswich should be seized on arrival at the station and destroyed. This was done. From one of the bags a sample was taken and examined bacteriologically, and I got thus good confirmatory evidence, this special sample being said to be "grossly polluted with sewage." The effect of this action—seizure and destruction on epidemiological evidence and without waiting for bacteriological examination—though I believe extra-legal, again can be represented most clearly by the subjoined Chart 2.

I have evidence that about a fortnight prior to the marked rise in the number of notifications more cockles than usual were consigned from Ipswich to the Norwich dealers. These latter were informed at the time of the first seizure that consignments of Ipswich cockles would be seized and destroyed. The local newspapers helped very handsomely in spreading abroad knowledge of the risks run in consuming cockles.

The inference I draw from the unfortunate experience of this city is that there exists an imperative claim to have local sanitary authorities armed with powers to prohibit the

¹ Inter. Med. M., Philadelphia, 1893, vol. ii.
² THE LANCET, Sept. 26th, 1908, p. 961.

importation into, or sale within, their districts of any shell-fish the safety of which there is reasonable ground to suspect, and to make it the duty of any authority which permits the exportation of shell-fish from its district to show

CHART 2.

Lines and dots have same signification as before. "A" represents date of re-issue of warning; "B" of seizure and destruction of cockles.

that every reasonable care has been taken to keep such shell-fish as are to be found within its jurisdiction free from pollution.
Norwich.

SOME COMPARATIVE MEASUREMENTS OF THE LIVES OF LEUCOCYTES¹ WHEN THE CELLS ARE RESTING IN THE PLASMATA OF DIFFERENT PERSONS, AND THE POSSIBLE APPLICATION OF SUCH MEASUREMENTS AS AN AID TO DIAGNOSIS IN INFECTIVE DISEASE. By HUGH C. ROSS, L.R.C.P. LOND., M.R.C.S. ENG., LATE SURGEON, ROYAL NAVY; PATHOLOGIST TO THE ROYAL SOUTHERN HOSPITAL, LIVERPOOL.

OF recent years I have been endeavouring to ascertain the effect produced by one person's plasma on the life of another person's leucocytes. It appeared reasonable to suppose that the plasma of a person suffering from an infective disease would be poisonous to the leucocytes of healthy persons. If this is the case it might also be reasonable to suppose that the same plasma would not be so poisonous to the leucocytes of another person suffering from the same disease, because it is probable that the cells would be already used to, or immune against, the toxin, and furthermore that if the toxin of one infective disease differs from the toxin of another infective disease, it might be inferred that an immunity on the part of a leucocyte against one disease will not render it immune against another. Therefore, provided it is possible to tell accurately when a leucocyte is dead—that is, if one can differentiate

a living from a dead cell—it also will become possible to measure the lengths of the lives of leucocytes after they have been removed from the body. And this will enable us to make comparative measurements of the lives of leucocytes when they are mixed with the plasmata of different persons. Supposing, therefore, it is true that an infected plasma shortens the lives of a healthy person's leucocytes but does not shorten the lives of the leucocytes of another person suffering from the same disease, it may be useful to reverse the process and assist in the diagnosis of infective disease by making measurements of the lives of such a patient's leucocytes when they are mixed with different plasmata. For instance, if the leucocytes of a person suffering from an indefinite infective disease are found to be easily killed by the plasmata of persons suffering from a variety of diseases but are not comparatively easily killed by the plasma of a person suffering from, say, typhoid fever, it might be inferred that the patient is suffering from, or has recently suffered from, typhoid fever, because his leucocytes are used to, or immune against, that disease.

The above is the enunciation of a problem which I set myself to solve several years ago, and this paper describes the experiments which have been conducted to investigate the last part of it—i.e., with the object of determining the actual measurements of the lives of leucocytes when they are placed in the plasmata of people who are suffering from various diseases. The earlier researches made in order to differentiate living from dead leucocytes have already been published in the *Journal of Physiology* (1)*, and the actual method employed to estimate how many living and how many dead cells there may be in a given volume of citrated blood has been described in THE LANCET of Jan. 16th, 1909 (2). This method may be again briefly summarised thus:—

Method for counting the number of living and dead leucocytes in a given sample of citrated blood.—The following solutions are prepared and a jelly is made from them. 1. A volume of Unna's polychrome methylene blue (Grübler) is diluted with two volumes of water. 2. A solution containing 2 per cent. of agar in water, filtered and sterilised. 3. An accurately neutralised solution containing 4.5 per cent. sodium citrate, 1.5 per cent. sodium chloride, and 0.225 per cent. atropine sulphate. 4. A 5 per cent. solution of sodium bicarbonate. In a test tube mix one cubic centimetre of the diluted stain, two cubic centimetres of the citrate solution, and three cubic centimetres of the molten agar solution. To this mixture a quantity of the alkaline sodium bicarbonate solution must be added in order to cause the excitant for leucocytes contained in the jelly to diffuse into the cells and the quantity added varies with the temperature of the room.² If measurements are going to be made in a room with a temperature of between 60° and 70° F., about 0.25 cubic centimetre of the alkaline solution should be added. The mixture is then boiled until it froths up the tube and a drop poured on to a slide and allowed to set so as to form a film. Supposing a given capillary tube contains the blood corpuscles of one person mixed with the plasma of another, the average number of living and dead leucocytes in the tube can be estimated by placing a drop of its contents on a cover-glass which is inverted and allowed to fall on the agar film. After two or three minutes the granules but not the nuclei of the living leucocytes will stain and those cells will show exaggerated amœboid movements, whereas the dead cells will remain immobile. Moreover, the dead cells may be achromatic (3), in which case they will not stain. Their nuclei may appear as a single nuclear mass, or their nuclei may even stain, or the dead cells may have undergone other changes which have been described in former papers (1, 2). Field after field should be rapidly passed in front of a 1.6th inch or equivalent objective and the number of the living and dead cells counted. Several preparations can be rapidly examined and an average struck so as to give an estimate of the number of living and dead cells in the given capillary tube. No difficulty is met with in making the counts, for living can be readily differentiated from dead cells by the presence or absence of exaggerated movements.

If all the leucocytes appear to be dead, and especially if the agar jelly has not previously been tested, it is as well to

¹ The word "leucocyte" refers to the polymorphonuclear leucocyte. ² A scale has been given in the former paper.

* The figures within parentheses refer to the bibliography at the end of the article.