ceding seven weeks from 924 to 774; 83 cases were admitted during the week, against numbers declining in the previous four weeks from 105 to 66. The deaths referred to diseases of the respiratory organs in London, which had been 183 and 167 in the preceding two weeks, were 172 last week, and were 7 below the corrected average. The causes of 78, or 2·4 per cent., of the deaths in the twenty-eight towns last week were not certified either by a registered medical practitioner or by a coroner. All the causes of death were duly certified in Sunderland, Portsmouth, Brighton, and in four other smaller towns. The largest proportions of uncertified deaths were registered in Sheffield, Halifax, and Oldham.

HEALTH OF SCOTCH TOWNS.

The annual rate of mortality in the eight Scotch towns, which had been 15·6 and 17·3 per 1000 in the preceding two weeks, declined again to 16·7 in the week ending Aug. 25th; this rate was 1·4 below the mean rate during the same week in the twenty-eight large English towns. The rates in these Scotch towns ranged from 13·5 in Aberdeen and 13·9 in Dundee, to 19·2 in Perth and 21·1 in Paisley. The 421 deaths in the eight towns showed a decline of 17 from the number in the previous week, and included 16 which were referred to diarrhoea, 10 to "fever" (principally enteric), 5 to measles, 4 to scarlet fever, 4 to whooping-cough, 3 to diphtheria, and not one to small-pox; in all, 42 deaths resulted from these principal zymotic diseases, against 44 and 43 in the preceding two weeks. These 42 deaths were equal to an annual rate of 1·7 per 1000, which was 1·9 below the mean rate from the same diseases in the twenty-eight English towns. The deaths attributed to diarrhoea, which had been 22, 18, and 20 in the preceding three weeks, declined last week to 16, of which 11 occurred in Glasgow. The 10 deaths referred to "fever" showed a considerable increase upon the numbers returned in recent weeks, and included 3 in Glasgow, 3 in Edinburgh, and 2 in Greenock. The 5 fatal cases of measles, of which 4 occurred in Paisley, were within 1 of the number in the previous week; while the 4 of scarlet fever showed a decline of 2. The 4 deaths from whooping-cough were fewer than in any previous week of this year, and were all returned in Glasgow; and the 3 fatal cases of diphtheria corresponded with the number in the previous week. The deaths referred to acute diseases of the respiratory organs in the eight towns, which had been 45 and 55 in the preceding two weeks, further rose last week to 70, and exceeded the number in the corresponding week of last year by 2. The causes of 62, or nearly 15 per cent., of the deaths registered in the eight towns during the week were not certified.

HEALTH OF DUBLIN.

The rate of mortality in Dublin, which had been 21·1, 19·4, and 18·2 per 1000 in the preceding three weeks, further declined to 17·9 in the week ending August 25th. During the first eight weeks of the current quarter the death-rate in the city averaged 19·6 per 1000, the mean rate during the same period being 16·2 in London and 15·5 in Edinburgh. The 121 deaths in Dublin showed a further decline of 2 from the numbers in recent weeks; they included 4 which were referred to diarrhoea, 3 to measles, 2 to "fever" (typhus, enteric, or ill-defined), 1 to scarlet fever, and not one either to small-pox, diphtheria, or whooping-cough. Thus 10 deaths resulted from these principal zymotic diseases, against 21, 12 and 13 in the preceding three weeks; these were equal to an annual rate of 1·5 per 1000, the rate from the same diseases being 3·8 in London and 1·2 in Edinburgh. The 3 fatal cases of measles were the first recorded in the city since the middle of July; while those of "fever" and diarrhoea showed a decline from the numbers in the previous week. Six inquest cases and 6 deaths from violence were registered; and 42, or more than a third, of the deaths occurred in public institutions. The causes of 12, or 10 per cent., of the deaths in the city were not certified.

VACCINATION GRANTS.—The following gentlemen have received the Government grant for efficient vaccination in their respective districts: Mr. John Winnall George, public vaccinator of the Malvern and Leigh districts, Worcestershire (fourth time); Mr. John Hamilton, Swadlincote, Burton-on-Trent (sixth time).

Correspondence.

"Audi alteram partem."

IN WHAT DOES CANCER CONSIST?

To the Editors of THE LANCET.

SIRS,—No disease has a more extensive literature than cancer (I use the word in its older and more comprehensive sense), and such being the case, one is somewhat chary in adding to its unwieldy bulk; but I observe that you are liberal in allowing your readers to express their views on this dread disease, and therefore I venture to state what I conceive to be the true explanation of the case, and I am all the more willing to do this because my views, although largely in harmony with recent thought, were formed quite independently; I mean that they have been uninfluenced by the modern literature of the subject. I mention this fact simply and solely because it is of argumentative value. Cancer is a localised tissue dissolution. Of this there can be no doubt, for the process fulfils all the terms of Herbert Spencer's formula. The dissolution is however incomplete, for were it complete there would be death of the part. It is necessary to observe that such partial tissue dissolution may occur in two ways: (1) by a partial undoing of evolution, as in progressive muscular atrophy, in which the anterior ganglion cells undergo a process of inverted evolution; (2) by a more or less complete dissolution, followed by partial evolution, as in the production of an imperfect tissue after inflammation. Now it is evident that in both these cases the result is the same—namely, a falling away of the affected tissue from its previous evolutionary level; the process is, in fact, a dissolution. There is no need to state here the exact part taken by dissolution and partial evolution, respectively, in the several varieties of cancer; it is sufficient to observe that the sarcomatous process is almost entirely dissolutive, but that partial evolution plays an important part in the carcinomata, for in them the affected tissue is first reduced to an embryonic form, and then subsequently undergoes an incomplete evolution. Inasmuch as cancer is a localised, incomplete dissolution, it follows that the process must be largely tinged with an element of reversion, for if an organic aggregate undergoes partial dissolution—becomes partially unbuilt, or what comes to the same thing, becomes partially rebuilt after having been previously unbuilt—it must tend to exhibit characters which it displayed at some one or other period of its evolutionary career.

Let us now seek for the cause of the cancerous dissolution. Let S represent the structure of an individual tissue cell and its connexion with other cells; let E represent the cell environment. Then the formula S + E will represent a set of material conditions, out of which the vital action issues. The behaviour of S will of course largely depend upon the nature of the E. If the E be normal, the S will run through its vital career in orderly fashion, subject of course to such vitiated hereditary tendencies as it may contain; but if the E be abnormal, the S will undergo change. Now, in cancer, the cell suddenly breaks away from its connexion with other cells, and subsequently undergoes a series of dissolutive and partial-evolutionary changes. There is only one way in which this can be effected—namely, by an alteration in cell environment. Sudden dissolution can never occur in any aggregate without sudden alteration of environment. We may therefore say with *absolute certainty* that the cancerous process is due to alteration in the cell E. What is the nature of this E? I have no doubt that it consists of a parasite. The arguments in favour of this view are so obvious to anyone who will think over the matter that there is no need to mention them here. We have arrived thus far: cancer is a localised reversion due to a specific parasite. It is now necessary to add that the reversion is not true or exact; cancerous tissue was never at any evolutionary epoch normal. A mal-environment of sufficient virulence to cause cancerous dissolution cannot possibly lead to a true reversion. Let us suppose, for the sake of argument, that the affected tissue undergoes in the first place a true reversion; the parasitic irritation would very soon cause vitiation of the same—a natural variation, as it were, of the true ancestral state; for unless the cell environment be exactly like that which obtained during the particular evolutionary epoch to which the reversion belongs, we cannot possibly

get a true reversion. I have, for argument's sake, supposed the reversion to be true in the first place, but it is obvious that the process will be vitiated from the very beginning. We may therefore define a cancer as a *localised vitiated reversion due to parasitic irritation*.

An apparent difficulty here presents itself. The secondary tumours are more or less exact reproductions of the primary tumour. How, it may be asked, are we to account for the tissue secondarily affected undergoing the same process of reversion as that primarily affected, even although the evolutionary history of the two be quite different? I can only give one example—squamous epithelioma secondarily affecting the glands of the neck. How can lymphoid tissue and mucous membrane revert to the same epitheliomatous type? I pass by the spermatic theory as unworthy of mention; indeed, this theory involves a misconception of what true spermatic influence is. The explanation is as follows: *epithelial cells plus the specific parasites* are carried away from the primary growth to the seat of secondary affection, and in this situation these epithelial cells behave in exactly the same way, under the same form of irritation, as at the primary seat, the tissue secondarily affected only sharing in the process in so far as it may form the stroma of the tumour. The epithelioma parasite can probably only thrive in a certain kind of epithelium. If carried to a tissue containing the proper epithelium this tissue becomes actually transformed like the primary tissue; but if the parasite be carried to a tissue not containing the needful epithelium, the part will not become secondarily affected unless some epithelial cells are carried away from the seat of primary affection. Space will not allow me to give other examples.

Finally, the question has to be answered: On what does proclivity to cancer depend? In my view, every *individual possesses the potentiality of every form of cancer*. Given the specific cell mal-environment of a certain tissue, and the same form of dissolution or vitiated reversion will occur in all. If only the parasite can multiply in the body and manufacture the poison (which is probably the immediate cause of the dissolutionary process) malignant change will occur in all. The immunity from cancer depends upon the ability of the tissues to prevent this parasitic growth, and not upon their inability to take on malignant change if subjected to the necessary mal-environment; and, on the other hand, proclivity to cancer depends upon the inability of the tissues to prevent this parasitic growth.

I am, Sirs, yours faithfully,

August, 1888.

HARRY CAMPBELL, M.D.

THE BACTERIOLOGY OF YELLOW FEVER.

To the Editors of THE LANCET.

SIRS,—Since the publication, in your issue of July 21st, of your well-drawn abstract of the *résumé* given by Dr. Delgado and myself of our bacteriological investigations on yellow fever in Havana, we have obtained results confirmatory of our previous observations, and suggestive of a modification in the methods of cultivation which, if uniformly adopted, might avoid, it is hoped, future discrepancies between different observers.

Some of the criticism to which our "tetragenous microbe" has given rise might perhaps have been obviated by substituting some more comprehensive name, inasmuch as the characteristic feature of the *Meristæ* (van Tieghem), consisting in the division of successive cocci in two perpendicular directions, does not always, in our case, culminate in the production of a pure tetrad. In drop cultures from pure colonies but a few days old, monococci and diplococci, and also chains mostly of four, but sometimes of three, five, or six, are seen oscillating and twisting about in the field. The monococci split into diplococci, and the latter into triangular groups of three and in tetrads. Many are seen as diplococci which in reality are perfect tetrads, as shown when, by twisting around, the four beads momentarily come into view, and also when, after drying and staining, the number of tetrads is found to be much greater than they appeared to be in the liquid drop. The chains are not usually straight, but more or less curved, or even bent at an angle; their formation may be due to some circumstance which has delayed the tendency to a lateral development. The cocci in these chains are apt to multiply *in situ* without dissociating from the others, whereby new beads appear at the sides or extremities, producing longer

irregular chains with, maybe, a tetrad in its course. During the "monococcus stage" the grouping, especially in dried preparations, may present all the characters of a "staphylococcus," and the chains might be called "streptococci"; but the fact that all these forms are found in the same culture, inoculated from a well-isolated colony in an agar Esmarch tube, shows that they are but phases of development of one and the same micro-organism. The property of liquefying gelatine might possibly appertain to one of these forms and not to the others. In the case of which Dr. Sternberg obtained "a micrococcus in tetrads, a streptococcus, and a liquefying staphylococcus" from a piece of preserved kidney, such an occurrence might be suggested; but the fact that a short bacillus was subsequently found in the same colonies would account for the liquefaction without recurring to that assumption. During the last six weeks I have endeavoured to clear up the two following questions:—

1. Whether the microbe in tetrads and short chains observed by us during the epidemic season of 1887 would again be obtained this year, when proper precautions would be taken to guard against the inclusion of germs previously lying on the surface of the skin.

2. Whether, in violent cases of yellow fever running such a rapid course as to exclude every likelihood of a "mixed infection" (which I believe occurs in most of the *melanic cases*), a *technique* could be devised to bring into evidence the primary germs of our micrococcus supposed to exist in the tissues.

The first point we have investigated by taking surface cultures according to Dr. Sternberg's method, after washing the patient's finger with soap and water and strong alcohol, and again after additional washing with 1 per cent. of bichloride and a second time with alcohol. The cultures, after the first washings (without bichloride), have sometimes shown the presence of surface germs and at other times not. Those after the bichloride application have hitherto remained sterile. In one case, in which both cultures remained sterile, the blood collected at the spot from which they had been taken produced colonies of our micrococcus in tetrads and short chains, and also a short bacillus, single or in chains, the latter resembling Babès' yellow fever *chainettes*. Blister serum, obtained with due precautions, likewise gave colonies of the same micrococcus and others of the bacillus in Babès' *chainettes*. This was a "melanic" case, with abundant black vomit, death occurring on the same day in which the serum was collected; it is possible, therefore, that the bacillus may have proceeded from a "mixed infection," originated in the gastro-intestinal tract.

For the purpose of investigating the second question we have been fortunate in obtaining a necropsy, three hours after death, in a case which proved fatal on the third day of illness, the diagnosis being founded on the usual symptoms: albuminuria, yellowness of the conjunctivæ (intensely marked after death), and congested mottled liver. No black matter or blood had been ejected during life, and only a grey-white semi-liquid mass was found in the stomach and intestines. We collected juices from the liver, kidney, and spleen in sterilised "bouillon bulbs," according to Dr. Sternberg's plan, every precaution being taken. Pieces of liver, kidney, and spleen were soaked in a 1 per cent. solution of bichloride, and wrapped in several layers of cloths steeped in the same solution. Some of the intestinal contents had been collected in dry bulbs. The *technique* which we adopted, as the result of previous experience, consisted in introducing the material to be examined in sterilised gelatine (15 to 20 per cent.), remaining semi-fluid between 30° and 32° C. (the prevailing temperature at the time), keeping the tubes in a slanting position, and preparing agar-agar Esmarch tubes from any sediment or film which might appear in the gelatine. The bouillon bulbs containing juices from the liver, kidney, and spleen, produced a whitish sediment after some forty-eight hours, which developed, in agar Esmarch tubes, round, pale straw-coloured, smooth-edged colonies, consisting of our micrococcus in tetrads and short chains. Those proceeding from the liver were larger and more developed, and those from the spleen the least so. The intestinal contents produced after a few hours a white film, which afterwards sank to the bottom, and gave in agar Esmarch tubes whitish colonies, round or with a notched border, consisting of a short bacillus, single, or in chains of two or three, with oscillatory movements, but not actively mobile. The preserved piece of kidney was