On October 25th, he was feeling quite well. The temperature was 97.4°. The patient rose, had a bath, and went out in the forenoon. The bruised sensation in the side remained for four days subsequently.

SECOND INOCULATION.

This was carried out by Dr. Segundo as before, with the same amount of vaccine, also in the left flank, a little lower down, on November 2nd at 9 A.M. The temperature was 99°. There was less pain after the injection. In two hours' time a large amount of pale urine was discharged, and there was some sweating during the forenoon. At 12.15 P.M. the temperature was 100.8°, at 3 P.M. 99.2°, and at 7 P.M. 98.6°. The pulse during the day was 80, bounding, and of moderate tension. The patient felt very poorly for half an hour about 2 P.M. In the evening a fresh patch of redness appeared at the site of inoculation, less marked than on the first occasion. Appetite was diminished, but there was less thirst than after the first injection.

November 3rd. He had passed a good night. The redness persisted, and was spreading round towards the loin. An enlarged gland had appeared in the left groin, and was distinctly tender. A loose motion was passed. The urine was diminished in amount, and darker in colour than usual. Ehrlich's reaction was not obtained in it. The patient was feeling quite well, and went out in the afternoon.

On November 9th an examination of the finger blood was made for Widal's reaction by Dr. Andrewes in the pathological laboratory of St. Bartholomew's Hospital. The reaction was extremely vigorous and well marked even in a 200-fold dilution.

An inoculation was made in a second patient with another portion of the same vaccine. Very similar symptoms followed, and there was slight poorliness for three or four days; the pain in the flank, though not severe, giving rise to the most urgent of the slight inconveniences experienced.

A second inoculation, ten or twelve days after the first, is highly desirable to secure the full benefit of the vaccine.

The points requiring attention in this practice are to secure perfect asepsis and sterilisation of all instruments employed. The patient should remain in bed for two days, and resolve to keep quiet till all reaction has passed off. With these precautions in healthy subjects there appears to be no cause for anxiety as to a satisfactory result. The subject would do well to have his blood serum tested by Widal's process at intervals of three or six months, or, whenever possible, for some years to come.

Lastly, I will express my thanks to the Pathological Department of Netley Hospital for supplying me with the vaccine, and my high appreciation of Professor Wright's public services in promoting this practice of inoculation which appears to hold out promise of a boon not only to the military forces of this country, but to the whole community.

THE RELATION OF LARGE-CELLED INDURATIVE HYPERPLASIA OF THE LYMPHATIC GLANDS TO TUBERCULOSIS.

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PATHOLOGISTS who have made many *post-mortem* examinations on patients who have died of chronic pulmonary tuberculosis, and who have examined microscopically the lymphatic glands, especially in the cervical and bronchial regions, must be perfectly familiar with this condition. Large-celled indurative hyperplasia of the lymphatic glands is distinguished by the transformation of the normal gland tissue into large-celled tissue, having none of the characteristics of the original structure. This large-celled tissue consists partly of rounded and polygonal cells, and partly of spindle cells. If the proper lymphadenoid tissue has not altogether disappeared, the remaining portions of it form reticulated bands pervading the large-celled tissue.

Fig. 1 shows clearly the microscopical structure of a cervical lymphatic gland that has undergone this change. The normal lymphoid tissue stains more deeply than the large-

celled portion, thus coming out in sharp contrast under the microscope. The process of transformation begins probably with an accumulation of leucocytes in the gland tissue, followed by the development of epithelioid cells with large nuclei. Now what is the significance of this large-celled hyperplasia in a lymphatic gland? Is it a tuberculous change? Are we to look upon a lymphatic gland so changed as tuberculous, although there may be no definite evidence of tubercle, using that term in the usual histological sense? The actual frequency of tuberculosis of the lymphatic glands is not easily determined by mere anatomical inspection, and it is often difficult to say whether a given glandular affection is tuberculous or not.

Fig. 1.

Professor Ziegler¹ in his textbook of pathology separates the large-celled hyperplasias from the tuberculous affections of the lymphatic glands, but he goes on to say that it is not unlikely that tuberculosis may assume this type, and that some of the so-called scrofulous inflammations of the lymphatic glands in which no typical tubercles can be found are really due to the invasion of the tuberculous virus.

The following case is, I think, worthy of record as bearing on this question:

E. T., female, aged 7, died in the City of London Hospital for Diseases of the Chest of morbus cordis, mitral and aortic. The *post-mortem* examination was made by myself twenty-seven hours after death.

Fig. 2.

The body was well nourished, and *rigor mortis* present. The heart was hypertrophied and dilated, and gave evidence of old and recent endocarditis which was the cause of death.

The larynx, trachea, and bronchi were normal. The pleural cavities contained a small quantity of clear straw-coloured fluid, and showed no adhesions. The bronchial glands were enlarged and in parts caseous. At the anterior margin of the middle lobe of the right lung there was a small caseous tuberculous nodule about the size of a small pea, radiating from this immediately under the pleural surface were numerous grey granulations. With this exception there was no evidence of tubercle in the body. The left lung was perfectly free. The tuberculous patch in the right lung was quite limited to the space indicated above. On making microscopical sections the bronchial glands were found to be typically tuberculous, with large giant-cell systems. (See Fig. 2.) The microscopical picture of the lung was typically that of grey miliary tubercle. The whole chain of cervical glands was carefully dissected out on the right side, and the glands were found to be in a condition of large-celled hyperplasia, but with no typical giant-cell systems. The tonsils were very diligently searched for tubercle, but none was found.

I think the inference from the above is clear that this large-celled hyperplasia of the lymphatic glands is of a tuberculous nature. In this case we have the two conditions occurring together. The bronchial glands being typically tuberculous, with secondary lung infection, and the cervical glands being in the condition of large-celled hyperplasia which I look upon as being equally tuberculous. How the bronchial glands became affected is not clear. As already said, diligent search was made in the tonsil, but with a negative result. The case is in other respects an interesting one, because the tuberculous infection was, at any rate in the lung, quite recent, there being no suspicion of tuberculosis until it was discovered at the *post-mortem* examination. Tubercle bacilli could not be found in the cervical glands, but I think we may consider this large-celled hyperplasia as a kind of pretuberculous change in the gland.

REFERENCE.

¹ *Textbook of Pathological Anatomy*, translated by MacAlister, vol. ii, p. 112.

THE DIPHTHERIA MORTALITY OF THE THREE PRINCIPAL AUSTRALIAN COLONIES FOR THE PAST FIFTEEN YEARS,

WITH SPECIAL REFERENCE TO THE INFLUENCE OF ANTITOXIN
ON THE DEATH-RATE.*

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THE antitoxin treatment of diphtheria was introduced into Australia at about the beginning of 1895, and almost immediately came into general use. In the city of Brisbane, for instance, there have been since then very few practitioners—the principal exceptions being those engaged in certain forms of club practice—who would omit to administer an injection of serum to a case of diphtheria. A few cases may have been so treated during the closing months of 1894, but I believe not sufficiently many to exert any appreciable influence on the mortality.

Judging from the great reduction in the case-mortality of the disease in the numerous hospital reports which have appeared since the introduction of the new treatment, it becomes interesting to inquire whether the total yearly mortality as recorded by the Registrars-General of the various colonies shows any signs of reduction. It is obvious that, although the more exact details of cases treated in hospital are best fitted to elucidate the problem as to the efficacy of the treatment, its actual value in saving life under present conditions of use can only be determined by such an inquiry.

The principal difficulty in this investigation is produced by the varying epidemic prevalence of diphtheria, or, to speak more accurately, of fatal cases of diphtheria in different years, quite apart from the question of the possible influence of treatment. If we had had notification of the number of cases of the disease occurring in each year, this difficulty

would have been for the most part removed. In the absence of this, we can only deal with it by taking the average mortality of a sufficiently long series of years so as to eliminate annual variations. We commence our inquiry at the year 1884, and from this to 1894 inclusive we have what may be termed a pre-antitoxin period of eleven years, which again we have divided into two periods of six years and five years respectively. The antitoxin period consists of the four years 1895 to 1898 inclusive. It would be convenient to have a longer antitoxin period, but this is all that is available to us at present. For the colonies of Queensland and New South Wales this period appears fairly satisfactory, as the annual variations in the mortality per 100,000 of mean population are not excessive. Contrary to what might be expected the colony of Victoria, with a population nearly three times that of Queensland, shows much greater variations in mortality. Probably the explanation of this is a climatic one. Queensland and New South Wales, both on the eastern coast of the continent and facing the Pacific, enjoy a very similar climate; Victoria, lying on the south coast and facing the Southern Ocean, has a different and much more variable climate. Whether, however, this is a true explanation of the more variable diphtheria mortality of the last-named colony is immaterial to our present purpose.

Another difficulty lies in the considerable number of cases registered as deaths from "croup." The great majority of these may be taken as cases of membranous croup, for death from true catarrhal croup is rare. Without entering into pathological discussions it may, I think, be taken as universally admitted, that if not all, at least the great majority, of fatal cases of membranous croup, are really cases of diphtheria of the air passages. Accordingly I have included deaths from croup in my statistics, and would point out that if in so doing I have included some deaths not actually due to diphtheria, these would tend to obscure, certainly not to exaggerate, any fall in the mortality due to the use of antitoxin, for it is not contended that non-diphtherial cases are influenced by this remedy.

The increasing proportion of children in the population since the proportional decrease of adult immigration, has, perhaps, had some effect on the annual death-rate, diphtheria being with few exceptions a disease of childhood; but although some slight apparent increase in the mortality of the later years of the series might have been expected from this cause, such is not apparent in the statistics, and evidently the comparative influence of this factor has been inappreciable. The varying meteorological conditions of different years has no doubt a direct influence on the death-rate, but as there still remains considerable obscurity as to the exact weather conditions that are of importance, I have thought it wiser to eliminate this varying factor by the method of averaging, rather than to attempt to consider it in detail.

QUEENSLAND.

Treating the mortality records of the colony of Queensland on these lines, we obtain the following table:

Queensland: Deaths from Croup and Diphtheria.

Year.	No. of Deaths.	Mean Population.	Deaths per 100,000.
1884... ..	135	298,694	45.2
1885... ..	208	318,415	65.3
1886... ..	200	332,510	60.2
1887... ..	159	354,777	44.8
1888... ..	156	377,201	41.4
1889... ..	165	397,061	41.6
1890... ..	232	414,716	55.9
1891... ..	205	404,772	50.7
1892... ..	176	415,813	42.3
1893... ..	156	426,798	36.6
1894... ..	157	438,727	35.8
1895... ..	96	452,852	21.2
1896... ..	60	466,364	12.9
1897... ..	67	478,440	14.0
1898... ..	96	492,602	19.5

Mean annual death-rate per 100,000 living for six years 1884-89, 49.2.

Mean annual death-rate per 100,000 living for five years 1890-94, 44.1.

Mean annual death-rate per 100,000 living for four years 1895-98, 16.4.

* Read in the Section of Public Health of the Intercolonial Medical Congress held in Brisbane.