

could ordinarily effect. Thus probably the motion of the air is turbulent, resembling rolling, any particle in effect alternately proceeding to the hot plate and then to outer cooler air in its progress upwards, thus actually carrying away the heat at a rate greater than could be the case if this turbulence were absent.

(b) *Variation with Total Height and with Temperature Excess.*—To investigate the matter further, a long electrically-heated cylinder was set up vertically and the heat loss was measured for various steady temperatures. The experiment was repeated with cylinders of different lengths obtained by cutting down the original cylinder, thus reducing the height, but leaving other details unchanged.

On plotting on logarithmic paper the experimental

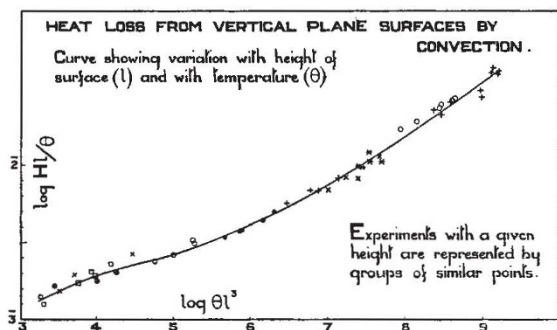


FIG. 4.

data obtained with these cylinders, it was found that for each cylinder the results gave approximately a straight line; that is, a relation of the form  $H \propto \theta^n$ , where  $H$  is the heat loss by convection per unit area for a temperature excess  $\theta^\circ\text{C}$ .

A study of the values obtained for  $n$  showed that the relation between convective heat loss and temperature excess depends upon the height of the wall, and, conversely, it may be shown that the effect of the height depends upon the temperature excess. This pointed to the important conclusion that the equation of heat loss cannot be expressed in such a form that the height and temperature excess are separate factors, but that these variables must be suitably grouped together.

Now the appropriate grouping of the variables may

be deduced from the Principle of Similitude, following the general lines of Rayleigh's treatment for convection in a stream of fluid (NATURE, vol. 95, p. 66, 1915). Solving the "dimensional" equations, we find, when only the temperature and size of the hot body are variable, that

$$H = (\theta/l) F(\theta l^3)$$

where  $H$  is the heat loss per unit area per unit time;  $\theta$  = temperature excess of body;  $l$  = linear dimensions of the body;  $F$  denotes an unknown function of  $(\theta l^3)$ .

The above formula is applicable only to bodies of similar shape, that is to say, the length, width, and depth of which are always in the same proportion. But with sufficiently wide vertical planes obviously the width will not appreciably affect the heat loss  $H$  per unit area, and it may, therefore, be assumed that the formula will also apply to these cases if it is taken to represent the height of the vertical surface. If the above grouping of variables is correct, it follows that points plotted for vertical surfaces with  $H/l/\theta$  as ordinate and  $\theta l^3$  as abscissa will all lie on one line, in spite of the fact that different heights ( $l$ ) and different temperature excess  $\theta$  are used. In Fig. 4, to cover the wide range, logarithmic values have been plotted, and it shows how well the points lie on a smooth curve.

The point of inflexion implies that there is a certain height for which the heat loss per unit area is a maximum or a minimum. The experiments on the wall composed of separate elements show that the minimum can be observed by direct experiment.

Another feature of the curve is that at its upper extremity it approximates to the form:

$$H/l/\theta = (\theta l^3)^{\frac{1}{3}},$$

i.e.  $H \propto \theta^{\frac{4}{3}}$ , and so is independent of the height and varies with the temperature according to a  $\theta^{\frac{4}{3}}$  law. This is also confirmed by direct experiment. Hence there is strong support for the theory employed in correlating the rate of heat loss with the temperature excess and height.

The detailed account of these experiments will be found in Special Report No. 9 of the Food Investigation Board, entitled "The Transmission of Heat by Radiation and Convection," by E. Griffiths and A. H. Davis.

### Local Immunity in Infectious Diseases.

THE usually accepted view that protection against pathogenic bacteria is due to the development of specific antibodies in the blood is disputed by Prof. Besredka of the Pasteur Institute. It is true that after recovery from any infection, or after inoculation with a vaccine consisting of the killed bacteria which cause this infection, the blood acquires properties which it did not possess before; for example, the power of clumping the bacteria or even killing or dissolving them. It was very natural to suppose that the development of these bodies in the blood is directly responsible for recovery from any infection or the failure of the particular organism to gain a footing in successfully vaccinated individuals. Prof. Besredka realised, however, that in certain cases a definite protection exists without the occurrence of such bodies in the blood, and he was led

to the belief that their appearance is a secondary and not a necessary sequel to a protection acquired by the special cells which the particular organism preferred.

In the case of anthrax, to which the guinea-pig is very susceptible indeed, Prof. Besredka has proved by ingenious experiments that inoculation of the killed *Bacillus anthracis* into the peritoneal cavity or tissues other than the skin, is not followed by the development of antibodies in the blood, and that no protection against subsequent inoculation of the skin with living organisms is obtained. If, however, the killed organisms are applied to the skin, or, in other words, if the skin is vaccinated, a definite immunity is acquired and the guinea-pigs, which previously would have contracted a virulent infection by the inoculation

of the skin with a very small dose of living bacilli, now are resistant to very large doses. This immunity is not associated with the appearance of antibodies in the blood. It is evident, therefore, that in order to obtain immunity against anthrax it is necessary to increase the resisting power of the special tissue, namely, the skin which the *Bacillus anthracis* favoured.

Prof. Besredka then turned his attention to other diseases, such as cholera, typhoid and dysentery, the organisms of which attack the lining cells of the intestine. If the same principle maintains in these diseases, then in order to produce an immunity it would be best to vaccinate the intestinal wall by bringing the vaccine of killed organisms in direct contact with it. Experiments on animals have shown that such an immunity can be obtained by administering the vaccines by the mouth instead of injecting them subcutaneously by the usually accepted method. Such an immunity, according to Prof. Besredka, is the direct result of the action of the vaccines on the intestinal cells, and not of their absorption into the blood.

Having obtained these results in animal experiments, attention was directed to human beings, and already certain data have been collected which can be interpreted as indicating that, during outbreaks of typhoid

and dysentery, those who come in contact with the sick can be protected by the ingestion of tablets of killed bacteria. Arrangements have been made by the Health Section of the League of Nations to carry out an investigation on a much larger scale in the case of outbreaks of cholera in Russia. Another application of this principle which is being investigated is the possibility of vaccinating the skin of human beings against staphylococci, the cause of boils and furuncles. In these cases the organisms develop exclusively in the skin, and already evidence is accumulating that protection and even cure can be obtained by applying vaccines to the skin in place of inoculating them subcutaneously according to the generally accepted procedure.

Prof. Besredka's views are undoubtedly revolutionary and, should they ultimately prove sound, are of the greatest practical importance. The inoculation of vaccines subcutaneously is followed by considerable local reaction and discomfort, and sometimes by actual illness, whereas their local applications to the skin or their oral administration give rise to practically no unpleasant symptoms. It is evident that it would be much easier to induce people to be vaccinated in these circumstances.

### Obituary.

PROF. MARCUS M. HARTOG.

BY the death of Prof. Marcus Manuel Hartog in France, at the age of seventy-five years, biological science loses a remarkably accomplished and enthusiastic worker. After a school education in London, he went up to Trinity College, Cambridge, and in 1874 was placed in the first class in the Natural Science Tripos. To many of his old friends it has seemed that if Hartog had had the chance of remaining in Cambridge a year or two after he took his degree to initiate and develop some line of research, he might have attained a position of the highest distinction among the group of Cambridge scientific men of that period; but having married a few months after graduation, he accepted the post of assistant to the Director of the Peradenya Gardens in Ceylon and never resided again in Cambridge. On his return from Ceylon in 1877 he was appointed demonstrator and lecturer in natural history in the Owens College, Manchester, a post which he held until he was appointed to the chair of natural history in Queen's College, Cork, in 1882.

The writings of Prof. Hartog on biological subjects are so widely scattered in English and foreign periodicals that it would be difficult to make a complete list of them, but it may be said that the principal subject which seemed to dominate his mind was the mechanical or chemical conception of some of the more important vital processes. For dealing with such subjects Hartog was particularly well equipped, having a wide knowledge of both botany and zoology and a love of dealing with abstruse philosophical problems. His explanation of the cytoplasmic figure of the dividing cell, published in the Proceedings of the Royal Society in 1904, as a strain figure under the action of a dual force analogous to magnetism, which he called "mitokinetic force," was the result of a long series of careful experiments

and profound philosophical consideration. His two papers published in the *Quarterly Journal of Microscopical Science* in 1891 and 1904 on some "Problems of Reproduction" included valuable criticisms and summaries of the results of the researches by various authors of that period, enlightened by the results of his own special work on the developing egg-cell and his keen critical powers.

Hartog was an enthusiastic member of the British Association, and at almost every meeting held within the British Isles he read a short paper on some investigation upon which he had been engaged during the year. At the Bradford meeting in 1900 he discussed the bearing of his discovery of the presence of a peptic zymase in the developing embryos of the frog and the chick, and expressed the conclusion derived from it that a cell which only accumulates reserve material has no need constantly to readjust its surface to its volume, but when a zymase is formed and it is able to use its reserves, the need for augmented surface asserts itself, and we get cell division.

There is not much from Hartog's pen on systematic zoology, but from the time when he was a demonstrator in zoology in Manchester he took a special interest in the Acinetaria, and at different periods he made several communications on the species and on the structure of this small group of Protozoa. Students of zoology are also indebted to him for the excellent accounts he wrote of the Protozoa and Rotifera for the "Cambridge Natural History."

Without in any way underrating the value of the work Hartog was able to accomplish during a long and active life, it may be felt that had he not been constantly harassed by his manifold duties as professor of three subjects and the want of adequate assistance and resources, his record would have been much greater and more important. He retired from the chair in Cork