is also the curve which expresses the results of certain laws of physical chemistry. It gives the form of the epidemic curve on the assumption that something present in the organism at the beginning of an epidemic decays according to the law of mono-molecular reaction. Of this there are many biological analogies.

2. That b , the constant expressing the rate of loss of infectivity, be not an absolute constant, but varies among the mass of infecting organisms according to the normal law. The coefficient of infectivity at time t will then be given by the form $e^{a-bt+ct^2}$, where c is in general small. This gives an epidemic equation

$$y = Ce^{at - \frac{1}{2}bt^2 + \frac{1}{3}ct^3}$$

In this case the decline of the epidemic is somewhat slower than its rise.

3. That the hypothetical substances necessary for infecting disappear according to some physico-chemical reaction of higher order than the first, or of a mixed order—that is, let it be bi-molecular or some other.

The epidemic equation for this is of the form

$$y = \frac{ae^{mt}}{(t+p)^n(t+p)}$$

None of these three equations are more than rough approximations.

In a previous paper² I have given a method of adapting the first assumption, so as to make it fit a considerable number of epidemics, but of the ultimate worth of this method I am doubtful.

4. That we assume $\frac{1}{y} \frac{dy}{dt} = \frac{a-bt}{m-nt+lt^2}$ instead of $a-bt$, as in (1), where in the denominator the values of m , n , and l are such as to ensure that the sign is always positive. In this case Pearson's Type IV is the solution. This describes many epidemics very closely, but I can offer no *a priori* reason for its probability. The form implies that the organism loses in infecting power from the commencement of the epidemic, but that the rate of this loss is at the beginning less than it ultimately becomes.

5. That the epidemic dies out from want of susceptible persons. Here let there be N susceptible persons: let c be the number of these an infected person is likely to come into contact with at the beginning of the epidemic: let d be a figure denoting the coefficient of the power of infecting, taken constant throughout the epidemic. The number of susceptible persons at time t is obviously

$$N - \int_0^t y dt$$

and consequently the number infected at time t will be equal on the average to

$$c \frac{N - \int_0^t y dt}{N}$$

The general equation thus becomes

$$\frac{1}{y} \frac{dy}{dt} = \log \frac{cd}{N} \left(N - \int_0^t y dt \right)$$

I have not been able to integrate this in a suitable form, though Sir Ronald Ross has succeeded in integrating a similar finite difference equation. Reading his note in the JOURNAL of March 27th suggests that I may have hitherto misinterpreted this equation, and that is partly, and may be wholly, true. Maxima and minima of curves in general are given by the relation $\frac{dy}{dx} = 0$.

For a maximum in this case

$$y \log \frac{cd}{N} \left(N - \int_0^t y dt \right) = 0$$

Thus, either $y = 0$, which is clearly not a maximum, or

$$\log \frac{cd}{N} \left(N - \int_0^t y dt \right) = 0$$

that is $\frac{cd}{N} \left(N - \int_0^t y dt \right) = 1$ or $\int_0^t y dt = N - \frac{N}{cd}$

This equation seems to show that the number of cases which have occurred up to the maximum is greater or

less than half the total number according as cd is greater or less than 2. But the smaller the value of cd the greater number of individuals are left uninfected (for instance, if $cd = 1.5$ and $N = 1,000$, about 400 persons remain uninfected), so that the decline of the curve found on this hypothesis is, even with low infectivity, more rapid than the rise. As cd increases the decline becomes more rapid. But with the value of cd approximately equal to 1.5 very nearly symmetrical curves arise. In addition, these have the relationship between the curve constants, which the epidemic requires as examined from actual statistics. If $cd = 2$, these relationships no longer hold. The exact point where the change from a probable to an improbable epidemic form takes place I have not determined. As in my previous work, such small values of cd were not contemplated; I was led to consider other hypotheses, and I still think that these must play a large, and even the most important, part.

6. Any of the hypotheses Nos. 1 to 4 may be combined with No. 5.

There is, however, another form of epidemic symmetry which possibly arises from reasons quite independent of those given above, or by Sir Ronald Ross. An example of this is the seasonal distribution of enteric fever in London, a curve of remarkable symmetry. If, as Sir Shirley Murphy considers likely, this epidemic was due to impure water, a possible solution offers itself. The seasonal distribution of temperature in London is very closely a simple sine curve, necessarily symmetrical. As most organisms grow better at higher temperature than lower, the simple rate of growth of the enteric organism in the water might quite well give rise on the average to an epidemic form of the type found, without the necessity of any other hypothesis.

Sir Ronald Ross's full paper will be awaited with much interest, whether it furnishes complete solutions or not. The whole subject is of the greatest importance and not intensely difficult, the chief factor which prevents progress being the immense amount of arithmetical work necessary to check the accuracy of any given hypothesis.

REFERENCES.

¹ International Congress of Medicine, London, 1913, Section XVIII, p. 153. ² *Proc. Roy. Soc., Edin.*, 1910, vol. xxxi, p. 252.

FOREIGN BODY IMPACTED AT THE BIFURCATION OF THE TRACHEA EXTRACTED BY AID OF THE BRONCHOSCOPE.

BY

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THE following case will illustrate the value of the direct method of bronchoscopy in the removal of foreign bodies:

A girl, aged 7, staying at Droitwich with her mother, was playing outside a room in which her parent was occupied; she suddenly rushed in and exclaimed, "Oh! mummy, I have swallowed a pin." Dr. Shirley Jones, of Droitwich, was called in and promptly referred the child to Drs. Hall-Edwards and Emrys-Jones, of Birmingham for x-ray examination. A pin shown to Mr. Emrys-Jones, similar in all respects to that swallowed by the child, was a steel pin 1½ in. long, with a blue opaque glass head. The child was screened and an opaque object like the glass head of the pin was seen 1 in. to the right of the spinal column and at the level of the vertebrae. On careful scrutiny, the shaft of the pin was made out pointing to the left horizontally. The pin shadow moved up and down with respiration and was foreshortened on rotation of the body. A radiograph was taken and the blurred head of the pin accurately located, but the shaft of the pin was not visible in the print. After further screening in my presence, it was concluded that the pin, from its movement with respiration and situation so far outside the vertebral column, was impacted in the trachea or lung.

The child was sent into a nursing home to be prepared for bronchoscopy under an anaesthetic the following evening. She had no symptoms, enjoyed her breakfast, and felt no pain on swallowing or respiration. Glucose was given three times in the day. One minim of liquor atropine was injected one hour before the operation to arrest secretion. This amount was effectual for the purpose, and did not mask the pupil signs of anaesthesia by undue dilatation. The anaesthetic was administered by Dr. McCardie, anaesthetist to the General Hospital, a mixture of 2 parts of ether and 1 of chloroform being given.

The second smallest size (No. 5) of Bruning's tubes was passed to the entrance of the larynx and the glottis was swabbed over with 10 per cent. cocaine and adrenalin. After

an interval the tube was reintroduced through the glottis and passed a considerable way down, when a glimpse was seen of the shaft of the pin lying across the bifurcation of the trachea—the head not being visible. Anaesthetic disturbances compelled the removal of the bronchoscope, and the question arose how to extract the

impacted pin—whether to break it in two or withdraw it whole by manipulation. The problem was solved in a fortuitous manner. On quiet deep anaesthesia the tube was reintroduced. The pin, however, could not be seen, and was concealed by a collection of blood and mucus at the bifurcation of the trachea. A cotton-wool carrier was inserted down to the bifurcation to mop away the secretion, and on bringing it away I found that the blue head of the pin was visible. This had accidentally been pushed down into the right bronchus, while the point of the pin was directed upwards, but was slightly impacted in the wall of the trachea where it merged into the left bronchus. One of Bruning's long pincettes was introduced at this critical and anxious moment, the pin grasped, eased from its point of impaction, and brought out.

The next day the child was very slightly hoarse, but the following day even this symptom had disappeared, and she was as well as ever.

One or two points concerning the ease and technique of the operation I should like to emphasize.

1. The value of x rays, which, in the absence of any symptoms, confirmed the child's statement and located the presence of such a small object.

2. The atropine administered before the commencement of the operation was of incalculable value in minimizing the secretion.

3. The swabbing of the laryngeal orifice with cocaine facilitated the introduction of the tubes by abolishing reflex spasm and cough.

Reviewing the circumstances, I would suggest that it would be good practice in a similar case, after the impacting pin has been located, to place the child in the Trendelenburg position, and then, by careful manipulation with a long probe with cotton-wool wrapped round the end, to push one end into a bronchus to disengage it.

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

SHRAPNEL WOUND OF THE SPINE, WITH EXCEPTIONALLY LOW TEMPERATURE.

WITH reference to the case of shrapnel wound of the spine, with exceptionally low temperature, described in the JOURNAL of February 6th, 1915, by Lieutenants Oliver and Winfield, R.A.M.C., may I be permitted to offer a possible physiological explanation of the phenomena recorded therein?

A fall of temperature, varying from 7° to 16° C., as a result of section of the spinal cord in animals has been recorded by various observers, including Tscheschichin, Bernard, Pochoy, and Riegel. Goltz and Ewald, too, found that animals deprived of their spinal cords from the medulla down lived for a long time, but were liable, in spite of all reasonable care, to die of cold, even after the blood vessels had returned to their normal calibre.

Now, Sajous of Philadelphia holds, as a result of experiment and an exhaustive study of the literature of the subject, that one of the main functions of the adrenals is the regulation of metabolism, including oxidation, and hence the regulation of body temperature. He also asserts that this function of the adrenals is controlled by

a centre in the pituitary body, and that these two bodies (pituitary and adrenal) are connected by a nervous path—the pituitaro-adrenal path—which passes from the pituitary to the tuber cinereum and the walls of the third ventricle, thence, by way of the pons and bulb, to the spinal cord. The fibres then leave the spinal cord by the upper four or five dorsal rami, and pass by the great splanchnic nerve and coeliac ganglia to the adrenals. The proofs he gives of these facts are numerous, and I need not go into them in detail here. Any one interested will find them all in his book, *The Internal Secretions and Principles of Medicine*.

How, then, can we explain the exceptionally low temperature and terminal hyperpyrexia in this case? There was evidently at first more or less complete interruption of the pituitaro-adrenal path, due presumably to haemorrhage—haemorrhage into the central canal was observed *post mortem*. The adrenals, thus deprived of the impulses from their controlling centre, would cease to secrete the normal amount of internal secretion, oxidation would be greatly diminished, and the temperature would fall. That the adrenals have an influence on the production of body heat is shown by the facts that: (1) Hypothermia is a constant and prominent symptom of Addison's disease in man and of removal of both organs in animals; (2) injections of suprarenal extract cause a rise of temperature (Oliver, Schäfer, and Reichert).

The terminal rise of temperature progressing to hyperpyrexia is explained by the softening and absorption of the blood clot relieving pressure and the irritation of uninjured fibres—or rather, the intact ends of the distal portion of the injured fibres—by the chemical substances thus produced. Thus a condition of hyperadrenia, with increase of internal secretion, and as a result increased oxidation and rise of temperature in proportion, would be brought about. In this connexion it is important to note that the wound had nearly healed, so that infection as a cause of the hyperpyrexia may be excluded.

Hypotonia of the muscles, as evidenced by the flaccidity of the leg muscles, which were not paralyzed, is a prominent symptom of adrenal deficiency.

The abnormal difference between the mouth and rectal temperatures points to dilatation of the splanchnic arterioles and consequent deprivation of a normal supply of warm blood to the peripheral parts. This would mean low blood pressure—another symptom of hypo-adrenia.

The position of the lesion is important, namely, from the fourth to the sixth cervical vertebrae—the latter marking the upper limit of the outflow from the spinal cord. Ott found animals could generate their usual heat when the section of the cord was below the fifth dorsal vertebra.

In any case, whether we accept Sajous's conception or not, it must be admitted that the lesion under discussion injured a path between some heat centre in the brain and an organ concerned in the regulation of the body temperature, and that this path leaves the spinal cord between the sixth cervical and fifth dorsal vertebrae. I have already shown that there is evidence in favour of the adrenal glands being directly concerned in the regulation of the body temperature.

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CONGENITAL ABSENCE OF BOTH EYEBALLS.

I AM in attendance on a female child, M. H., aged 22 months, suffering from bronchitis. Both eyeballs are absent, and her mother and the midwife, who was present at the birth, inform me that the child was born in this condition. The eyelids on both sides are complete, and are kept constantly closed. On separating them the eye socket formed by the capsule of Tenon is seen to be complete, and there is no sign of either eyeball. There is some contraction of the eye socket and a shortening of the palpebral fissure. There is no other apparent malformation, and the child is bright mentally. She can talk to the other children and call them by their names, and can sing "Tipperary." She cannot yet walk, but this may be due as much to defective nutrition as to loss of vision. There is no history of maternal impression.

Warrington.

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