The exit of the fly is naturally a very critical period in its life-history, and in many cases it is fatal. The freeing process takes between five and ten minutes. When undisturbed the emergent fly rests for a time until its wings and limbs are sufficiently hardened to enable it to fly, or at least to walk about. Sometimes the mosquito takes its first flight straight from the pupa case; at other times it rests awhile before taking to the air. The young imago is pale in colour, the thorax being brown and the abdomen transparent, with a greenish tinge. At first the abdomen is much longer than it is later, for almost immediately after the mosquito's exit from the pupa-case its abdomen begins to contract and from its hinder end four or five drops of a white, glistening, greenish fluid are exuded.

The newly-born imago generally take to flight between five and ten minutes after they have emerged, and they at once begin to darken in colour, and in two hours assume the normal dusky colour of the adult. If anything hinders the insect from properly extending its limbs immediately on issuing from the pupa-case, the parts harden and remain distorted throughout life.

Anyone who has spent a day or two in Lille or Bruges, or other towns in Picardy and in Southern Belgium, will understand why, as my Uncle Toby has it, "Our army swore terribly in Flanders." The incessant and tireless biting of mosquitos would make any army swear, even though they were ignorant, as my Uncle Toby's army certainly was ignorant, that the gnats, as they called them, conveyed tertian and quartan ague. In Europe the trouble is a summer or early autumn trouble; but our troops are fighting in many tropical and subtropical countries, where the mosquitos—like the poor—are always with them.

That the plague can now be checked is shown by the making of the Panama Canal, and that this check is due to British science is shown by the work on the life-history of the malarial organism, first investigated by Sir Ronald Ross, and, as regard the human parasites, by certain Italian savants. It is also due to the public health services of one or two British medical officers of health in the East. Their methods have been applied and improved by those responsible for the channel which now separates North from South America.

We have seen that the larva and the pupa hang on to the surface film of the water by means of certain suspensory hairs, and by the openings of their breathing apparatus. Anything which prevents the breathing tubes reaching the air ensures the death of the larva and pupa, and then there is no issuing adult. Hence the use of paraffin on the pools or breeding places. It, or any other oily fluid, spreads as a thin layer over the surface of the pools and puddles, and clogs the respiratory pores, and the larvae or pupae die of suffocation.

In Ismailia the disease has been reduced to an amazing extent, and remarkable results have followed the use of these preventive measures at Port Swettenham in the Federated Malay States. Within two months of the opening of the port in 1902, 41 out of 49 of the Government quarters were infected, and 118 out of 196 Government servants were ill. Now, after filling up all pools and cleaning the jungle, no single officer has suffered from malaria since July, 1904, and the number of cases amongst the children fell from 34.8 to 0.77 per cent. The only melancholy feature about this wonderful alleviation of suffering due to the untiring efforts of the district surgeon, Dr. Malcolm Watson, is that his fees for attending malarial cases dropped to zero.

Thus, even ten years ago, a considerable degree of success had attended the efforts of the sanitary authorities, largely at the instigation of Sir Ronald Ross, all over the world, to diminish the mosquito plague. It is, of course, equally important to try and destroy the parasite in man by means of quinine. This is, however, a matter of great difficulty. In Africa and in the East nearly all native children are infected with malaria, though they suffer little, and gradually acquire a high degree of immunity. Still, they are always a source of infection; and soldiers stationed in malarious districts should always place their dwellings to the windward of the native settlements.

Knowing the cause, we can now guard against malaria; mosquito nets and wire-protected windows and doors are a sufficient check on the access of *Anopheles* to man. If the mosquito and man could only be kept permanently

apart, we might hope for the disappearance of the parasite from our fauna. In relieving man from this world-wide pest, all genuine lovers of animals will rejoice that we are also relieving the far more acute sufferings of one of the most delicate and beautiful insects that we know.

It has always been a source of surprise to me that the great resources and the very evident enthusiasm of the anti-vivisection societies have not been turned in this direction. In the malarial parasite we have a most potent vivisection of the entrails of one of the most charming and graceful of creatures, whose poetry of movement is hardly approached and never equalled by the ladies of the Russian ballet. A little help, a very little help, would free these fascinating flies from a form of trouble far worse than the human, alternative, host suffers. Yet as far as I know these societies and the societies for the prevention of cruelty to animals have declined to help in any way, and have knowingly allowed hundreds of millions of animals to perish annually by a most painful death and have never stretched out a helping hand to the fairy-like and fascinating mosquito.

ON THE CURVE OF THE EPIDEMIC.

BY

JOHN BROWNLEE, M.D., D.Sc.

In the issue of the JOURNAL for March 27th Sir Ronald Ross published a preliminary communication on the theory of the form of epidemics and allied subjects from a very interesting point of view. My work, as he says, has developed on different lines. As in the immediate future I shall have little time to extend the investigation, I wish to state the conclusions I have come to since my last published paper.¹

Though I have never denied that an epidemic might not end by lack of susceptible persons (the epidemic of small-pox in Boston, U.S.A., 1721, is a marked instance of this), yet I have hitherto been of the opinion that the loss of infecting power on the part of the organism is much the most potent factor in determining the end of an epidemic. This opinion has been based on three grounds: (1) The form of the curve of the epidemic; (2) the variation in infectivity of the organisms of certain diseases (of which measles and small-pox are prominent examples) between epidemic and interepidemic times—this, I think, is proved by observation; (3) the nature of space distribution of an epidemic—for example, the last great epidemic of small-pox in London in 1901, which had a well-marked centre of great prevalence, from which the amount of disease radially decreased. Sir Ronald Ross's notes demand that my arguments on the first count should be reconsidered.

I have formed a general epidemic equation by means of which any conceivable law can be reduced to an approximate mathematical formula and tested against the facts. The general equation is obtained as follows: Let there be an amount of disease y at any period of time; after a unit of time equivalent to the incubation period, let there be $y + \Delta y$: let $\frac{y + \Delta y}{y}$ be called the infectivity at t , t being counted from the commencement of the epidemic. This will be some function of the time, say $f(t)$. Then $\frac{y + \Delta y}{y} = f(t)$.

On the assumption that the law holds also for small fractions of this time, the equation becomes

$$1 + \frac{\Delta y}{y} = (f(t))^{dt}$$

or $1 + \frac{\Delta y}{y} = 1 + \log f(t) dt + \text{terms in higher powers of } dt$. So that in the limit $\frac{1}{y} \frac{dy}{dt} = \log f(t)$.

The most easily imagined cases are as follow:

1. That the infectivity of the organism decreases in a geometrical progression—that is, let $f(t) = e^{a-bt}$, then $\frac{1}{y} \frac{dy}{dt} = a - bt$ and $y = C e^{at - \frac{1}{2}bt^2}$, C being a constant.

This is the normal curve of error, and represents the form of the epidemic curve given by Farr in 1866, though he did not state it in an equation, but as a relationship between the successive numbers in the epidemic wave. It

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

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

The general equation thus becomes

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

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

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

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

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

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

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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.