#### **Epidemic poliomyelitis: some biological studies / by William Mackenzie.**

#### **Contributors**

Mackenzie, William Colin, Sir, 1877-1938. Royal College of Surgeons of England

#### **Publication/Creation**

Melbourne: Shipping Newspapers, 1913.

#### **Persistent URL**

https://wellcomecollection.org/works/htgarxg9

#### **Provider**

Royal College of Surgeons

#### License and attribution

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. Where the originals may be consulted. Conditions of use: it is possible this item is protected by copyright and/or related rights. You are free to use this item in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s).



5.

# EPIDEMIC POLIOMYELITIS

## SOME BIOLOGICAL STUDIES

--- BY ---

WILLIAM MACKENZIE, M.D. (Melb.), F.R.S. (Edin).

(Lecturer on Applied Anatomy to the Melbourne University.)

BRARY

Reprinted from the Australian Medical
——Journal, 25th January, 1913 ——

Melbourne:

Shipping Newspapers Limited, 31 William Street.

EPIDEMIC POLIOMYELITIS

Digitized by the Internet Archive in 2015

https://archive.org/details/b22445997

### EPIDEMIC POLIOMYELITIS.

## Some Biological Studies.

WILLIAM MACKENZIE, M.D. (Melb.), F.R.S. (Edin.).

(Lecturer on Applied Anatomy to the Melbourne University.)

In a pamphlet<sup>1</sup> recently received from Professor Simon Flexner from New York detailing later investigations on Poliomyelitis, which subsequently formed the basis of the Huxley lecture, this distinguished scientist states the present bacteriological position of the disease. It may be summarised as follows:—

The nature of the infecting agent is still unknown.

The virus attacks chiefly the central nervous system. It has been detected in the spinal cord, brain, lymph-nodes, among all internal organs, in the mucous membrane of nose and throat, and in the mucous secretion of the stomach and small and large intestine.

The virus has not been detected in the spleen, kidneys, liver or bone marrows.

The nasal mucous membrane is regarded as the site both of ingress and egress of the poliomyelitis virus in man. Insects are capable of taking up the virus from the blood where it exists in minimal quantities and in harbouring it for a considerable period in an active state.

The disease is one of early childhood, especially the first five years, and appears in a frank and in an

Mode of Infection in Epidemic Poliomyelitis, "Journ. of Amer. Med. Assoc.," Oct. 12, 1912, vol. lix.. No. 15.

abortive or ambulatory form. It can be transported by active infected carriers, having been detected in the secretions of the nose, throat, and intestines, and is limited in its extension by a high natural indisposition or insusceptibility to infection existing among persons of all ages.

As regards two statements made by Professor Flexner, viz., that there exists "no true curative treatment," and chiefly because for the most part when the disease is first recognised it has already caused "irreparable damage," I cordially disagree. In this disease, to speak of "curative treatment" and "most perfect means of cure," is illogical unless the term "cure" be defined. In a child affected with infantile paralysis, it is not a question of "cure," but of restoration of function—a different problem altogether. This fact bacteriologists have to recognise; and the standard not of "cure," but "recovery of function," is high since it denotes an aesthetic as well as a utilitarian factor. In this disease—so distinctive—it is not merely a question of prevention and cure, but "prevention," "cure," and "restoration of function" of paralyzed muscles, or, more correctly, the resumption of certain functions of muscle which are lost.

As regards the second point, viz., "irreparable damage," that also is illogical, for who is to define it. So-called irreparable damage would be recognised through the degree of muscle "paralysis" present, which depends upon the nature of the test applied to the muscle. Thus "irreparable damage" might be applied to the cell condition when the quadriceps failed to contract sufficiently to enable the patient to elevate a lower limb off the bed in one piece, yet such a quadriceps tested from zero might show every other function present. "Irreparable damage" and "paralysis" are really referable not to the muscle but to the loss of part of its function, the restoration of which follows a biological law. So-called "irreparable damage" must be judged by loss of all functions of a muscle—all must be tested, not some, or any. This simple biological question bacteriologists who discuss treatment or cure have signally failed to appreciate in this disease, yet it is the basis of scientific treatment. In such a disease as

Pseudo-Hyp paralysis<sup>2</sup> the same mistake has been Muscles are demonstrated as irreparable owing to loss of part of function, e.g. loss of power necessary to maintain the erect attitude-the last acquired the first to go. Thus the Ectogluteus may be regarded as irreparably diseased and the Biceps-Pronator or other upper limb muscle as non-affected, yet histologically such muscles will show the same degree of degeneration as the Glutei. A deficiency of structure may be associated with good elbow flexion and good forearm pronation, but, throw an extra strain on the upper limb, such as weight of the body, by asking the patient to support himself on a trapeze bar, and he will be unable to do so, this in spite of the fact that only one or two upper limb muscles may be regarded as being slightly affected. Professor Flexner's pamphlet is most important, for the issue is now clearly defined: On one side the bacteriologist seeking for the cause, be it chemical or bacteriological, and hoping to mitigate the evil by the prevention of its spread; on the other, recognition of the fact that the principle of treatment is a biological one. The hope of cure of "paralysis" by drugs or serum is scarcely worthy of consideration in this disease, which, dealing with the relation

<sup>2.</sup> I refer more particularly to the case of a boy kindly sent to me by Drs. Florence and Henry with the usual history of affection of family groups—the girls escaping and the males being affected. Portions of muscles were removed from the upper and lower limbs under chloroform, and examined histologically by Alex. Thwaites. The Zero test showed, also, that muscles ordinarily regarded as non-affected were really affected ones. An endeavour might be made to give effect to Weismann's views of inheritance (viz., the somatic and germinal), of which this disease must be cited as illustrative. We have a mother free (somatic element unaffected, germinal affected), but her male children develop the disease (somatic and germinal). In other words, the female, though affected germinally, possesses some element that keeps somatic affection in abeyance, and hence the suggestion might be made of giving the male ovarian extract. And, as the disease is not localised to certain muscles, but generalized, one sees that massage and electricity applied to muscle fibres breaking down, as in this disease, is scarcely scientific. The idea would be rather rest (e.g. in a double Thomas splint), and if the patient be seen early an attempt at re-education of muscles from zero, as was actually done in the above case.

of nerve cell and muscle, strikes at the very genesis of structure. The real treatment is that at the outset, when, by scientific rest and the recognition that the resumption of function of the muscle is along a biological plane—i.e., follows the line of the acquisition of its function—the damage is limited and function restored. Later, through the muscle our treatment is directed towards the restoration of function of the partially damaged cell, or the bringing of new nerve control factors into play, remembering always cell adaptability and the precedence in biology of function over structure. I have already dealt with the significance of the "zero" position of muscle in this disease, and would again point out that it must be a basic factor in cases of nerve injury involving muscle, or disease in muscle itself. The significance of the biological factor in an old case could be illustrated by a condition of foot eversion following paralysis, the Tibials being "paralysed" and the unaffected Peronei pulling the foot out. The idea of the German school would be to strengthen the weaker group by grafting on to the Tibials from unaffected or recovered extensors or flexors. But, biologically one knows that inversion and eversion of the foot are diminishing, that the Tibia is the dominant bone in the leg, that the Fibula is disappearing. Just as the appendix<sup>3</sup> approaches the Ileum, becoming finally incorporated in its wall, as in Phascolomys, so does the Fibula the Tibia, as evidenced by Macropus; and thus a struggle exists round the ankle as elsewhere between individual muscles, not only for position, but for existence—a fact Hunter seemed to recognise. Hence the biological idea would be not to strengthen the weak, but to weaken the strong. The suggestion might then be made of division of the Peronei since the Tibials were "paralysed," but, recognising again that paralysis in this disease may denote merely loss of part of function, the abolition of eversion might mean recovery of inversion with deformity. Hence we wish to weaken eversion, but not to abolish it: so which is to go, the Longus or

<sup>3.</sup> This is beautifully illustrated in a series of dissections from Phascolomys, particulars of which will be published shortly.

Brevis? Now, in Phascolarctus the Peroneus longas is an adductor of the big toe, and in Phascolomys, with his diminutive big toe, we see a relative diminution in size of the Longus, which is markedly seen in Macropus. The Longus attachment is retreating, and a retreating muscle Low down in weakening one. position of the Brevis tebrate scale the fixed, and a comparison of these muscles in Phascolomys, Macropus, and Phascolarctus4 shows the greater belly muscularity of the Brevis, which is the principal everter, and not the Longus; so, to weaken eversion without its abolition, the Brevis must go. Traction made with a forceps on the exposed Peronei in the living subject demonstrates the comparative weakness of the Longus in everting as against the Brevis. And similarly with the Tibialis Post and Anticus: the Anticus is the selected muscle, and not the Posticus, and low down in the vertebrate scale we may see a Tibialis Anticus even larger than the extensors. In Macropus, a powerful inversion is noticed, not to counteract the Peronei, but to overcome the gravitational effect of a large body poised on practically one toe; the Anticus, which is relatively enormous, is chosen, while the Posticus has diminished to an almost tendinous thread dependent for its diminutive pull on the flexors<sup>5</sup>. It may be mentioned as interesting that, al-

4. These three members of their order I have chosen for the following reasons:—

In Phascolomys, without doubt the "selected" member, we have extraordinary muscular development (perhaps the greatest in existence) for purposes of

strength.

In Phascolarctus Cinereus, with a double pollux and the big toe acting as a pollux, we can study muscular co-ordinations and adjustments. And in Macropus, where we have the attempt at the erect position, we see the development of certain muscles and the retrogression of others, especially in the upper limb and leg and foot—thus enabling us to estimate selected members by survival results.

5. In bad cases of foot eversion following paralysis, where the gravitational effect is paramount, and the internal lat ligature has yielded, it may be necessary to divide the two peronei and encourage the development of the tibials, more especially by a position of inversion of the foot and division of the deep fascia over the

anticus.

though the rapidity or perfection of recovery is greater on the right than the left side, occasionally the reverse is noted, a condition I have found to be always associated with left-handedness.

In spite of the temptation, I do not wish to deal with the biological question further in this review. I will do so shortly, when certain comparative studies are completed, and then I hope to show that the question of cure can be definitely placed on a scientific basis, and that the "paralysed" swinging contracted lower limb is not "pathological," but biological. The magnitude and difficulty of the work will be recognised when it is stated that merely the question of the selection of the foot muscles has involved numberless dissections extending over the last two years. Perhaps I may be pardoned for drawing attention to the brilliant results obtained by Professor Mayer, of Koln, Germany, in the treatment of early cases of infantile paralysis based on the writer's paper on treatment of Infantile Paralysis of the Upper Limb, originally published in this Journal. Mayer's results, read at the Orthopedic Congress in Berlin, were published last year in the Deut Med. Woch., and later in the International Clinics, 1911.

Needless to say, Simon Flexner has our thanks and congratulations for all he has done to help in the elucidation of this difficult problem. True, he has not discovered the actual cause, but his work on the nature of its infection will remain a classic. In conclusion, one may be forgiven for an expression of envy at the vast sums devoted to research in America when we reflect that in Australia, with its enormous wealth and its magnificent hospitals, gaols, asylums and penitentiaries, there is not a penny for the endowment of medical research, nor an institution for the carrying it out. Everything must be paid out of the investigator's pocket, and the wonder is not that so little has been done. but that with the almost insuperable difficulties placed in the way anything has been done at all.