SUMMARY.

(a) What, then, may be expected from the use of a strong salt solution in a wound?

By a process of osmosis water is extracted from intact cells, blood vessels, and closed lymph spaces. The same interaction increases the NaCl content of intact cells and those lymph spaces which may be slowly drained. When the strong salt solution is diluted or withheld, a rapid increase of water within the cells is to be anticipated. If continued for a long period, the colloid constituents of the cells imbibe water and the basis of oedema is established. A small amount of dialysable albuminous substances will emigrate from the cells, due to the absence of such substances in the dressing solution. This emigration will be unaffected by the NaCl concentration of the solution.

By a process of diffusion an interchange of NaCl and water will occur between the free fluids in the intercellular spaces and cut lymph channels which are in contact with the dressing solution, resulting in a concentration of the salt in these fluids.

At the same time a slow diffusion outward of all soluble proteins and other bodies (with the exception of NaCl) will occur, due to their absence in the dressing solution. Again, the NaCl concentration will not affect the rate of diffusion of these substances, nor will an outward current be produced by the interchange of NaCl and water and thus carry with it the free lymph. There remains the normally brisk reaction of secretion upon which we have to fall back for the desirable increase in antibacterial substances. This may be seriously inhibited by the excess of NaCl in the cells.

The migration of leucocytes will be checked and those on the surface of the wound probably destroyed.

The tryptic digestion of sloughs will be suppressed.

The character of the antibacterial substances secreted or diffusing into the wound may possibly be so altered as to become inactive by their dilution and solution in strong salt solution. The activity of the bacteria may be slightly reduced by the antiseptic action of NaCl in concentrations of 5 per cent. or more.

(b) What is to be expected from "physiological saline"?

The amount of osmosis is negligible as far as the interchange of NaCl and H₂O is concerned; there will be only a slight increase in the NaCl content of the cells. There will, however, be the same positive osmotic pressure of the albuminous substances within the cells resulting in the attraction of water. Therefore the waterlogging of the tissues will occur if the solution be used for a long period.

The process of diffusion will allow of an equal outward movement of soluble proteins as in the case of hypertonic saline solution.

The secretion of the cells will proceed without serious interference by the dressing solution until the gradual accumulation of water diminishes their power of reacting.

The migration of the leucocytes will proceed normally or be increased and their beneficial activities remain unaltered.

The tryptic digestion and dissolution of the sloughs in the wound will proceed at its maximum rate, free from the inhibition of strong salt solution.

The character of the antibacterial albuminous substances, diffusing or being secreted in the surface of the wound, will be unaltered.

(c) What is to be expected from hypotonic saline?

By the process of osmosis the combined positive osmotic pressure of the salts and proteins within the cells will be exerted. The result will be a rapid extraction of salts from the cell, and a rapid accumulation of water, with the early establishment of a waterlogged condition.

The process of diffusion will allow of an equal outward movement of soluble proteins as in the case of hypertonic and isotonic solutions. In addition, there will be a similar emigration of NaCl. The secretion by the cells will be interfered with as a result of the extraction of salts and the rapid accumulation of water. The migration of leucocytes will be checked.

The tryptic digestion of the sloughs will proceed a little more slowly than with isotonic solutions, but much more rapidly than with hypertonic.

The character of the antibacterial substances will probably be altered, and possibly be rendered inactive by dilution and solution in an abnormal menstruum.

CLINICAL EVIDENCE.

What clinical evidence is there of the success of the treatment with hypertonic salt dressings? Aside from the claims of its author, there have been scattered reports in its favour appearing in various medical journals. There is also a strong minority which is disappointed in the treatment. It is claimed that the infection is not diminished and that the wound is left in poorer condition by its use than would have been the case if it had been left alone. Attempts are made to reinforce the weak antiseptic action of the high NaCl content by the addition of a more active germicide. This is merely additional evidence that it has proved unsatisfactory.

We have seen it used rather extensively, and have not been convinced that it produced the results claimed for it. The saprophytic bacterial flora persisted quite as long as with the ordinary methods of antiseptic treatment, and the separation of sloughs did not appear to be appreciably hastened. We have, in addition, felt that oedema of the tissues around the wound occurred more frequently after the use of hypertonic saline than with other dressing solutions, possibly as the result of an increased deposit of salt. Furthermore, the recuperative reaction in the wound appeared to be retarded.

CONCLUSION.

The clinical superiority of the treatment by hypertonic saline dressings does not appear to be so decisive that the theoretical and experimental objections to their use can be disregarded.

The theoretical indications for the use of salt solutions appear to be based on an untenable hypothesis of the structure of the tissues about the wound, and an erroneous interpretation of physical and physiological laws.

Certain characters possessed by hypertonic NaCl solution are theoretically undesirable in a dressing solution, and their harmful action can be demonstrated experimentally and clinically.

Certain desirable results claimed to be effected by the use of hypertonic NaCl dressings cannot be demonstrated *in vitro*.

The chief beneficial character held by strong salt solutions, aside from the cleansing property possessed by all watery solutions, is their mild antiseptic action.

A detailed investigation of the specific character of special antiseptics may be a more profitable undertaking than an exclusive concentration upon the physical and physiological properties of sodium chloride.

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RECENT EPIDEMIC OUTBREAKS OF ACUTE
POLIOMYELITIS.

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It seems to be clearly established that acute poliomyelitis has only in comparatively recent years appeared in epidemic form. The first definite and clear reference in the literature to an epidemic outbreak occurs in 1881, when Bergenholtz recorded the occurrence of 18 cases in one district in Northern Sweden; but it was not until the appearance of Medin's well-known record of 43 cases in Stockholm in 1887 that attention was called to the subject. Since that time outbreaks have been more frequent and ever-increasing in extent. In 1899 Wickman reported 54 cases in Stockholm, and in the same year Leegaard described an outbreak, also of 54 cases, which had occurred at Bratsburg in Norway, between July and October. Wickman's excellent record of his observations in over 1,100 cases in Sweden in 1905-6, his accurate description of the various clinical types met with in that epidemic, and of the *post-mortem* findings in the fatal cases, mark a great advance in our knowledge of the disease. In the same years—1905-6—Norway also suffered severely, about 1,000 cases being reported.

In Germany small outbreaks had been reported from time to time since 1886; the first large outbreak there

* Abridged from a paper read before the society.

occurred in Westphalia in 1909, 700 cases being reported, and there were at least 1,000 cases in Germany in that year. About the same time outbreaks were reported from Austria, Holland, Switzerland, Russia, and Australia. In France reports of epidemic outbreaks began to appear in 1888, and gradually became more numerous. Netter reported 100 cases in Paris in 1909. Italy had much the same experience, and small outbreaks also occurred in Spain. America came in with a record of 38 cases in Massachusetts in 1892, and since then has had annual visitations of ever-increasing magnitude. Epidemics increased in number and extent until 1910, when, all over Europe and in the United States and Canada, the disease was very prevalent. Between the years 1907 and 1910 America had, according to Flexner, 23,000 reported cases, and it is estimated that at least 20 per cent. of the cases were not reported.

In the British Isles the first report of an epidemic outbreak was made in 1897 by Dr. Pasteur, who met with 7 cases in one family. Reports of small outbreaks then began to appear more frequently. The first large outbreak was reported by Parker of Bristol in 1909; there were 37 cases. In 1910, 34 cases were reported in Carlisle, 37 in Barrow, and it is known that quite a large number of cases occurred in the towns and rural districts in the North of England. In that same year—1910—quite an extensive epidemic occurred in Leicestershire and Nottingham, Bingham and Melton Mowbray districts being most affected. In this outbreak, as has so often been the case elsewhere, both in Europe and America, the true nature of the epidemic was not at first recognized. For a considerable time the cases were diagnosed as cerebro-spinal fever. This mistake was the more easily accounted for by the fact that the meningitic type was common, and that there occurred at the same time cases of true meningococcal meningitis in the city of Nottingham. In 1911 also there were many cases in England, and quite an extensive epidemic was reported from Devon and Cornwall. The exact number of cases in this outbreak is not definitely known, but it was certainly over 200. This epidemic was very severe and the mortality in it high. In this outbreak also the cases were at first reported as cerebro-spinal meningitis. In Scotland the records are few, but outbreaks have occurred. Low reported 62 cases in and near Edinburgh in 1910.

In Ireland, at least since 1909, cases have been more common, and many small outbreaks have been observed. In the autumn of 1911 there were many cases in Belfast and the neighbouring counties. It was again very prevalent in our own district in 1913. Severe localized outbreaks have occurred in several districts—mainly rural districts—throughout the country. The best known of these occurred at Coagh, co. Tyrone, mainly in the practice of Dr. Burgess, commencing in October, 1913. Dr. Burgess had within a few weeks thirty-seven cases in a comparatively sparsely populated country district. A few months later Dr. Burgess had a single house outbreak; in this house there were six cases of very virulent type, with four deaths. During the progress of this outbreak I had an opportunity of visiting the district with the late Dr. Brian O'Brien and Dr. Burgess, and of examining some seventeen or eighteen of these patients, then in all stages of the disease. At the same time as the Coagh outbreak some fourteen cases occurred in quick succession at Irvinestown. It is not possible to obtain anything like complete figures for Ireland. I am indebted to Dr. Bigger, of the Local Government Board, for a return of the cases reported to the Board since 1912. From that return it does not appear that, with the exception of these two instances, there has been any considerable localized outbreak.

Seasonal Prevalence.—Practically all these outbreaks have occurred in the late summer and autumn. Some variation is shown in the times at which the period of maximum intensity has been reached. In many instances it has been in August, at other times as late as October. In some places cases have continued to crop up all through the winter months.

Symptomatology.

In all these epidemics the clinical types met with have been similar. Wickman's main types are—

I. The *abortive* type, in which the prodromal symptoms are present, but which clears up quickly without any paralysis.

II. The *spinal* type. In this group is included a large percentage of the cases. It is characterized by the usual onset followed by paralysis, temporary or permanent, of the limbs or muscles of the trunk.

III. The *ascending* or *descending* type, fortunately a small group in which the paralysis generally begins in the lower limbs, less frequently in the arms, and gradually extends and involves the muscles of respiration, terminating in death after a few days' illness.

IV. The *bulbar* or *pontine* type, in which some of the cranial nerves are affected, frequently in conjunction with some involvement of the spinal centres.

V. The *cerebral* type in which changes occur in the cortex of the brain, giving rise to spastic hemiplegia or monoplegia.

VI. The *meningitic* type, in which the symptoms closely resemble those of cerebro-spinal fever, the pia mater being much involved. Netter found that 29 per cent. of his cases were of this variety.

Wickman also describes (a) an *ataxic* type, but it seems doubtful if this variety should not be classed with the bulbar and pontine cases; and (b) a *polyneuritic* type, which many writers think should be included in the "spinal" class.

The incubation period is said to be generally five to ten days, but many observers believe they have met with instances of much longer periods, and Wickman reports one in which he was satisfied it could not have been longer than one day.

Whatever the type, the onset is always much the same, varying only in severity: An initial rise of temperature, generally digestive disturbances with vomiting and occasionally diarrhoea, drowsiness, profuse sweating, irritability, marked hyperaesthesia, with pain and general tenderness. Wickman lays stress on the diagnostic value of (1) drowsiness, (2) pain and tenderness, (3) stiffness of the neck, and (4) profuse sweating. According to Müller the outstanding signs are (1) the profuse sweating, (2) the hyperaesthesia, and (3) leucopenia.

In the cases which occurred in our own neighbourhood in the last few years all Wickman's types were met with, and I have seen examples of them all. The spinal type was common; several showed involvement of the cranial nerves.

In 1909 I saw with Dr. Gaussen two cases in one family at Lambeg, children of 10 and 12 years; both had taken ill on the same day. When I saw them, on the morning of the fourth day of illness, one showed complete hemiplegia, was unconscious, with rapid breathing and a temperature of 104°, with drenching sweating. This child died a day or two later; this was a typical example of the cerebral type. The other had a comparatively mild attack of the *spinal* type with partial paralysis of both legs.

In the autumn of 1913 there were many cases in the city. At that time I saw with Dr. McLorinan four cases in one family; all had taken ill within a couple of days. When I first saw them two had comparatively mild attacks of *spinal* type—one with partial paralysis in both legs, the other with partial paralysis in one arm. Another was still very acute, showed profuse sweating, with high temperature, and very great tenderness all over, especially in the legs. The dread of being touched was so great that the child when any one approached the bed held itself quite rigid and gripped the mattress with a hand on each side; the sweating was so free as to drench the pillow and bed. There was no mental confusion. We inquired from the mother if the other members of the family were quite well; she told us that the youngest child, an infant of ten months, seemed dull but not really ill. On examining this infant we found it had a temperature of 101°, was rather apathetic, and had complete absence of knee-jerks. This child did not develop any further signs. The temperature next morning was normal, but the absence of the patellar reflex remained for some time. This I take to be a typical example of the *abortive* type.

At this time, when there were many cases of poliomyelitis occurring in the city, I saw two cases of "Landry's paralysis" within a fortnight. One was a powerfully-built man of 21 years who had taken ill with headache, general tenderness, and mild fever with some vomiting. The vomiting and headache cleared off but the temperature remained elevated without obvious cause. I saw him with Dr. Martin on the fifth day of his illness: he had that morning shown some weakness in his legs and had complete loss of patellar reflex. He was removed to Pardysburn Hospital. Next day there was complete paralysis of the legs; the paralysis quickly extended to the arms and later to the respiratory muscles, and he died from the respiratory paralysis on the ninth day. The other was a case of Dr. Burnside's, a boy of 15 years. In this case the paralysis had extended much more slowly. When I saw him the legs were completely paralysed and the arms almost so. He was unable

to swallow and there was great difficulty in respiration. He died the following day.

Of the *ataxic* type I have only met with one case. This case I saw with Dr. Rentoul in Lisburn. Two children in one family had been taken ill on the same day. One developed a very severe attack of the combined meningeal and spinal type with complete paralysis of both legs, which was permanent. The other, a boy of 7 years, after a short feverish attack with gastric disturbance lasting a couple of days, developed a very ataxic gait. When I saw him the fever had cleared up. In bed he appeared quite well and very bright. He showed complete loss of knee-jerks, and when stood upon the floor he was able to walk without assistance, but with a *very* staggering gait. There was then no pain or tenderness. He was most anxious to give exhibitions of this staggering, which he treated as a great joke. He was kept at rest in bed and the ataxia rapidly disappeared. The knee-jerks remained absent for a considerable time.

Landry's Paralysis.—The position of "Landry's paralysis" in relation to acute poliomyelitis has given rise to much writing. Strümpell had insisted from clinical observation that they were identical. Wickman satisfied himself from histological findings that this view is correct. Römer states that "Landry's paralysis is only infantile paralysis with a fatal issue." Zimmerman as early as 1885, from *post-mortem* evidences, came to the conclusion that "Landry's paralysis and infantile paralysis formed only different degrees of one and the same disease." Römer refers to Landry's paralysis as "poliomyelitis acutissima."

Second Attacks.—Second attacks have been reported by many observers, but appear to be very rare. They have also been experimentally produced in the monkey.

Case Mortality.

In examining the records it is impossible to avoid the conclusion that the figures are not always closely comparable. In some outbreaks the abortive cases appear to have been carefully looked for, in others to have been generally overlooked. Wickman found that the abortive cases in some outbreaks accounted for over 50 per cent. of the total cases. In most of the mortality tables the abortive cases are excluded, only those in which there was definite evidence of paralysis being counted. Then the case mortality has generally been between 10 per cent. and 30 per cent., though in some outbreaks it appears to have been considerably higher.

Case Mortality Recorded by Various Observers.

		Mortality.
Wickman	868 cases in Sweden in 1905	16.7%
Leegaard	577 cases in Norway in 1905	14.5%
Krause	633 cases in Westphalia in 1909	12.3%
Müller	100 cases in Hesse-Nassau in 1909	16.0%
Frost	150 cases in Cincinnati in 1911	30.7%
Frost	1108 cases in New York in 1912	16.5%
England:		
Reece	154 cases in Cornwall and Devon in 1911	22.1%
Farrar	74 cases in the Midlands in 1910	10.8%
Ireland:		
Burgess	37 cases in Coagh, co. Tyrone, in 1913-14	27.0%

Etiology.

Dr. Robb gave an account of the researches which led to the establishment of the fact that the infecting agent is a filter-passing organism, and proceeded as follows:

It was soon found that specific antibodies were present in the serum of humans and monkeys who had recovered from the disease. The blood serum from such cases when mixed with the virus destroys the infective power of the virus. The serum retains this power for a very long period. Netter records a case in which the blood serum was found to be still active after thirty-two years. By this test it has been possible to prove (1) that epidemics in different countries are due to the same infecting agent; (2) that sporadic cases and epidemic cases are due to the same cause; (3) that the "abortive" cases are true cases of poliomyelitis, and it is also of great value in settling the diagnosis in doubtful cases. It has the disadvantage of being very expensive, as two monkeys are required.

Immunization.—All attempts to find a method of immunizing monkeys have so far failed. (1) Inoculations with active virus in minute doses; (2) inoculations with dried virus; (3) inoculations with virus attenuated or killed by chemical agents; (4) inoculations with heated virus; (5) inoculations with mixtures of virus and serum containing antibodies. So far no method which is effective and free from danger has been found.

Serum Therapy.—At present there does not seem to be much prospect of the successful employment of serum therapy in poliomyelitis. Where monkeys are infected by intracerebral injection of the active virus, the giving at the same time of large doses of serum containing antibodies, either by the intravenous, intraspinal, or intraperitoneal route, produces no results. Even if such a method of treatment were efficacious the fact that the ordinary "serum animals" could not be made use of must prove a great difficulty. Attempts have been made to treat acute cases of the disease with the serum from human cases which have recovered from an attack. The BRITISH MEDICAL JOURNAL published recently a note of the employment of this method by Netter in 32 cases. The results are still doubtful.

Method of Spread.

Two main views on the method of transmission of the disease were for a time held: (1) That the disease is communicated by personal contact; and (2) that it is conveyed by insects, the stable-fly being held mainly responsible.

Rosenau, of Harvard, in 1912 reported that he had successfully transmitted the disease by allowing numbers of the stable-fly (*Stomoxys calcitrans*) first to feed on an infected monkey, and by then transferring them to a healthy monkey. Anderson and Frost, of Washington, also reported that they had conveyed the disease in this way; but many other observers, after careful and elaborate experiments, obtained completely negative results, and Anderson and Frost failed in all their attempts to repeat their experiment. The fact that only after the injection of *very large* doses of the active virus can the disease be produced when the intravenous route is employed also goes far to establish the improbability of biting insects being the means of spread. After an exhaustive inquiry the stable-fly has now left the court without this stain upon its character.

It is now generally accepted that the disease is spread by personal contact (1) by acute cases; (2) by abortive cases; (3) by chronic carriers; and (4) by healthy carriers. It has been proved that the active microbic agent is at times to be found in the nasal washings of all these classes, and it is generally believed that the disease is conveyed by the passage of this active agent from the upper respiratory tract of one individual to the upper respiratory tract of another. It is possible readily to infect monkeys by rubbing in the active virus to the *abraded* nasal mucous membrane. It has been long known that the disease spreads along routes of traffic, and that schools are often important factors in spreading the infection, and there is no doubt that should the disease appear in epidemic form in Belfast the picture palaces would be accused of helping in its dissemination, as they have been accused, perhaps with truth, of helping to keep alive our scarlatina.

General Measures for the Control of Epidemics.

The attack-rate in epidemic outbreaks of poliomyelitis is, as a rule, a low one; to this rule, it is true, there have been exceptions. For instance, in the outbreak in Nauru there were 700 cases in an island population of 2,500.

In England a step in the right direction was taken at the beginning of 1913, when the disease was made compulsorily notifiable. Scotland and Ireland followed, but with a shorter step, for in Scotland and Ireland it is left to the option of each sanitary area to adopt notification, or not to do so, as they may think advisable. This is much to be regretted, for only universal notification is of much value. With us in Belfast the disease has been notifiable for a couple of years.

In America isolation of the cases, and in some places also of the contacts, is the rule. In Vermont the acute cases are being isolated for a minimum period of six weeks, and all known contacts for two weeks.

Drug Treatment for Contacts.—It had been found by Flexner that the administration of large doses of urotropin to monkeys before giving to them an infective dose of the active virus modified the results. In some cases the attack was prevented altogether, in others the incubation period was increased from the normal, of five to eight days, up to twenty-four days, and the attack, if it occurred, was generally mild. Urotropin has been much prescribed for known contacts,