VOL. XXVII No. 1

The Nova Scotia Medical Bulletin

OFFICIAL ORGAN OF THE MEDICAL SOCIETY OF NOVA SCOTIA
CANADIAN MEDICAL ASSOCIATION NOVA SCOTIA DIVISION.

JANUARY, 1948

Published on the 20th of each month and mailed to all physicians and hospitals in Nova Scotia. Advertising forms close on the last day of the preceding month. Manuscripts should be in the hands of the editors on or before the 1st of the month. Subscription Price:—$3.00 per year.

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Contents January, 1948

The Epidemiology of Poliomyelitis*

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DURING the past ten or fifteen years there have been a number of interesting and important additions to our knowledge of the clinical aspects of poliomyelitis and to our understanding of its epidemiological features. It is not possible in the time available to do justice to all of the recent work in the epidemiological field, and I have chosen to discuss only those aspects which, I think, are likely to be of interest to the practicing physician or which are of basic importance to an understanding of this disease.

We frequently hear the statement that poliomyelitis is a disease that we know little or nothing about. A few months ago I heard a physician make the following comment: "It is too bad that we don't know anything about the epidemiology of poliomyelitis. If we did we could bring it under control. It is still a mystery to me how one case will occur in Amherst and the next in Halifax with absolutely no relation between them."

Perhaps most of us would hesitate to make quite such a sweeping statement as this, but comments of like nature occasionally appear even in the medical journals. It seems to me that members of the medical profession exhibit undue modesty on this subject. Of course there are still wide gaps in our knowledge of poliomyelitis, but we are far from being in complete ignorance of its epidemiological characteristics. Definite advances have been made, and much promising work is in progress. Poliomyelitis excites an intense fear, almost panic, in the minds of the general public, and any understatement concerning the extent of medical knowledge adds unnecessarily to these fears.

Poliomyelitis is a disease of virus etiology. That fact has been definitely established, and the earlier theories concerning streptococci or intracellular globoid bodies have been discarded. The proof of etiology consists not only in the isolation of virus from human cases and transfer of the disease to laboratory animals, but in the occurrence of accidental human infections. Twelve children developed poliomyelitis after inoculation with Kolmer or Brodie vaccines, made of poliomyelitis virus suspensions that were insufficiently inactivated[2].

However, it must be emphasized that although the primary causative agent is a virus, there may be accessory etiological factors which facilitate invasion of the central nervous system. It is also known that there are several strains of the virus which differ in antigenic characteristics, as do the various strains or types of influenza virus, pneumococci or streptococci. The extent and importance of these antigenic differences have not yet been fully determined.

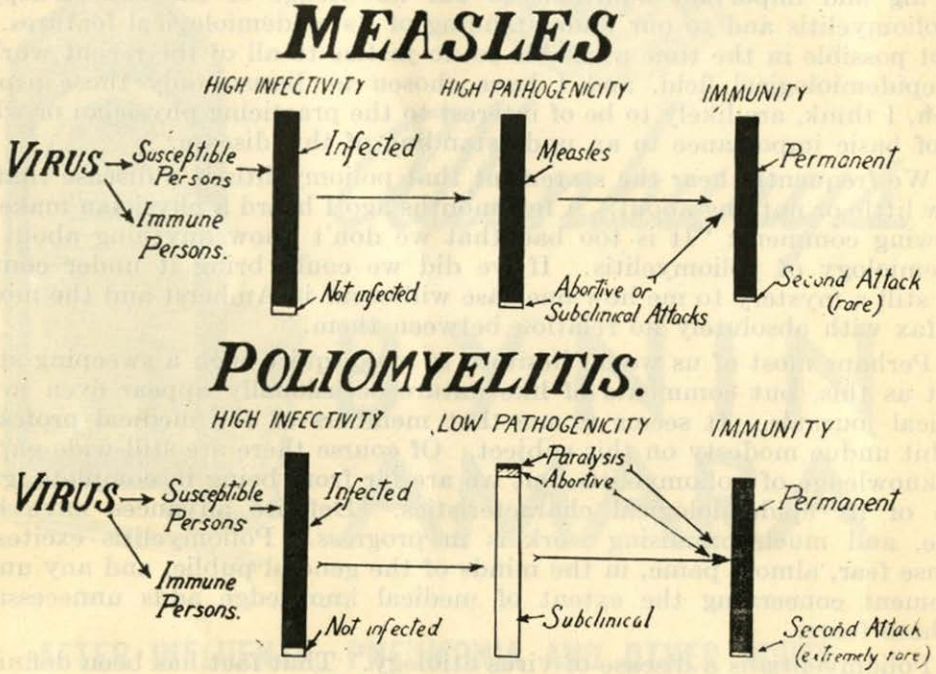
Poliomyelitis virus possesses three characteristics that are of major importance in determining the epidemiological picture, namely, high infectivity, low pathogenicity and lasting immune stimulus. An appreciation of these characteristics is fundamental to an understanding of the disease. A com-

*Paper presented at the joint meeting of the Dalhousie Refresher Course and The Medical Society of Nova Scotia, Halifax, October 6, 1947.

parison is made in Figure I between measles virus, which is of high infectivity and high pathogenicity, and poliomyelitis virus, of high infectivity but low pathogenicity.

FIGURE I

COMPARISON OF THE VIRUSES OF MEASLES AND POLIOMYELITIS WITH RESPECT TO INFECTIVITY, PATHOGENICITY AND IMMUNE STIMULUS

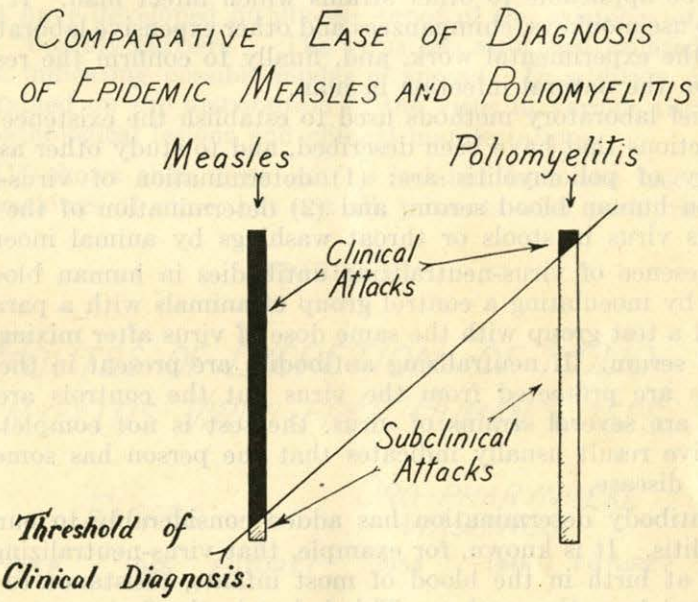


It is a well-known fact that, if a group of children who have not had measles come in contact with an infectious case, almost all of them become infected with the virus. Very few escape. There is good evidence that poliomyelitis is almost equally infectious. Both viruses have a high infectivity or invasive power in man. But here the similarity ends. Almost every child infected with measles virus develops a full-blown clinically recognizable attack of measles. There are only a few mild, abortive or atypical cases. The virus has a high disease-producing power or pathogenicity, as well as a high invasive power or infectivity. On the other hand, very few of the children who are infected with poliomyelitis virus develop paralytic poliomyelitis. A few others experience milder symptoms that may lead to a diagnosis of abortive poliomyelitis, especially if there are a few paralytic cases in the district at that time to arouse the doctor's suspicions. But most of the infected children will experience only mild symptoms or none at all. Yet, almost all of them, not matter how mild the attack, will respond with an immunity reaction that will protect them for life, just as occurs with measles. It has been known for many years that a few cases of poliomyelitis were of the mild or abortive type, but it is only recently that laboratory and epidemiological investigations have shown how frequent the occurrence of subclinical attacks is. If a group of one hundred susceptible children are exposed to

poliomyelitis infection, it is estimated that on the average only one will develop paralytic poliomyelitis. From two to eight others may be diagnosed as abortive cases. But over ninety will have an unrecognized immunizing experience with the virus during which they will have no typical symptoms, if any at all, but will probably be capable of spreading the infection to others. It has frequently been said that poliomyelitis rarely attacks more than one member of a household. As a matter of fact, it rarely leaves a single susceptible member uninfected, although it is unusual for more than one to develop paralysis.

Figure II indicates that poliomyelitis is for the most part a submerged epidemic, which is below the level of clinical recognition but which may be just as extensive as an epidemic of measles.

FIGURE II



If one thinks of the infection in this way, it is easy to understand the scattered geographic distribution of the paralytic cases. One may occur in Amherst and the next in Halifax with no traceable connection between them, but there may have been ninety or more intermediate links in the chain of infection. It is also easier to understand the periodicity of the epidemics. For example, there were 102 cases of paralytic poliomyelitis in Prince Edward Island during the summer of 1946[9]. If the average figure of 90 or more hidden infections for each paralytic case held true in that area, the epidemic must have been of staggering proportions. Approximately 9,000 persons probably acquired an infection with the virus, helped to spread it, and developed an immunity to it. This figure is only an estimate; there may have been as few as 5,000 or as many as 15,000. In any event the number would be large enough to explain why epidemics tend to die out and not recur in the same area until a new "crop" of susceptible children has grown up. Of course the suscept-

ible population is never completely involved in any epidemic, and isolated cases will occur in the inter-epidemic periods.

Man is the only host known to be subject to natural infection with the poliomyelitis virus. Although the lower primates can be infected by artificial methods and will develop a fairly typical paralytic poliomyelitis, the disease apparently does not occur among them in their natural state. Man is therefore the only known source of infection to others through paralytic cases, abortive cases, healthy carriers or convalescent carriers. It has been reported that the virus has been isolated occasionally from the house fly[8] and the stable fly[1], but there is no evidence that it multiplies in either of these insects. They may be mechanical vectors, but even in this respect their importance is not clear[14]. A few strains of the virus have been adapted by experimental methods to infect the mouse and cotton rat. These laboratory animals are cheaper and more plentiful than monkeys for experimental work, but, unfortunately, data obtained by using such specially adapted strains of virus may not always be applicable to other strains which infect man. It is therefore necessary to use monkeys, chimpanzees and other expensive laboratory animals in most of the experimental work, and, finally to confirm the results by observations on the natural infection in man.

The chief laboratory methods used to establish the existence of the subclinical infections that have been described, and to study other aspects of the epidemiology of poliomyelitis are: (1) determination of virus-neutralizing antibodies in human blood serum, and (2) determination of the presence of poliomyelitis virus in stools or throat washings by animal inoculation.

The presence of virus-neutralizing antibodies in human blood serum is determined by inoculating a control group of animals with a paralyzing dose of virus and a test group with the same dose of virus after mixing it with the specimen of serum. If neutralizing antibodies are present in the serum, the test animals are protected from the virus but the controls are paralyzed. Since there are several strains of virus, the test is not completely specific, but a positive result usually indicates that the person has some protection against the disease.

This antibody determination has added considerably to our knowledge of poliomyelitis. It is known, for example, that virus-neutralizing antibodies are present at birth in the blood of most infants, resistance to the disease being acquired from the mother. This is lost in the first year or two of life. The percentage of persons with antibodies in the blood then increases rapidly with age until in early adult life 75 to 80 per cent of people have virus-neutralizing antibodies against poliomyelitis[14]. The number of clinically recognized cases of the disease is far too small to explain such a high incidence of immunity, but it can be accounted for by the large number of subclinical attacks. The typical age distribution of paralytic cases is also explained by the presence or absence of virus-neutralizing antibodies in the various age groups. The incidence of paralytic attacks is highest in young children, 80 per cent of patients being under 10 years. The disease becomes progressively less common in older children and adults, but is not at all rare. The percentage of older children and adults showing virus-neutralizing antibodies is usually lower in rural populations than in urban. This is often parallelled by the occurrence of more paralytic poliomyelitis in adults in the country than in the city, although this has not been true of all epidemics in recent years[14]. Opportun-

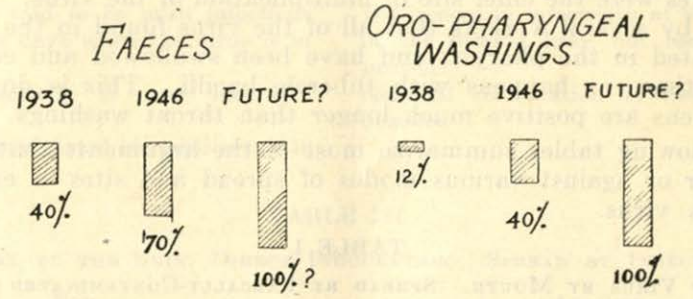
ity for contact with other people is usually less in the country, and the first immunizing experience with poliomyelitis virus tends to occur later in life.

The other important laboratory procedure used in epidemiological studies is determination of the presence of poliomyelitis virus in faeces or in pharyngeal secretions. Investigations were carried out on this subject as long ago as 1913, but most of the early work is discounted because diagnostic criteria were not very strict. The determination is made by treating the specimen with ether to kill the bacteria and injecting the suspension into the brains of monkeys. Poliomyelitis virus is not destroyed by ether, and if present, will cause paralysis in some or all of the inoculated animals. To obtain trustworthy results one must use four to six monkeys for a single specimen, each animal costing twenty-five to fifty dollars. The cost is therefore prohibitive for routine diagnostic use. However, experimental studies have yielded valuable data. It has been shown that the virus is present in the faeces or pharyngeal secretions of healthy persons who have had contact with paralytic poliomyelitis, but have developed no illness—proof of the existence of subclinical infections or "healthy carriers." The virus has also been isolated from sewage and flies, indicating possible modes of spread. In addition, information has been obtained on the distribution of the virus in various tissues and its relation to pathological lesions and clinical manifestations.

Figure III shows that virus can be isolated from the faeces and oropharyngeal washings of some, but not all, patients with paralytic poliomyelitis.

FIGURE III

ROUTES OF EXCRETION OF POLIOMYELITIS VIRUS FROM HUMAN CASES.



Because it has been possible to isolate the virus more often from faeces than from throat-washings, many writers have assumed that the danger of spread from faecal-contamination of food or water was greater than the danger of droplet infection from the nose or throat. This may not be a valid assumption. It overlooks the fact that a small amount of virus in the throat might be more widely disseminated into the environment than a larger quantity in the intestines. In addition, improved laboratory methods in recent years have resulted in more virus isolations both from the pharynx and the bowel than were possible a few years ago. If this trend continues, the apparent

difference may disappear, as suggested in Figure III. It has been reported by Toomey and Takacs[8] that certain toxic products of bacterial origin tend to reduce the resistance of the experimental animal to paralytic poliomyelitis. It is conceivable that the isolation of virus from stool suspensions may be facilitated by the presence of toxins from enteric bacteria, which make it more likely that the virus will produce recognizable disease when injected into monkeys. The greater number of successful isolations of virus from faeces than from throat washings may result from the presence of this accessory factor and cannot be taken as proof that there is more virus present in faeces. In this regard it has also been suggested by some writers that the gastro-intestinal infections which are often prevalent during the poliomyelitis season may act as an accessory factor in breaking down human defences against intra-neural invasion by the virus, but this is pure speculation at present.

Most studies on the isolation of virus show that it persists for two or three weeks in the faeces but for only two or three days in the pharynx. Horstman et al[6] showed that 61 per cent of poliomyelitis patients excreted virus during the first two weeks, 50 per cent during the third and fourth weeks, 27 per cent in the fifth and sixth weeks and 12.5 per cent in the seventh and eighth. However, there is at least one case on record where stool specimens were reported positive for 123 days[12], and another was reported in 1913 with positive pharyngeal secretions for four months.[13] Such convalescent carriers may explain some of the sporadic cases which occur throughout the winter and in other inter-epidemic periods.

Virus grows and multiplies only intra-cellularly. We do not know whether the poliomyelitis virus found in faeces comes from the intestinal mucosa, lymphoid tissue or lymphocytes in the lumen. Or it may come from the nerve cells of Meissner's and Auerbach's plexuses, being excreted from them into the lumen as rabies virus is excreted into the saliva. A lymphoid hyperplasia occurs in poliomyelitis, and the virus has occasionally been isolated from tonsillar tissue or Peyer's patches, but not as often as might be expected if these tissues were the chief site of multiplication of the virus. It has been pointed out by several workers that all of the virus found in the faeces may have originated in the pharynx and have been swallowed and concentrated in the intestines, as happens with tubercle bacilli. This is doubted since stool specimens are positive much longer than throat washings.

The following tables summarize most of the arguments that have been advanced for or against various modes of spread and sites of entry of the poliomyelitis virus.

TABLE I

ENTRY OF VIRUS BY MOUTH. SPREAD BY FAECALLY-CONTAMINATED WATER, MILK OR OTHER FOODS

FOR	AGAINST
Virus has been isolated from stools of cases and carriers, from sewage and flies.	Tubercle bacilli are present in stools and sewage, but are not important in spread.
More virus is found in faeces than in throat-washings.	Differences in isolation may be due solely to technical procedures, as described above.
A few epidemics have followed sewage-contaminated rivers.	Most epidemics follow normal routes of travel, not necessarily rivers.
A few epidemics have proven to be milk-borne.	Most epidemics cannot be related to food or water.

Seasonal distribution is the same as that of typhoid and other gastrointestinal infections.
Some experimental animals can be infected by the gastro-intestinal route.
Family epidemics are often common-source. resembling food-borne type.

Seasonal distribution of various air-borne infections differ greatly, as shown in Figure IV.

Poliomyelitis incidence is usually the same in areas with good and poor sanitation.
The incidence is no higher where flies are numerous.
Less poliomyelitis occurs in the tropics, where gastro-intestinal infections are more common.
Poliomyelitis has shown no decrease over the last 30 or 40 years with improvement in sanitation.
Age of infection is usually later in the country than city, although sanitation and flies are often worse.

TABLE II

ENTRY OF VIRUS BY THE UPPER RESPIRATORY TRACT. SPREAD BY DUST OR DROPLET NUCLEI CONTAMINATED BY NASO-PHARYNGEAL SECRETIONS OF A CASE OR CARRIER. INFECTED MATERIAL MAY THEN BE SWALLOWED.

FOR	AGAINST
Virus is present in pharyngeal washings.	Stools contain virus more often than pharyngeal washings.
Virus has been found in the tonsils in a few fatal cases.	Virus in tonsils might have been swallowed or inhaled.
The disease is said to be most infectious in the first few days when the virus is in the pharynx.	Differences in infectivity at various stages of the disease have not been very well proven.
The high infectivity is more like air-borne diseases.	Seasonal distribution is like filth-borne diseases.
Occasionally very casual contacts result in infection.	

TABLE III

ENTRY BY THE SKIN, DIRECT INOCULATION. SPREAD BY INSECTS.

FOR	AGAINST
Virus has been isolated in flies.	There is no evidence that virus multiplies in insects. Probably they are only mechanical vectors.
Monkeys can be infected by cutaneous injection.	Virus is not present in human blood, and there is no source for infection of biting or sucking insects.
Twelve human cases resulted from incompletely inactivated poliomyelitis vaccine given subcutaneously.	

TABLE IV

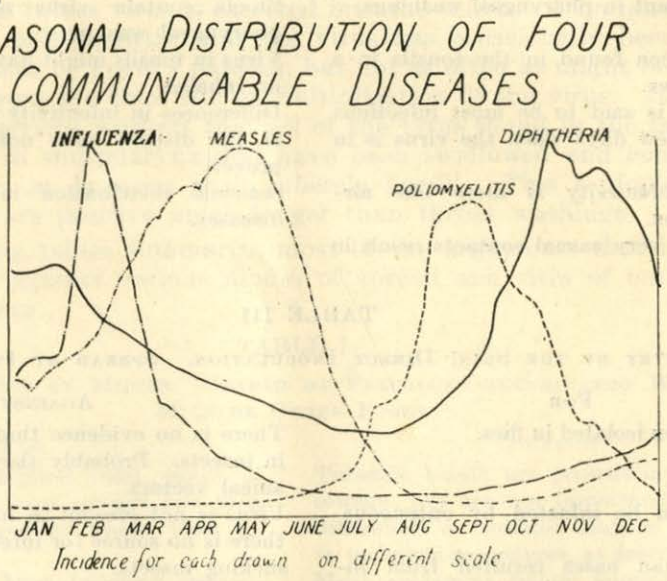
ENTRY BY NASAL MUCOSA, OLFACTORY NERVES, BULB AND TRACT

FOR	AGAINST
Intranasal virus results in poliomyelitis in 90 to 95% of rhesus monkeys.	
Typical poliomyelitis lesions are found in the olfactory tract of these monkeys.	Poliomyelitis lesions are rarely, if ever, found in the olfactory tract in humans.
Cutting the olfactory tract or zinc sulphate nasal spray prevents the disease in rhesus monkey.	Zinc sulphate spray was a complete failure in human experiments.

Ten years ago the final answer seemed to have been obtained on the mode of infection and of control, when the entry of virus by the olfactory nerves was established in the rhesus monkey. There were no flaws in this experimental work. The rhesus monkey can be infected by that route, and can be protected by zinc sulphate nasal spray. The error lay in the assumption that the virus behaved in the same way in monkey and man, and it does not.

At the present time there seems to be no good evidence in favor of an insect vector, but one still cannot rule it out with absolute certainty. The chief argument is whether the disease is air-borne or filth-borne. The seasonal distribution has always been quoted as one of the strongest pieces of evidence for the latter. In Figure IV a comparison is made of the seasonal distribution of three air-borne diseases, influenza, measles and diphtheria, and that of poliomyelitis.

FIGURE IV



When three infections that are usually air-borne can differ so widely from each other, there seems little reason to exclude the possibility that polio-

myelitis may also be in this class. It differs less from diphtheria in seasonal distribution than the latter does from measles.

We do not know, then, which mode of excretion of the virus is the more dangerous, intestinal or pharyngeal. Both can occur, and it seems logical to suggest that whatever precautions can be taken against both air-spread and faecal-spread should be employed. The perennial argument as to which is more important seems somewhat unprofitable. Other diseases, such as scarlet fever, may be air-borne in one epidemic, milk-borne in another, or spread by both agencies in the same epidemic. Poliomyelitis may also have more than one important mode of spread. One of these may eventually prove to be more important than the other as a source of epidemics, but both must be considered by the practicing physician and health officer in applying control measures in the individual case. Stools and pharyngeal secretions must both be treated as potentially infectious agents.

It has already been stated that poliomyelitis virus has a very widespread distribution in the population during an epidemic. In most persons it seems to be little more than a harmless temporary parasite, but in about one per cent of those whose gastro-intestinal tract is invaded, the virus gets into the central nervous system and produces serious lesions. The epidemiological picture would not be complete even if we possessed full knowledge of the modes of spread of the virus from person to person. Consideration must also be given to factors which permit invasion of the central nervous system of the new host, or which favor multiplication of the virus within the neurones to a level at which it can produce serious damage.

Howe and Bodian[7] have shown that monkeys can be readily infected by immersing the cut end of a nerve in a suspension of poliomyelitis virus. They have demonstrated that the virus travels up the nerve within the axis cylinder itself. It seems logical to suppose that any superficial lesion of the skin or mucous membrane in which nerve fibres are exposed might serve as the portal of entry, if the area became contaminated by poliomyelitis virus. Since the virus can be isolated from the pharynx of healthy carriers as well as 40 per cent of persons with paralytic poliomyelitis, and has been found in tonsillar tissue on post-mortem examination of a few fatal cases, the function of the tonsil and the effect of tonsillectomy are worthy of some consideration.

Unfortunately it is not possible to obtain clear-cut answers from the medical literature on all aspects of the relationship between tonsillectomy and poliomyelitis. Most published reports include data on the presence or absence of tonsils in patients with poliomyelitis, but give no information on a control group of children for comparison. It is not surprising that such a misuse of statistics results in contradictory conclusions. Pedersen[15] recently reviewed 30 reports on poliomyelitis following within 2 months after tonsillectomy. A total of 314 cases were included in his review, and he reported an additional 11 cases himself. He concluded from his own study that the incidence of poliomyelitis following recent tonsillectomy was "not greatly out of proportion to the ratio of disease to the general population during an epidemic year." A close scrutiny of his report reveals that he drew this conclusion by comparing two ratios that were not comparable. His own data therefore fail either to confirm or deny his conclusion.

Fischer and his associates[4] have presented the most conclusive evidence on this matter. Following the 1937 epidemic in Toronto they made

a comparison of the incidence of poliomyelitis in (a) children between 3 and 12 years who had had a tonsillectomy within one month before the epidemic and (b) the remaining children in Toronto schools between 3 and 12 years who had been operated on at an earlier period or who still retained their tonsils. Each group had equal opportunity for contact during the epidemic. Table V summarizes a portion of Fischer's data.

TABLE V

INCIDENCE OF POLIOMYELITIS IN RECENTLY TONSILLECTOMIZED CHILDREN COMPARED WITH THOSE NOT RECENTLY TONSILLECTOMIZED OR WITH TONSILS PRESENT

		Estimated No. of Children Exposed	Poliomyelitis No. of Cases	Poliomyelitis Rate Per 1000
August.	Recent Tonsillectomy.....	654	3	4.6
"	No recent Tonsillectomy.....	84,058	228	2.7
Sept.	Recent Tonsillectomy.....	429	5	11.7
"	No recent Tonsillectomy.....	84,261	225	2.7

In August the observed rate in the recently tonsillectomized children was 1.7 times higher than in the controls. However, the numbers are small and do not show a large enough difference to be considered statistically significant. For the month of September the rate was significantly higher in the recently tonsillectomized children, being more than four times as high as in the controls. Although the number of children with recent tonsillectomy was small, the difference was greater than could be accounted for by chance alone.

More striking than the total increase in poliomyelitis is the proportion of bulbar or spino-bulbar cases in tonsillectomized children. Of the 325 cases following within 60 days after tonsillectomy that were reviewed or reported by Pedersen[15], 195 were bulbar or spino-bulbar (60%). Top[19] reports that cases with involvement of the bulbar centres comprise 15 to 30 per cent of the total, and in one series of 498 cases reported in his book (Appendix, Table XV-A) 23 per cent were of this type. It would appear, then, from a review of the literature that, if a child develops poliomyelitis within a month or two after tonsillectomy, there is a greater likelihood that it will be of the bulbar type and hence a greater chance that it will terminate fatally. There might be a fallacy in drawing such a conclusion from these data alone, especially if medical writers had a tendency to report more serious cases than uncomplicated ones in the medical journals—a possibility which should not be ignored, but usually is. However, the data of Fischer et al[4] tend to confirm this observation. Of the 507 diagnosed cases of poliomyelitis in Toronto in 1937[4], 267 children had tonsils and adenoids present and 231 had been subjected to a tonsillectomy at some previous date. Information was missing on only 9 of the children. The percentage of bulbar cases was significantly higher in the tonsillectomized than in the non-tonsillectomized group, 18.6 and 7.5 per cent, respectively. If non-paralytic cases are excluded, because criteria for diagnosis of abortive cases are difficult to standardize, the proportion of bulbar cases in tonsillectomized and non-tonsillectomized children would be 34.9 and 15.8 per cent of the paralytic cases, respectively. Table VI summarizes a part of Fischer's data on bulbar poliomyelitis.

TABLE VI

INCIDENCE OF BULBAR POLIOMYELITIS IN RECENTLY TONSILLECTOMIZED CHILDREN COMPARED WITH THOSE NOT RECENTLY TONSILLECTOMIZED OR WITH TONSILS PRESENT

		Estimated No. of Children Exposed	Bulbar Poliomyelitis No. of Cases	Bulbar Poliomyelitis Rate Per 100
August	Recent Tonsillectomy.....	654	2	3.1
"	No recent Tonsillectomy.....	84,058	27	0.3
Sept.	Recent Tonsillectomy.....	429	2	4.7
"	No recent Tonsillectomy.....	84,261	28	0.3

Bulbar involvement was ten to fifteen times more common in recently tonsillectomized children. Although these figures are small, the difference is much greater than could occur from chance alone, and is highly significant. These writers showed that the increase in total incidence shown in Table V was wholly accounted for by this increase in bulbar cases.

The most striking example of the relation between bulbar poliomyelitis and tonsillectomy was that of the "K" family in Akron, Ohio[5]. Five children of a family of six had a tonsillectomy on August 22. Between September 3rd and 5th all five developed poliomyelitis. All attacks were of the bulbar type, and three of the children died. The one member of the family who did not have a tonsillectomy was found to be carrying virus in his stools, but he developed no illness. Seven cousins who were contacts also had positive stools, but none became ill. There was no epidemic of paralytic poliomyelitis in the area at that time, but it is obvious from the virus studies that unrecognized subclinical attacks were occurring, and tonsillectomy apparently precipitated a clinical attack in the five children.

From all this evidence there seems to be little doubt that, if a tonsillectomized child is infected with poliomyelitis virus, the attack is more likely to be of the bulbar type. It seems to have been concluded by all writers that recent tonsillectomies (i.e. within 1 or 2 months) were dangerous, but that an earlier operation had no significance. It has been suggested that the exposed nerve endings of the healing lesion in the throat served as the portal of entry. As a matter of fact, the evidence is not at all conclusive as to whether it is simply absence of the tonsils or a recent tonsillectomy that is important, or possibly both. Fischer et al[4] showed that the risk of blubar poliomyelitis was greater for the more recently tonsillectomized than for those operated on some months before infection. However, their data showed that there was a significantly higher proportion of bulbar cases even in those who had been tonsillectomized six months or more before infection than there was in the children who had retained their tonsils. It appears possible that the tonsils play some role in preventing entry of the virus, and their absence permits invasion of the bulbar centres. However, the danger seems considerably greater within one month after the tonsillectomy than it is during later periods. Possibly the presence of a partially healed lesion in the throat is a factor. This whole subject deserves further well controlled study.

The medical profession has not yet reached full agreement on the practical

procedure to follow as a result of the demonstrated relationship between bulbar poliomyelitis and tonsillectomy. A considerable number of surgeons do not perform tonsillectomies during the poliomyelitis season of July to October, or, at least, limit the operation to the small number of persons whose infected tonsils might constitute an immediate and serious hazard to health if there were any delay. Many others argue that there is no good evidence that the risk of poliomyelitis is significantly increased, and they therefore have made no change in their procedure. It is quite true that the statistics on total incidence contained in most reports are not above criticism but almost every writer shows at least a small trend toward a higher incidence of poliomyelitis in tonsillectomized children. The greater weight of evidence seems to be on that side. But even if there were no actual increase in total incidence, there is undoubtedly a greatly increased risk that an attack will involve the bulbar centres, if a child does develop poliomyelitis after a tonsillectomy. And bulbar poliomyelitis has a much higher mortality rate than the spinal type. The surgeon must balance this hazard of bulbar poliomyelitis against the risk of leaving the diseased tonsils for several months. Whatever the decision may be in an individual case there seems to be enough evidence to justify the school physician in condemning the usual parental custom of delaying tonsillectomy until the summer holidays.

Other writers have presented evidence purporting to show that there was a higher incidence of poliomyelitis in children who had exposed dental pulp. It was suggested that the virus might gain access to exposed nerve fibres in the tooth and extend up the nerve to the central nervous system. Recently Finn, Korns and Bahlke[3] have reviewed the literature on this subject and criticized the methods of selecting control children for comparison. They considered it possible that the control children, who did not have exposed dental pulp, had less opportunity for contact with poliomyelitis infection, and that this may have explained the lower incidence of disease. Finn and his associates conducted a well-organized study, using children in the same family as controls and they found no increase in the incidence of poliomyelitis in children with exposed dental pulp.

Although trauma to the pharynx may possibly be one means by which the likelihood of intra-neural invasion is enhanced, it is probably not the only one, or even the most important. Certain experimental work has suggested that intestinal trauma may be important[18]. The possibility that bacterial toxins may also facilitate intra-neural invasion has been mentioned already. Paralytic poliomyelitis is practically unknown in the tropics, but virus-neutralizing antibodies are found in 75 to 80 per cent of the adult population. This indicates that subclinical infections occur there as often as in the temperate zones. It has been suggested that this might be explained if cold predisposed to intra-neural invasion. If this were true, one wonders why poliomyelitis is for the most part a summer disease. Yet, several experienced clinicians have been impressed by the frequency with which severely paralyzed persons give a history of chilling or exposure. Top and Vaughan have also reported a correlation between severe paralysis and a history of recent and unusual stress, such as participation in a strenuous athletic contest[20]. Some of their data are reproduced in Table VII.

TABLE VII

HISTORY OF STRESS OR ILLNESS WITHIN 4 TO 14 DAYS OF THE ONSET OF POLIOMYELITIS, DETROIT, 1939

		Total	History of Stress or Illness	
			No.	Per Cent
All Poliomyelitis cases		521	106	20.3
Fatal cases		23	8	34.8
Controls in same family		497	27	5.4
Controls in 3 Other	1	129	7	5.4
Districts of City	2	167	15	9.0
	3	141	8	5.7

A higher percentage of the poliomyelitis patients gave a history of recent stress or illness than did any of the control groups. The authors point out that too much emphasis cannot be placed on data of this type, because parents of a patient with poliomyelitis are far more likely to recall an illness or injury just prior to the onset of the disease than are parents of children used as controls. Nevertheless, Levinson et al[11] have shown a higher incidence of paralytic poliomyelitis and more extensive paralysis in monkeys subjected to exhausting exercise during the incubation period than in non-exercised animals, and a higher incidence also in animals subjected to chilling than in controls. Until we have more definite information it would appear that limitation of activity might be a wise precaution for any child who develops a minor illness during a poliomyelitis epidemic, and possibly for children who are contacts of known cases and may already have a subclinical infection.

Sandler[17] has reported that rabbits can be infected with poliomyelitis virus if they are first rendered hypoglycemic with insulin. Ordinarily these animals are completely resistant to poliomyelitis. This observation requires further study, as does the possibility that hypoglycemia may be a factor in producing a higher incidence of paralytic poliomyelitis in persons exposed to unusual stress.

There is a consistently higher incidence of poliomyelitis in males than in females in a ratio of 3 to 2. It is also reported that pregnancy enhances the risk of poliomyelitis in females. Hormonal factors may therefore be of importance, but their mode of action has not been demonstrated.

Howe and Bodian[7] have shown that it is possible to alter the susceptibility of nerve cells to virus invasion. Recent injury to a nerve fibre allows the virus to travel up the axone more readily and invade the nerve cell. This might be the mechanism in recent tonsillectomy. But repeated trauma to a nerve fibre results in chromatolysis within the nerve cells, which then become resistant to the entry or multiplication of the virus.

The above paragraphs are not by any means a complete review of factors affecting intra-neural invasion, but they suggest the extensive nature of the investigations. It seems probable that purely metabolic, hormonal or other physiological factors, regardless of the level of immune bodies, may have an important influence on facilitating or retarding intra-neural invasion or multiplication of this virus.

Finally, the practical question must be asked: "Has any of the recent work indicated a sure method for preventing the spread of poliomyelitis?"

As you know, the answer is essentially in the negative. We can plan an attack in a more logical manner, but the results have not been encouraging. Even if we knew all of the missing facts concerning the epidemiology of the diserve, it does not necessarily follow that we could prevent it. We know measles very well but cannot control it yet. Methods for controlling the spread of any communicable disease may be grouped into three classes:

(1) Procedures which attempt to isolate the source of infection and prevent the infectious agent from contaminating the external environment or reaching a new host by direct contact.

(2) Procedures that attempt to destroy the agent in the external environment.

(3) Procedures designed to increase the resistance of susceptible persons so that no disease will result even if contact with the infectious agent is unavoidable.

Barriers around the source of infection are erected by such procedures as isolation of cases, quarantine of known contacts, control of known carriers, reduction of person-to-person contact by closing places of meeting, disinfection of excreta and of contaminated articles, etc. Any or all of these methods may logically be applied to poliomyelitis control, but to date they have not achieved any significant results. The reason is obvious. There are over ninety subclinical cases that are uncontrolled sources of infection for every one source that is fully controlled. The family physician and health officer are probably justified in handling each diagnosed case and the contacts in such a way as to minimize danger of air-borne or faecal-borne spread from this known source. They may thus protect some persons in the community. However, they cannot expect to stop an epidemic by such procedures, when there are so many unrecognized carriers spreading the virus. Attempts to limit person-to-person contact are also logical but not very successful, except in a few individual families. Perhaps they are usually instituted too late. Schools are rarely closed until several cases have occurred in an area. But "several cases" mean several hundred other subclinical sources of infection, and the epidemic is well established by that time. It is possible that results might be achieved by early action, when an epidemic seems to be moving toward an area where there has not been an outbreak for several years, but this is problematical. Attempts to limit person-to-person contact in children usually result in little more than a change from contact in the school to that of the streets, playground or theatre, and in adults such limitation is not economically feasible.

Procedures designed to destroy the virus in transit come under the heading of environmental sanitation. Chlorination of water, pasteurization of milk, fly control, sanitary sewage disposal, swimming pool sanitation, etc., are methods aimed at preventing filth-borne diseases. Since at least a part of the spread of poliomyelitis is probably by faecal contamination, these methods are important. Once again, however, results have been disappointing, probably because there are other modes of spread as well. The spraying of large areas with DDT to destroy flies has not shown any appreciable effects. This may be due to the fact that such procedures have usually been introduced as a result of public hysteria when the epidemic has already been well

established. Such control methods, if used at all, must be introduced early. As a matter of fact there is little evidence that flies are of much importance in the spread of this infection, and the money spent on DDT spraying from aircraft could probably be put to much better use. The effectiveness of chlorine in destroying the virus in water requires further study. The Lansing strain of poliomyelitis virus is inactivated in 10 minutes by as little as 0.05 parts per million of free chlorine if the water has a pH of 6.85 to 7.4 [10] However, in a more alkaline water the virucidal action is much less or absent in one hour, even with higher concentrations of chlorine than are ordinarily used in drinking water or in swimming pools. If, as seems probable, air-spread is important as well as faecal-spread, the above methods alone could not be sufficient to control the spread of poliomyelitis. Unfortunately sanitation of the air that we breathe is still in the experimental stage. Ultra-violet irradiation of school rooms, and prophylene or triethelene glycol vapor have given promising results in the control of some infections. However, there is no evidence yet as to their effect on poliomyelitis virus.

The third group of control procedures are those designed to raise the resistance of the susceptible persons to such a level that they will not be infected even if they do have contact with the virus. To date there has been no immunizing agent or chemotherapeutic procedure that is of practical value against poliomyelitis. However, some of the research work on immunity is very promising, and may eventually lead to an effective method of control. The successful preparation of other virus vaccines in recent years adds further encouragement for the future.

The medical profession and the public are naturally anxious that some agent should be discovered to prevent poliomyelitis, and rightly so. However, it might be well, in conclusion, to consider poliomyelitis in its proper perspective in relation to other common communicable diseases for which we have effective preventive methods. Table VIII shows the average number of reported cases and deaths per year from whooping cough, diphtheria, measles and poliomyelitis, in Canada between 1941 and 1944.

TABLE VIII

NUMBER OF REPORTED CASES AND DEATHS FROM WHOOPING COUGH, DIPHTHERIA, MEASLES, AND POLIOMYELITIS IN CANADA—FIVE YEAR AVERAGE 1941-44

	Average No. of Reported Cases Per Year	Average No. of Deaths Per Year
Whooping Cough	17,272	475.6
Diphtheria	2,831	261.0
Measles	53,788	210.6
Poliomyelitis	762	48.8

It is permissible to include measles as a partially preventable disease since most deaths occur in children under two years and gamma globulin administered at the time of suspected contact will provide considerable protection. In spite of the fact that we have effective protective agents against whooping cough, diphtheria and measles, each of these cause 5 to 10 times the number of deaths per year that poliomyelitis does. But, it may be argued, poliomyelitis causes permanent disability in those who survive. But so may

complications of measles, whooping cough and diphtheria; deafness, chronic bronchitis and cardiac or renal complications are not too uncommon. Besides, less than 25 per cent of paralytic poliomyelitis results in permanent disability. Even if we assume that all the 762 reported cases shown in Table VIII were paralytic, which is a great over-estimate, the number with varying degrees of permanent disability would be less than one quarter of this or 190 per year. Add to this the 49 deaths, and the total of killed and disabled is still less than the number who die annually from either of the *preventable* diseases, diphtheria or whooping cough. Until an effective agent for poliomyelitis control becomes available, and even when it does, let us, therefore, keep our eyes on the more important targets.

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Chronic Constrictive Pericarditis (Pick's Disease)

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CHRONIC constrictive pericarditis consists of a chronic fibrosis or callous thickening of the wall of the pericardial sac which is so contracted that the normal diastolic filling of the heart is prevented. Not only does it present a typical clinical syndrome, but it shows a characteristic pathological picture and specific X-ray appearance.

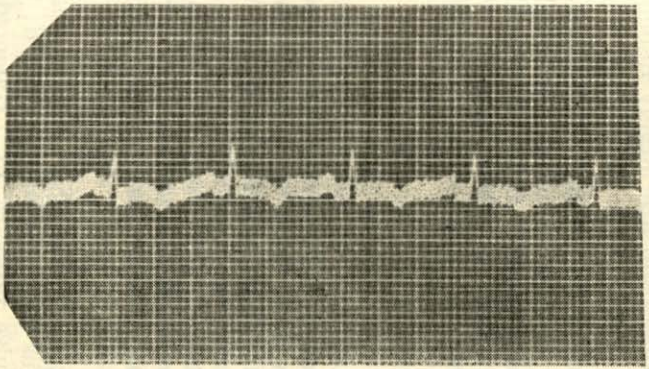
Historical—The condition was first clearly defined by Lower in 1669, who described the clinical features of a case and reported that, at autopsy, the pericardium was thick and callous. Pericardiectomy was first performed in 1910 by Hallopean and, since then, a number of other cases have been reported.

Etiology—This form of chronic pericarditis has a number of immediate or exciting causes. It has followed pneumonic and septic conditions and tuberculous pericarditis has been described frequently. The modern opinion is that rheumatic infection does not produce Pick's Disease. Subacute tuberculous infection is an important etiological factor. In many cases no definite etiological factor can be named. The majority of patients are in the second decade but it may occur at any age. Males are affected more frequently than females.

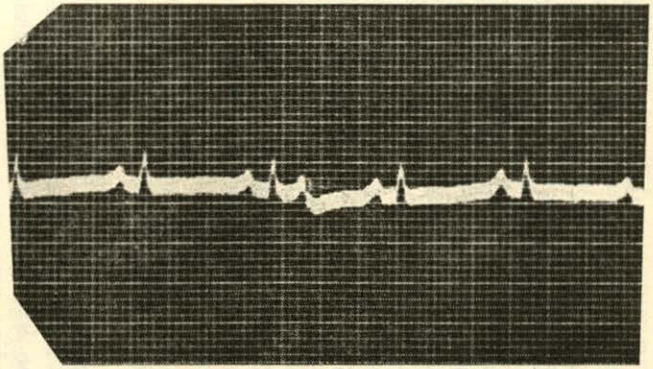
Pathology—The important changes are in the pericardium, but there may be changes in the surrounding tissues. The pericardium is thickened, sometimes extremely so and consists of dense white fibrous tissue in which there is very little elastic tissue. It varies from 1/16 to 1/4 of an inch in thickness. It may be general or localized to one area. This variability in distribution accounts for a satisfactory result in some cases and a disappointing outcome in others. It may be impossible to demonstrate any serosal cavity. Calcification is commonly present in the dense fibrous tissue and actual bone may be present. The contraction of the thickened tissues compresses the heart or the great vessels. Surrounding tissues may be affected by the same process and dense adhesions may be present between the pericardium and pleural or chest wall.

Pathological Physiology—The constriction of intra-pericardial structures is responsible for marked alterations in the circulation. Diastolic relaxation of the heart is prevented and there is some interference with systole—the result being that insufficient blood is taken into the heart thus reducing its output. The reduction in output is reflected by the low arterial pressure and raised venous pressure. There is an associated increased heart rate but, even so, the output per minute is diminished. The systolic blood pressure is usually low and diastolic high, giving a small pulse pressure. Obstruction to the circulation can be demonstrated by the retarded circulation time Vital capacity is sometimes greatly diminished due to collection of fluid in pleural cavities.

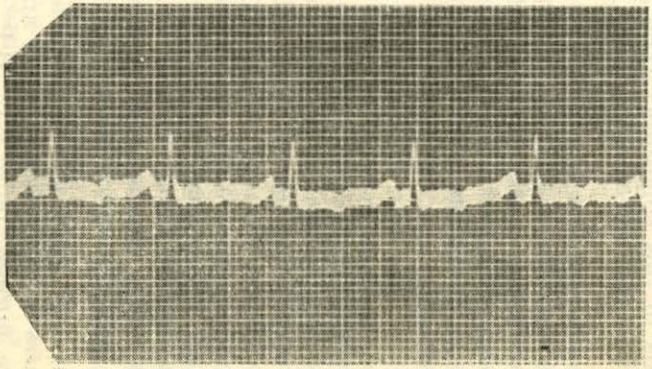
Symptomatology—Symptoms and signs depend on the circulatory changes just described—the only one invariably present is abdominal distension,



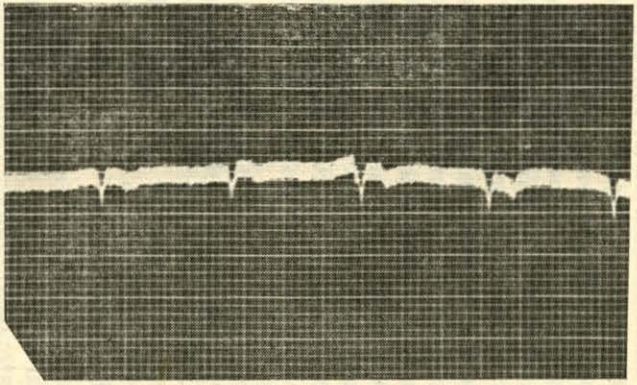
Electrocardiogram Lead I.—Nov. 19th., 1947.



Electrocardiogram Lead I.—Sept. 17th., 1947.



Electrocardiogram Lead II.—Nov. 19th., 1947.



Electrocardiogram Lead II.—Sept. 17th., 1947.

which is the characteristic feature of the disease. The abdominal distension is due to liver enlargement, ascites or both. Oedema of the feet and ankles, of abdominal and thoracic walls, of face and arms may be present. Cynaosis may be present—dyspnoea is a common symptom. Weakness and wasting are occasionally present. In general, pain, pyrexia and symptoms referable directly to the heart are absent. The characteristic signs are enlarged liver, with ascites, and distension of the veins of the neck in which visible pulsation may be present. Pleural effusions are usually present in the late stages of the disease. Associated with these signs are usually a low arterial pressure and low pulse pressure. The typical heart is small. Fluoroscopic examination may reveal almost no change in heart size from systole to diastole. In the electrocardiogram two abnormalities have been noted (a) marked dimunition in voltage. (b) flattening or inversion of T waves in lead 1 and 2.

Diagnosis—Determination of suitability for operative treatment is an important aspect of the diagnosis. The most serious contra-indicat on to operation is the presence of active infection, especially tuberculosis. This may be ascertained by evaluation of pyrexia, blood counts, blood sedimentation rates, reaction to tuberculin tests, examination of aspirated fluid, and other diagnostic aids.

Constrictive pericarditis must be distinguished from (a) other forms of pericarditis (b) from other conditions such as valvular disease (especially mitral stenosis), polyserositis, cirrhosis of the liver and, rarely, mediastinal tumor or aneurysm and oedema of extra cardiac cause.

Prognosis—Untreated patients with Pick's Disease may live for many years but are usually chronic invalids. Pericardiectomy has revolutionized the prognosis in this condition. The results of operation are largely dependent on the position and type of lesion.

Treatment—The treatment is surgical. The first essential, however, is the correct choice of the suitable type of case. It is futile to attempt to release the constriction in the presence of active tuberculosis. Since the condition is not supposed to be a result of rheumatic infection, those cases in which there is a strong rheumatic history should not be regarded as favorable for operation.

Case Report

A white female, age sixteen years, was admitted to the Victoria General Hospital on September 13, 1947, complaining of a fullness in her abdomen and never having menstruated. Her history went back six years when, at the age of ten, her doctor had aspirated fluid from her abdomen and later had done an exploratory laparotomy. A tentative diagnosis of tuberculous peritonitis was made. Following the operation, she noticed a mass in her upper abdomen which has persisted. She was advised to rest and, for the past six years, has not gone to school and has avoided all exertion. Family history was non-contributory.

Physical Examination—Patient appeared pale and there was a general coarseness of skin texture; neck and arm veins were noticeably dilated; temperature normal, pulse 85. B.P. 110/85, pulse pressure 25. Haemoglobin 70%. R. B. C. 3,500,000. W. B. C. 4,000 (Neutrophils 60%, lymphocytes

34%, monoeytes 6%). Examination of chest revealed dullness in mid-clavicular line up to fifth interspace on left and third interspace on right. Heart sounds were normal but percussion was unsatisfactory due to fluid in pleural space. Examination of abdomen revealed the liver to be enlarged to three inches below costal margin but smooth and regular. There was shifting dullness in both flanks. Lower extremities showed moderate oedema. X-ray of chest revealed fluid in pleural cavity commencing at third rib anteriorly on right and fourth rib on left. Pulmonary congestion was marked in both lungs. Fluoroscopic examination of heart showed that the excursion of chambers was not as full as usual. Several specimens of pleural fluid were taken, the average specific gravity of which was 1.010 (i.e. a transudate) and all cultures were negative for tubercle bacilli. Patch test was strongly positive, sputa were negative for tubercle bacilli. Blood cholesterol was 270 (N 150-250), Cholesterol Esters 91.8% (N 65-75%), Albumin: Globulin ratio (0.64/1) reversed. Venous pressure—upper right limb 230 mm H_2O (N 40-120). Circulation time (a) with decholin 30 secs. (N 10-18) (b) with ether 13 secs. (N $3\frac{1}{2}$-8).

Electrocardiogram was repeated five times with patient postured in various ways with following results:

1. Low voltage.
2. Inverted T_2, T_3, T_4 waves—flattened T_1.
3. Absence of normal variation of electrocardiograph tracings in various postures, indicating that heart is abnormally fixed.

Icterus index, Fouchet's test, Congo Red test, Mosenthal test, Hippuric Acid test, CO_2 combining power, prothrombin time, N.P.N., acid and alkaline phosphatase, haematocrit, blood sugar, and sedimentation rate were all done and found to be within normal limits.

In view of the above findings, a diagnosis of constrictive pericarditis was made and, after aspirating 1480 cc's of fluid from each pleural cavity, pericardiectomy was carried out on October 27, 1947, under sodium pentothal and endo-tracheal cyclopropane anaesthesia with intravenous novocaine. Operation report: "Curved incision over middle of sternum and along left costal margin, 3rd, 4th and 5th costal cartilages, short portion of corresponding ribs and about half of corresponding sternum were removed. Left and right pleura were separated from the pericardium, left pleura was inadvertently opened at a point where it was densely adherent to the pericardium. A vertical incision was made in the pericardium along the left cardiac margin from the great vessels to the diaphragm. There was no free pericardial space. Pericardium was dissected away from the myocardium over the entire anterior surface of the heart. At certain points the fibrous tissue was much denser than at others, there being a particularly tight constriction about the great vessels. The dissected portion was completely removed. 100,000 units of penicillin injected into area. The pleural cavity was closed with chromic catgut and incision closed. Left pleural cavity connected to pneumothorax machine by aspirating needle and patient placed in oxygen tent."

Pathological report revealed a simple chronic pericarditis with some fibrosis beween the adherent layers of pericardium. No evidence of tuberculosis. Convalescence was uneventful and patient was ready for discharge

three weeks following operation, at which time the following observations were made:

Patient was able to walk about freely without dyspnoea, veins of upper limb and neck were no longer distended, oedema of lower limbs was no longer present, venous pressure in upper right limb had dropped to normal and circulation time was within normal limits. B.P. is now 116/68 giving a pulse pressure of 48 in comparison with a pulse pressure of 25 previous to operation. Albumin: Globulin ratio has now become normal. Blood cholesterol has dropped to 128. Liver has receded in size, there is now only a small amount of fluid in pleural spaces and pulmonary congestion is much less marked. Heart now appears to be about normal in size and electrocardiogram shows marked increase in voltage, although T waves have remained inverted.

Summary and Discussion

1. Constrictive pericarditis has been discussed with case report.
2. The following is a summary of the pre and post-operative findings showing the change as a result of pericardectomy:

	Pre-operative	Post-operative (3 weeks)
Distension of veins of neck and arms	greatly distended	no distension
Venous pressure (right arm)	230 mm. H^2O (N 40-120)	52 mm. H^2O
Blood Pressure	S110-D85 P.P. 25	S116 D-68 P.P. 48
Oedema of lower limbs	Moderate	no oedema
Pleural effusion	moderate	greatly diminished
Enlargement of liver	3″ below costal margin	diminished
Excursion of heart chambers	diminished	normal
Blood cholesterol	270 (N 150-250)	128
Cholesterol Ester	91.8%	86%
Circulation time with Decholin	30 secs. (N 10-18)	16 secs.
Circulation time with Ether	13 secs. (N $3\frac{1}{2}$-8)	6 secs.
Albumin Globulin Ratio	reversed	normal
Electrocardiogram low voltage	present	normal
T waves	inverted L's 1, 2, 3, 4, flat in L1	remain inverted

3. The etiology in this case has not been proven but, with an old history of tuberculous peritonitis, the tubercle bacillus was almost certainly the etiological agent.
4. The post-operative progress in this patient to date gives an excellent prognosis.

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Mrs. Flannagan

Two years ago I had the pleasure of attending a lecture by the late Prof. MacNeal of the New York Post-Graduate Hospital on the subject of Acute Bacterial Endocarditis. The room lights dimmed and on the screen appeared the admission chart of Mrs. Flannagan, a young Navy Officer's wife, suffering from that dread and fatal disease. Mrs. F. was indeed a very sick girl and did not respond, as expected, to the usual supportive treatment. The screen showed her down-hill progress, mounting fever, rising sedimentation rate, positive blood cultures, signs pointing to embolic phenomena in *all* organs, convulsions, paresis, paralysis, to coma and impending death. No one case seemed to show so many of the signs and symptoms found in that disease. The organism was found to be succeptible to penicillin but, this being in the early days of the war as viewed from the United States standpoint, all available supplies of that wonder drug were tightly bound up with red tape and could only be unwrapped for use on service personnel.

By means not exactly ethical a small supply was rescued from the incinerator to which it was consigned as being not up to army standards. In desperation this was used and the immediate results were dramatic. High officials were contacted and all reasonable (and unreasonable) pressure brought to bear. The resulting refusals were in the main sympathetic. It made one rebel at the bureaucracy which was permitting this young woman to die.

The rejected supply was nearing exhaustion when Dr. MacNeal was called by a drug manufacturer who suggested that the Doctor might like to tour his plant and see how penicillin was being made. Dr. MacNeal replied that he was too busy at the moment to even consider it and his caller then asked if his secretary would like to visit the plant. This visit was immediately arranged and on its completion the young lady left the plant aware that her handbag had greatly increased in weight. Its contents, as you might guess, allowed us to see a further improvement in Mrs. Flannagan's progress as viewed on the screen. On another, and desperate, occasion a rather mysterious patient visited the doctor and did not wish to consult with him, merely asking to rest a few moments in his office. She "forgot" her parcels and again the screen registered favorable progress. By many such incidents and accidents a supply of penicillin was maintained. The progress was not smooth but eventually the screen recorded her discharge and cure. The lecturer held us all spellbound and there was a hearty cheer when the cure was reported.

Finally there appeared on the screen a photo of Mrs. Flannagan, a beautiful young lady, radiating good health. I leaned to the chap at my right and said "That girl must think a lot of Dr. MacNeal." He replied, "I know she does, she's my wife." At this moment the lights in the lecture hall came on and Dr. MacNeal said "Stand up Marion." Just two seats to my right stood Mrs. Flannagan, the first patient to recover from Acute Bacterial Endocarditis.

D. F. Macdonald
Yarmouth

Plan Industrial Conference on Alcoholism

The first Industrial Conference on Alcoholism will be held in the Morrison Hotel, Chicago, on Monday, March 15, 1948, it was announced to-day (Thursday, Dec. 18) by Dr. Anton J. Carlson, of the University of Chicago, chairman, and James H. Oughton, Jr., director of the Keeley Institute, Dwight, Ill., co-chairman.

Sponsored by the Chicago Committee on Alcoholism, the Conference has been designed to bring to the attention of industry leaders throughout the country facts pertaining to the problem of alcoholic employees and to discuss ways and means of overcoming the problem.

"Alcoholic employees now cost industry almost one billion dollars per year, according to one estimate," Dr. Carlson stated. "In most cases, when an employee is found to have alcoholic tendencies, he or she is usually fired. What industry does not realize is that they have invested time and money in these employees and that it is not necessary to fire them. If proper measures are taken, most of them can be rehabilitated and when this is accomplished, they make the best possible type of employee."

The Conference has been divided into three parts—morning session, luncheon, and afternoon session.

In the morning session, to be held in the Terrace Casino of the Morrison Hotel, the discussion will centre around "The Problem." Alcoholic definitions such as "What and who is an alcoholic" and the physiological and psychological aspects will be covered by one speaker. A second speaker will discuss "Alcoholism as a community problem," setting forth the social aspects, relationship of problems of alcoholism to family and community life. The role of public institutions, such as courts, hospitals, welfare agencies, jails, etc., will be reviewed briefly.

The morning session will conclude with two speakers covering the subject "Alcoholism in Industry." One will discuss "The problem of alcoholic employees" with emphasis on hazards, accidents, loss of work, time, etc. The second speaker will dwell on the all-important phase of the subject "The cost to industry."

A special luncheon is planned, to be held in the Mural Room of the Morrison, at which an outstanding authority on the subject will be the keynote speaker.

The afternoon session will cover the subject of "What to do about the problem."

Four speakers will discuss "What can be done for the individual alcoholic." Various types of individual treatment will be covered; the work of the Yale School of Alcohol Studies, the Keeley Institute treatment, and various types or rehabilitation centres will be outlined. The Alcoholic Anonymous program will also be covered.

The final subject "Responsibility for alleviation and prevention" will be covered by two speakers. One will talk on industry's part in the problem; the other on industry and the community.

Speakers will be prominent medical authorities who have specialized in the disease of alcoholism, and outstanding industry representatives who are conscious of the problem.

Reservations for attending the technical sessions and the luncheon may be made by writing Walter O. Cromwell, Vice-President, Chicago Committee on Alcoholism, 816 South Halsted Street, Chicago 7, Illinois.

Change Date of Industrial Conference on Alcoholism

The first Industrial Conference on Alcoholism, originally scheduled to be held on March 15, will now be held on Tuesday, March 23, it was announced today (Monday, Jan. 12) by Dr. Anton J. Carlson, of the University of Chicago, chairman, and James H. Oughton, Jr., of the Keeley Institute, Dwight, Ill., co-chairman.

Sponsored by the Chicago Committee on Alcoholism, the one-day conference will be held in the Terrace Casino of the Morrison hotel, Chicago.

At the same time, it was announced that Dr. John I. Norris of the Eastman Kodak company, Rochester, N. Y., would be the keynote speaker at the conference's luncheon to be held in the Mural room of the Morrison. Norris, a Fellow of the American Association of Industrial Physicians and Surgeons and of the American Medical Association, has been doing considerable work on the subject of alcoholism through the Rochester Committee for Education on Alcoholism. His talk will be highlighted with the latest information available to industry.

The conference has been designed to bring to the attention of industry leaders throughout the country facts pertaining to the problem of alcoholic employees and to discuss ways and means of overcoming the problem.

First International Poliomyelitis Conference.

The National Foundation for Infantile Paralysis announced recently it would sponsor the First International Poliomyelitis Conference at the Waldorf-Astoria Hotel in New York City next July 12 to 17.

The Department of State has been requested to transmit invitations to more than 60 foreign governments to send official delegates to the conference. These officials will be asked to present summarizations of the problems of poliomyelitis in their countries at a special session. Presiding officer at this session will be Thomas Parran, M.D., Surgeon General of the United States Public Health Service

Official host to the delegates will be Basil O'Connor, president of the National Foundation, while Hart E. Van Riper, M.D., the Foundation's medical director, has been appointed general chairman of the conference.

The program will include scientific and technical papers on research and treatment of poliomyelitis to be presented by professional authorities in the field from this country and abroad. In addition, there will be panel discussions on the various subjects.

It was announced also that conference headquarters have been established in the Waldorf-Astoria Hotel under direction of Stanley E. Henwood, of Chicago, who has been appointed executive secretary of the conference. Arrangements for the conference will be directed from there by Mr. Henwood.

The program for the conference is being arranged by a seven-member advisory board which includes: Irvin Abell, M.D., clinical professor of surgery at University of Louisville; Morris Fishbein, M.D., editor of *The Journal of the American Medical Association*; David Lloyd, Ph.D., associate member of Rockefeller Institute for Medical Research; Kenneth Maxcy, M.D., professor of epidemiology at The Johns Hopkins University; Rustin McIntosh, M.D., professor of pediatrics at Columbia University; Frank Ober, M.D., professor emeritus of orthopedic surgery at Harvard University, and Thomas Rivers, M.D., director of Hospital of the Rockefeller Institute off Medical Research.

In addition to the sessions, there will be a scientific exhibit section, demonstrations of muscle testing and treatment procedures, and a film program. Co-ordinating this phase of the conference will be an advisory committee of Thomas G. Hull, Ph.D., director of scientific exhibits of The American Medical Association, and Charles F. Branch, M.D., director of scientific exhibits of The American College of Surgeons.

Empire Advisory Bureau British Medical Association

Tavistock Square, London, W.C. 1

The Council of the B.M.A. has decided to establish an Empire Medical Advisory Bureau at its headquarters in London, and we would draw attention to an advertisement in a recent issue inviting medical practitioners with administrative experience and organizing ability to apply for the post of Medical Director of the Bureau, the work of which will be under the general direction of a Committee of Management. It has long been felt both by medical men from the Dominions and Colonies and by those who do their best to help them when they come to this country that there should be one place and one person to whom they can come for advice on the numerous medical and personal matters the solut on of which would go such a long way to helping them to make their visit pleasant and profitable. Most medical men from the Dominions and Colonies come to this country with a view to obtaining postgraduate instruction. Great Britain now has a wealth of medical talent and experience which is unequalled in Europe. The reputation of British medicine has probably never stood higher than it does to-day, and we may justly be proud of the fact that some of the fundamental contributions to medical science during recent years have been made by men working in this country. Postgraduate education is now in the process of being put on a more firm footing with the development of the British Postgraduate Medical Federation, under the Direction of Sir Francis Fraser. Although the facilities for this work are not yet available, men working in the principal medical centres in Great Britain are always pleased to welcome and to help the medical man and woman from the Commonwealth and Empire who come here to learn new things. Part of the work of the new Empire Advisory Bureau, therefore, will be to see that the Dominion visitor has all the information he wants about postgraduate study whether it is for the purpose of taking a higher qualification or for learning about new surgical and medical techniques.

In this country we perhaps tend to forget that the Commonwealth visitor arriving in England for the first time feels very much a stranger, is faced with unfamiliar customs, and is naturally ignorant of those various details that can be so harassing to the stranger. An important part of the job of the Medical Director of the Bureau will be to see that the newcomer is put in touch with suitable lodgings and hotels, receives information about facilities for sport and travel, and is introduced to medical men in this country who are prepared to offer private hospitality. Through members of the Branches and Divisions of the B.M.A. it is hoped also to arrange that the man or woman doctor from the Commonwealth will be greeted as soon as he arrives, whether it is at an airfield or at one of the ports. Here indeed is a job well worth doing and one which will enable the holder of it to play a more than useful part in tightening the bonds that already exist between the medical men of Great Britain and the Commonwealth and Empire.

December 16, 1947

Spot News

Mr. Donald Gordon of the Foreign Exchange Control Board has informed me, in answer to a question, that funds spent by Canadian Doctors attending Medical Conventions in the United States of America will be regarded as business expenses and will not be deducted from the allowances available to Canadian citizens for pleasure travel in the United States.

T. C. Routley

Notice Concerning the Treatment of Civilian Paraplegic Patients

The letter following has been published so as to bring to the attention of the physicians of Nova Scotia the fact that the Department of Veterans' Affairs are now accepting civilians in centres which have been established for the treatment and rehabilitation of paraplegic patients.

135 St. Clair Avenue, West
Toronto 5, Ontario
July 3, 1947

Dear Doctor:

Re: Treatment of Civilian Paraplegic Patients

At our recent annual meeting in Winnipeg a letter was received from Dr. W. P. Warner, Director General of Treatment Services, D.V.A., stating that the Department is operating four Departmental Special Treatment Centres—Shaughnessy Hospital, Vancouver; Deer Lodge Hospital, Winnipeg; Christie Street and Lyndhurst Lodge, Toronto; Queen Mary Veterans' and Ste. Anne's Hospitals, Montreal—where much has been accomplished for the rehabilitation of paraplegic patients.

As the number of veteran patients is now decreasing, the Department would be willing to treat a certain number of civilian paraplegic patients where no civilian treatment centres are available, and they ask the Canadian Medical Association to express its approval of this policy.

After considering the matter in the Executive Committee and General Council, it was agreed:

"That the plan outlined by Dr. Warner be approved, subject to the approval of the Divisions."

It would be appreciated if you would take this matter up with your Division and let us have your reply as soon as convenient.

Yours sincerely

(Sgd.) T. C. Routley
General Secretary

Invitation to All Surgeons to Attend International Surgical Assembly

The Sixth International Assembly of the International College of Surgeons will be held in Rome, Italy, at the invitation of the Italian Government, during the week of May 16-23, 1948, under the presidency of Professors Raffaele Bastianelli and Raffaele Paolucci of Rome, and Mario Dogliotti of Turin. The Secretary of the Assembly is Prof. Giuseppe Bendandi of Rome. Attendance is not limited to the membership of the College: all surgeons in good standing in their medical organizations are invited. Scientific meetings, scientific and commercial exhibits, visits to the Universities of Turin and Milan have been arranged, together with tours to other medical centres in Europe. A special exhibit of ancient texts on surgery is being arranged by Prof. Davide Giordano of Venice, Honorary President, under the active presidency of Prof. Adalberto Pazzini, Professor of History at the University of Rome. This extraordinary exhibit dealing with ancient surgery will be on display in the Vallicelliana Library in one of the historical buildings of the Vatican. Detailed information may be obtained from Dr. Max Thorek, General Secretary, 850 Irving Park Road, Chicago 13. For travel information, address the All Nations Travel Bureau, 38 S. Dearborn Street, Chicago, the official travel representatives for this Assembly. Those desiring to present scientific papers address Dr. Karl Meyer, Cook County Hospital, Chicago; Dr. Henry W. Meyerding, Mayo Foundation, Rochester, Minnesota; or Dr. Herbert Acuff, Acuff Clinic, 514 W. Church Street, Knoxville, Tennessee. Those from Canada should direct their inquiries to Dr. Lyon Appleby, 925 W. Georgia Street, Vancouver, B. C.

Errata or We Have Erred

The editor acknowledges with some embarrassment certain sins both of commission and omission in the December BULLETIN. Under the former heading there are at least two glaringly misspelt words. There may be more but so far they have not been found or drawn to our attention. First of all "Editorial Foreword" is spelt "Forward"—not once, but twice. This makes us look silly—an experience perhaps wholesome once in a while.

The other error in spelling is in the quotation from Doctor N. H. Gosse's letter to the Secretary concerning the minutes. In the last phrase which should read "in fact or *intention*" the last word is spelled intuition. For the information of any who may have noted the peculiarity we hasten to say that Doctor Gosse did not use the word intuition—and that the substitution was apparently made by the printer at the last minute for reasons best known to himself. Our apologies to Doctor Gosse.

The sin of omission also makes us look silly. We stated in our "Editorial *Foreword*" that by a happy co-incidence we were able to publish the Discussion on the five papers forming the Symposium—but no Discussion appeared in the BULLEEIN. The reason for this, which emerged after some research, was that the proof of the discussion was retained by one of the five authors. It was our belief that it had been returned to the printer, and, when asked if all material had been returned the printer replied that it had. We doubt if the Discussion is of sufficient interest to make it worth while to put it in another number. If sufficient requests are received, we will furnish mimeographed copies. Further and more intensive proof-reading is obviously indicated. This we shall endeavour to supply.

M. E. B. G.

Personal Interest Notes

Country Doctor has no Thought of Retiring

THERE are ten subscribers listed on the St. Peter's, P.E.I. exchange, and the most popular belongs to 89-year old Dr. R. J. McDonald, probably the oldest practising country doctor in Canada.

Big, tall Doctor McDonald has made that little village in Eastern Prince Edward Island the centre of his practice for sixty years.

The weather hinders him about as much as people who ask him if it were not about time he was retiring. In summer he drives his own car over rugged secondary roads; in winter he uses horse and sleigh.

Somebody asked him recently if he were thinking of giving up his practice and taking it easy for a while. Retorted the doctor: "What do you think I am slipping?" He got a faint "No" to that question.

The nearest doctor east of St. Peter's is at Souris, about fifteen miles away, and south, at Montague, some sixteen miles off.

Born at Maple Hill, P.E.I., Doctor McDonald receive his medical degree at the University of Toronto in 1888 and returned home to hang out his shingle. He has seven children. His wife died last fall. They had been married fifty-three years.

A life-long friend of Doctor W. J. P. McMillan of Charlottetown, leader of the Progressive Conservative Party, Doctor McDonald served for one term in the Island Legislature.

A voracious reader, he peruses thoroughly nearly everything he can lay his hands on, with the exception of fiction. He does read some fiction, but it must have an historical background. He says he can learn from such stories but that not much can be gained from the rest. If a book, magazine or newspaper cannot teach him something, he leaves it alone.

He was rushed to so many thousands of cases that his walk rather resembles an average person's sprint. Most people have to trot briskly to keep up with him.

Doctor McDonald accepts calls at any time of the day or night and he continues to deliver babies, mend broken limbs and treat sore throats in scores of little communities like Five Houses, Cable Head, Goose River and Shipwreck Point. By now it's strictly old hat.

Announcement from St. Martha's Hospital in Antigonish tells of the success of two doctors in recent examinations. Doctor J. A. MacDougall, recently appointed anaesthetist, has received work that he passed examinations conducted by the Royal College of Physicians and Surgeons and has been certified as an anaesthetist by that body. At the same time, Doctor F. J. Hogg, who took over a practice in surgery early in January also passed the examinations of the Royal College of Surgeons (Canada).

Doctor E. D. Levittan and Mrs. Levittan left early in December for England. Doctor Levittan practised in Guysborough for six years and during the past year has been studying at the Pratt Diagnostic Hospital in Boston, and the Nova Scotia Sanatorium in Kentville. He is continuing his studies now with the British Post-graduate Medical School at Hammersmith Hospital, in London, for a two year period. Doctor Levittan is a graduate of Columbia

University in Science and graduated from Dalhousie University in 1940 in Medicine.

Find Tiny Virsus Causes Common Cold

A virus too small to be seen through the most powerful microscope in Britain is causing the common cold. This has been discovered by the British Government's common cold research unit, set up eighteen months to carry out experiments, never before attempted on such a scale. After tests on 300 volunteers—honeymoon couples, students and office workers anxious for a holiday and rest at government expense—research has found, by the use of special filters, that the virus is 1-10,000th of a millimetre in size. It has also been discovered that it can withstand freezing at temperature as low as 94 degrees below zero Fahrenheit.

Doctor S. J. Shane, of Yarmouth who graduated from Dalhousie in 1940, has been awarded the Degree of Follow of the Royal College of Physicians of Canada. Doctor Shane is at present on the research staff of Cornell University Medical College, New York, where he is engaged in investigations concerning the treatment of various forms of heart disease.

Obituary

DOCTOR William Joseph Barton died at Halifax, Nova Scotia, on January 5th, after an illness of three years. Doctor Barton was born at Pubnico, Yarmouth County, on April 12, 1872. He studied his pre-medicine at Dalhousie University and his medical course at the College of Physicians and Surgeons in Baltimore, graduating in 1896. He practised medicine for fifteen years at Pubnico, and then came to Halifax where he practised until ill health forced him to retire three years ago.

During his lifetime Doctor Barton was devoted to his profession and his family. His wife, the former May Penny, predeceased him in 1927. He is survived by two daughters, Rita and Margaret of Halifax, three sons, Doctor Frederick of New Waterford, William at the University of Ottawa, and Frank at Saint Mary's College in Halifax, one granddaughter Anne Marie of New Waterford, two sisters, Sister Leo Cadia at Saint Joseph's Convent in Halifax, Mrs. Patrick Sullivan, Boston, and two brothers, James of Homewood, Illinois and John of Reading, Massachusetts.

The funeral took place on January 7th, from his late residence, 325 Brunswick Street, in time for nine o'clock Mass at St. Patrick's Church. Interment in Mount Olivet Cemetery.