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Poliomyelitis : with special reference to the role of physiotherapy in the treatment of the acute phase

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POLIOMYELITIS

WITH SPECIAL REFERENCE TO THE ROLE OF PHYSIOTHERAPY IN THE TREATMENT OF THE ACUTE PHASE

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"And if so be any man
object unto me that this
discourse is only com-
pilede together of cert-
ayne rapsodyes of anti-
que Chyrugians I will-
ingly heere confes and
acknowlegge that in this
treatise there is verye
little or nothing at all
of myne own Invention."

Jaques Guillemeau
A.D. 1594

INTRODUCTION

Infantile Paralysis is a term well-known to the layman of this day and age. In daily life he is forced to visualize the ravages of the disease: the workman beside him has a "lame leg"; the child next door wears braces and can't run or play with the rest of the children; his brother has a twisted-hunched back, is weak and unable to support himself. At the movies, newsreels show flashes of President Roosevelt with his ever present aide. The disease plays no favorites; it strikes at the rich and mighty, as well as the humble.

Each year large donations are made to the cause of the fight against poliomyelitis. In the annual report of the National Foundation for Infantile Paralysis made September 30, 1942, 77 grants and appropriations totaling $1,142,009 were recorded (1).

In the last three decades many clinical studies have been made and a vast amount of experimental work has been done. Great advances have been made in many fields, yet, even today, the picture is far from clear. No true cure has been devised, and the people still fear the scourge of this disease. Consequently it is easy to understand the great amount of publicity which accompanied Sister Kenny's

"mission" to this country two years ago. This austere nurse came out of the Australian bush with wonderful stories of children sent home, well and with no residual paralysis two or three months after the onset of the disease. What was this new method of therapy the people wanted to know?

The story of her fight for medical recognition (which she has at last attained) is quite romantic and reads like one of the chapters from Horatio Algers. The fact that her method is just the opposite of the so-called orthodox treatment has, no doubt, a tendency to enhance the tale. To many she has become an "angel of mercy", the heroine of the age.

The National Foundation for Infantile Paralysis made grants, and provision was made for field studies. Glowing successes have been reported and many physicians, among them distinguished orthopods, have acclaimed the Kenny method of treatment. Like the sulfonamides, when they first were used therapeutically, her method has swept the country and taken it by storm. But just as in the case of the famous "cure-all drugs", here and there through the recent medical

literature have come reports not substantiating Sister Kenny's theories and methods. Arguments have sometimes been bitter on either side of the question. The confusion surrounding poliomyelitis still reigns.

Perhaps this is a poor time to be writing a thesis upon this particular subject. However, an interest in the fight that is progressing between the "right and left wings" of this therapeutic problem and the desire to analyze and draw conclusions myself has prompted me to take up this subject.

HISTORY

The mysteries which shroud the disease Poliomyelitis extend even into the realm of its history; many of its historians are loath to say whether it is ancient or comparatively modern in origin. The latter would certainly be of prime import were this discussion limited to its epidemic nature. However, a number of things have been found which indicate the possibility of its being one of the earlier diseases of man.

An "archeologist's delight" they call the country of Egypt, and it was here that the first reference to Poliomyelitis in man was found. In a village south of Cairo a skeleton was found dating from approximately 3700 B. C. It was important from the standpoint of this disease because one femur was shorter than the other by 8.2 cm. with no evidence of having been broken. Shortening of the leg is one of the characteristic deformities often noted in the more severe forms of poliomyelitis. Therefore, this was considered by certain authorities as the first known case of infantile palsy (2). However, the point is in dispute since MacAusland (5) and others have pointed out the possibility that its

origin could easily have been of a congenital nature.

Other facts have been uncovered, however, which aid and abet the "ancient" theory. An Egyptian stele of the 18th dynasty (1580-1350 B.C.) pictures a man with a typical "equinous position." Skeletons with similar deformities have been discovered in Greenland (2). They are thought to be Norsemen who settled there early in the 15th century.

These facts can only suggest the early appearance of poliomyelitis, but, if true, may be of importance from an epidemiological standpoint.

On the "modern" side of the question the first clinical studies of the disease recorded begin early in the 18th century. Certain small references can be found here and there among the literature; among these was one made by Sir Walter Scott (as recorded in Lockhart's Life of Sir Walter Scott). He describes it as "the fever which often accompanies the cutting of teeth." In 1773 Scott was attacked by the disease at the age of 18 months; one leg was paralyzed and permanent lameness resulted.

The first description of the disease of any importance was made by Underwood in 1793, in the first edition of his treatise on "Diseases of Children." (2)

He managed to devote two pages to the "Debility of the Lower Extremities." About treatment he had the following to say: "These cases seem to be benefited by external use of the waters at Bath." Here in the very beginnings of its recorded history is a suggestion or idea on the possibilities of hydrotherapy--a process still in use almost two centuries later. Surely in this point, if no where else, is an example of the "art of medicine," practiced by men of these times.

In 1799 Underwood adds this to his idea about treatment: "In any case, the only remedies I have found necessary, have been calomel or some other purgative; sometimes an emetic, and volatile embrocation to the limbs. Electricity, I am told, has been advised in one instance; and if the complaint should not otherwise yield, may as properly be had recourse to in this, as the former instances." He was evidently gaining a clearer concept of the disease.

Dr. J. Badham of Worksop, Notts in 1835 gave an account of four cases occurring in children (2). He drew attention, for the first time, to the cerebral symptoms. In these cases treatment consisted of one or all of the following: calomel, cold applications to the head, blisters to spine, cataplasms to affect-

ed limbs, and strychnine externally.

Despite notes made on the disease up to this time, Jacob Heine's monograph in 1840 gained for him a signal honor--and he is today considered the pioneer contributor to modern knowledge of poliomyelitis. Heine concluded from his studies that symptoms of the disease "point to an affection of the central nervous system, namely of cord, of an irritative and congestive type." The additional fact that the disease tends to occur in epidemics was noted some thirty years later by Medin in Stockholm. Hence, even today, the term "Heine-Medin disease" is commonly used in Germany and Scandinavia to include all the various clinical forms of poliomyelitis (3). The first adult case of poliomyelitis was described by Vogt in 1859.

Up to this time medical men had been little concerned with the pathological processes involved. However, in the year 1863 von Reinecker and von Recklinghausen in Germany and Cornil in France described lesions in the anterior horns and lateral columns of the spinal cord. Not long afterwards in 1888 Rissler made his classical histological study of five cases--three of which died in the acute stage of the disease. It was his luck and opportunity

to observe the "earliest changes in the cord that has thus far been examined by anyone" (4). His work has since been enlarged upon and completed by a number of workers--notably among these is Wickman. It was this man who started certain concepts of pathological anatomy. In 1905 he wrote, in effect, that he believed infection took place via the alimentary tract; that transmission of the virus was along nerves by way of lymphatics; the disease is probably spread by person to person contact--and this last idea is still being forwarded by many investigators today.

In 1884 it was Strumpell who first noted the resemblance between cases of acute encephalitis with spastic paralysis in children and certain cases of poliomyelitis (2). It was his conclusion that these two diseases probably had a common etiological factor and pointed out for the first time the possibility of an external infectious agent for both of them. Marie corroborated Strumpell's observations and theories in the following year. Therefore, the cerebral type of poliomyelitis is referred to as the "Strumpell-Marie type."

Until the last two decades of the nineteenth

century only sporadic cases, or at the most only a few to a community, were reported. Since then many large and severe epidemics have occurred in many places throughout the world. The reason for this sudden up sweep in the number of cases is another fact about poliomyelitis history that belongs properly under that shroud of mystery. From an epidemiological point of view, poliomyelitis must be considered a disease of the present.

From the standpoint of physiotherapy, Osler in 1906 suggested that the child should be kept in bed and the affected parts wrapped in cotton. Rubbing and kneading affected parts was started as soon as the child could stand it. In this same year A. H. Tubby and Robert Jones (6) wrote, "the element which has to be combatted is the secondary change in the contracted stronger group of muscles. This change is termed contracture; it involves tissue shortening due to prolonged contraction.....treatment is by means of splints." Here is one of the first occasions in which immobilization is suggested. Later, in 1910, Lange suggested the use of plaster of paris casts. (7)

In 1916 the Harvard Infantile Paralysis Commission was formed. It advocated use of massge,

muscle training and braces if necessary. In addition, Peabody, a member of this commission, suggested use of wire splints at night and active and passive motion during the day, along with heat and massage.

During the period of the last two decades complete immobilization has become the treatment of choice. Now Sister Kenny is advocating symptoms heretofore unrecognized, and a treatment in which immobilization is condemmed. In the next section, symptomatology, the old and the new, will be discussed.

SYMPTOMS and DIAGNOSIS

Acute anterior poliomyelitis has been defined as "an infectious disease which primarily involves the central nervous system, attacking especially the gray matter of the spinal cord, but capable of producing widely separated lesions throughout brain and cord and also in extra-neural tissues. The most striking changes are found in the gray matter of the anterior horns of the cord; and it is these lesions which give rise to the characteristic clinical feature of the disease, namely, the flaccid paralysis of various groups of muscles" (8).

In the short time since this was published (1941) new ideas and concepts of the disease have been forced on the attention of the medical profession. Now we cannot be quite sure that this particular definition is either completely true or adequate. The reasons for this statement will be given presently.

Anterior poliomyelitis has been divided into three stages on basis of clinical and pathological manifestations. The stages are (1) acute, (2) convalescent, and (3) chronic. According to the classical description of Lovett (9), the acute phase lasts from onset to disappearance of muscle tenderness, usually about

eight weeks; the convalescent phase begins at the end of the acute phase, continuing so long as spontaneous improvement is marked (about two years)--the time for greatest gain in matter of ultimate function; the chronic period continues onward from end of convalescent phase and is the period in which treatment is largely operative. It is with the acute phase that I will largely concern myself in this paper.

The onset is variable, but in the main can be classified according to three types (2):

(1). No prodromal symptoms or signs occur. Onset of paralysis is sudden.

(2). Onset occurs with indefinite symptoms of a gastrointestinal or anginal nature. Fever usually occurs (100°-103° average) for a few days, followed by a remission of one to several days. Then fever and systemic symptoms reoccur and the condition may go on to paralysis.

(3). Systemic symptoms progress rapidly and uninterruptedly to those indicating involvement of the central nervous system. This type is most common.

The following symptoms and signs is a composite of those listed in various textbooks printed since 1932. The percentages quoted are taken from J. A. Adamson's review of an epidemic that occurred in Manitoba Canada, 1941, (10) and are not to be taken as entirely typical--however they do represent a close average of the 408 cases studied.

(1). Headache. The is one of the most common subjective findings; 83.7%; usually frontal; duration varies directly with severity of case; usually intermittent.

(2). Pain in neck, back, and limbs. Valuable diagnostically. Absent in only 9.6%. Pain in neck, 69%, corresponded with headache in duration and intensity but disappeared long before objective evidence of neck stiffness.

(3). Fever. Quite constant symptom; 100-103°. Course is similar in all cases. Pulse rate in proportion to fever usually.

(4). Vomiting. 46%, in some epidemics is leading symptom and often associated with diarrhea.

(5). Congestion of throat and pharynx--25%.

(6). Blurred vision and double vision. Minor complaints about vision sometimes occur, but are usually rare.

(7). Sweating--sometimes profuse. Importance controversial, considered not greater than with any other fever by some men.

(8). Somnolence and insomnia. Most cases appear to be more alert and sensitive than normal. In Adamson's review somnolence occurred in 12.7% of cases and was mild and short.

(9). Extreme drowsiness alternating with irritability when disturbed.

(10). Hyperesthesia. Often occurs early, with slight touch or pressure, especially over the spine or large nerve trunks.

(11). Tremor or twitching of muscle groups. Sometimes occurs in the tongue.

(12). Stiffness of back and neck. 42.8%, occurs more often among "paralytic group," not always associated with pain--often persistent much longer, sometimes as long as two weeks.

(13). Kernig's sign--rarely present.

(14). Speech and swallowing difficulties; 15%; mostly due to palatal paralysis.

(15). Absent tendon reflexes. Early: deep reflexes often exaggerated and equal, occasionally a Babinski or clonus is present. Late:

deep reflexes are unequal, diminished or lost. Absent in 18.4% of cases. 47% with definite leg involvement had tendon reflexes intact up to the time of discharge. Therefore their persistence or disappearance is poor evidence on which to base the chance of paralysis.

(16). Bladder and bowel involvement. Usually transient if present. In severe cases may have incontinence.

(17). Absent abdominal reflexes--occurs mainly in paralyzed groups, 16%.

(18). Paralysis. Usually develops on the second or third day, but has occurred as late as the fifth day. The paralysis is most common in the legs, but can occur in any muscle or muscles and to varying degrees. According to McNalty (3), the paralysis may be of either the flaccid or spastic type depending on site of the lesion. The usual view taken, however, is that this disease produces a lower motor neuron type of paralysis.

(19). Tenderness. Varies from tenderness on slight pressure to exquisite sensitiveness to touch, usually located in affected

muscle. Average duration is six weeks; has been known to last 16 weeks, especially with too early manipulation.

As pointed out, not all these symptoms occur in the same proportions. The main ones, upon which the diagnosis is most often made is headache, fever, stiffness of neck and back, muscle tenderness and spontaneous pain, and paralysis when it occurs. The main laboratory aid is spinal puncture. The spinal fluid may show the following changes, which are not diagnostic in themselves, but do often help point toward the diagnosis:

(1). Total cell count. May be from 10-1,000. Averages 10-150

(2). Differential cell count. First day, lymphocytes predominate. Following three days, lymphocytes and granulocytes may be present in equal proportions. Fourth day, the granulocytes decrease and may disappear by the seventh day. Adamson found that a preponderance of granulocytes in the fluid after the second day was of good prognostic significance from the standpoint of paralysis. Cell count usually falls to normal at end of the second week.

(3). Protein may be slightly increased; not parallel with the cell count. Protein may remain up until the seventh week or there about.

(4). Colloidal gold curve. 1122100000 to 1123321000 characteristically according to study of Peabody, Draper, and Dochez (11).

(5). Spinal fluid is said to have a ground glass appearance.

(6). In rare instances spinal fluid will be completely normal.

For purposes of diagnosis the disease has been divided into clinical types. Many types have been listed in the literature; I have chosen to reproduce here the one recommended by the International Committee organized by Jeremiah Millbank in 1932 (2). I have chosen this particular one because of its simplicity and practical significance.

(1). Abortive type. Cases with mild constitutional symptoms but with no evidence of central nervous system involvement. Common in epidemics; no mortality associated.

(2). Nonparalytic type. Cases in which nerve

cells not sufficiently injured to produce paralysis, although there may be a transient weakness--few hours to days. Also common in epidemics; no deaths.

(3). Type with subcortical paralysis. This is the usual type found in epidemics. May also include ascending and descending paralytic types and bulbar forms with paralysis of cranial nerves (facial and abducens most commonly involved, olfactory and auditory never). The bulbar form has a high mortality rate, sometimes around 75-100% in severe epidemics.

(4). Central or encephalitic type. This may occur either with or without upper neuron involvement. As a rule spastic paralysis is rare. Disturbances of sensorium manifested by drowsiness and stupor also form a rare type. This form has a very low mortality rate and is seen only with large epidemics.

(5). Ataxic type. In this type the motor cells evidently are not involved. The type is characterized by incoordination, ataxia, and nystagmus (on post-mortem examination

find cerebellum, Clarke's column and inter-vertebral ganglia involvement). This type is quite rare.

The paralysis which occurs is usually of the flaccid type; the stronger unaffected muscles over-powering antagonistic, but paralyzed muscle groups. This leads to characteristic deformities of the chronic stage such as foot drop, hyperextension of the legs, scoliosis, lordosis, etc. unless adequate treatment can be instituted. Cases of the abortive or nonparalytic type are sometimes hard to diagnose, especially where the cases are of a sporadic nature. Diagnosis, here is based solely on the acuity of the physician.

This picture of the disease in the acute stage represents the so-called orthodox view of acute anterior poliomyelitis. The disease is not well understood in most all of its phases; the epidemiology, pathology, and even the etiology have been in dispute from its early history starting in the middle of the 19th century up to the present moment. However, for the greater part, most medical men were quite well agreed on clinical symptoms if not their cause. Therefore, it was some-what of a shock when out of

the Australian bush came rumblings of views diametrically opposed to all that had gone before.

Sister Kenny's concept of this disease has given rise to three new terms--based on her idea of the symptomatology of acute anterior poliomyelitis. These terms are (1) muscle spasm, (2) incoordination, and (3) "mental alienation."

These three symptoms have been described by Cole, Knapp, and Pohl who have worked with Miss Kenny in Minneapolis for the past two years (12).

Muscle Spasm. "This term denotes a group of symptoms including fibrillary twitchings, hyperirritability of the muscle to stretching and more or less tonic state of contraction of the muscle fibers which frequently cannot be overcome even by great force.....A patient acutely ill has muscle weakness, the muscles are painful, tender, irritable, and in spasm.....A muscle in spasm is one attempting to shorten itself. Temporary contraction early, which if unattended, soon develops into fixed deformities.....Examination of a patient who has not been treated for spasm leads to the conviction that the patient suffers frequently from stiffness and

contractures rather than paralytic weakness. Muscle is normally an elastic tissue and depends for its function upon the property of being able to lengthen and contract itself. If the muscle is allowed to become short through spasm or any other process its useful power is definitely lessened. Furthermore inability of the muscle to lengthen imposes a severe restriction on the action of its opposing muscle.

"The principal locations of demonstrable muscle spasm in approximate order of frequency are: (1) hamstrings, (2) back and neck, (3) posterior calf muscles, (4) pectorals, (5) muscles of respiration, (6) quadriceps, (7) biceps of the arm, and (8) forearm and anterior leg muscles.

"Detection of spasm is by (1) observation, (2) observation and palpation, (3) passive stretching of muscle in spasm."

Muscle spasm in different regions of the body also produces certain characteristics which aid in diagnosis, according to this disease concept. For example, in the neck: prominence of the extensor muscles; head in hyperextension; patient cannot flex

neck; pain in back on neck with passive flexion.

Incoordination. This is said to be principally of two types, "(1) that due to spreading of motor impulses intended for a certain muscle to other muscles or groups of muscles due to such conditions as pain on attempted motion of involved muscles or inability of that muscle to perform its proper function; (2) that occurring within the involved muscle itself so that ineffective contraction is produced instead of a coordinated rhythmic contraction producing maximum motion at the insertion of the muscles.....uncontrolled voluntary motion when attempted in presence of spasm leads to symptoms of incoordination by the development of abnormal motion patterns and misuse of muscles. Active motion on the part of a patient unless carefully supervised and directed may do great harm, therefore, and lead to difficulties in securing the return of normal function."

Mental Alienation. This is a physiologic block; not organic. This symptom may be produced in the following ways:

(1). A muscle is pulled beyond its normal resting

length by its opponent which is in spasm.

(2). "Alienation" produced by pain in an involved opponent by the attempt of such unaffected muscle to contract.

(3). Spasm produces a braking action on the normal opposing muscle, and may be so severe as to produce alienation.

(4). Changes in the nervous system may occur which do not actually destroy nerve cells or fibers but do cause loss of conduction power.

This whole symptom-complex is neatly described by A. E. Deacon (13). "According to Miss Kenny..... acute anterior poliomyelitis should be classed as spastic paralysis....flaccid paralysis is secondary, a result of the spastic paralysis.....If one group of muscles is in spasm and cannot relax, the antagonistic group is prevented from fully contracting due to the brake-like action of the opposing muscles; any attempts at contraction of the non-spastic group stretches its antagonistic spastic group and increases the spasm and pain; a fear complex is set up and the patient refrains from using his non-

spastic group; a functional breakdown between the brain control and the non-spastic group develops and the non-spastic muscles undergo a flaccid paralysis:.....the patient loses his mental awareness of these flaccid muscles, and the flaccid muscle becomes 'alienated' from their brain control."

The detection of incoordination according to Sister Kenny (14) depends on the observation of whether the acting and opponent muscle groups are attempting to contract together or when one group of muscles (unaffected) are used to perform the function of a second group (affected). Mental alienation occurs when no voluntary motion can be obtained from an uninvolved muscle group.

Since these views expressed by Sister Kenny and her "backers" are so radically different from the accepted idea of poliomyelitis, it might be well to see if there is any pathological basis upon which such views might be founded. The pathological picture and course of poliomyelitis is as yet not clear-cut. For this reason a resume of "known" pathology will be given first.

PATHOLOGY and PATHOGENESIS

The pathology of poliomyelitis is centered about two main processes; one involving the ganglion cells and the second involving the supporting tissue. Both of these components are markedly affected, the former showing a wide range of changes indicative of degeneration and necrosis, the other characterized by hyperemia, edema, cellular infiltration, and hyperplasia. These changes are confined almost entirely to the gray matter, although of varying topographical intensity.

From the time of Rissler (1888) up to the present day a literary argument has ensued over which of these processes occurs first in the tissues. It was Rissler's idea that ganglion cells were affected first and that changes in supporting tissue were secondary. Wickman, along with other early workers, promulgated the opposite view. He believed that nerve cell degeneration was the result of pressure and blood-vascular changes occurring in the interstitial tissues. These early workers believed that poliomyelitis was a generalized infection, affecting mainly the anterior horn cells of the spinal cord and the nervous tissues in general (15).

However it is quite generally agreed today that the virus of poliomyelitis is neuronotropic, that its transmission in the body is by axonal progression, that the primary effect of the virus is on susceptible neurons, that the inflammatory reaction is of a secondary nature being the result rather than the cause of the neuronal damage.

The pathologic course as described by Sabin (16) is as follows: "The cells show a diffuse chromatolysis and sharply outlined, acidophilic, intranuclear inclusions generally have been found one or two days before onset of paralysis. This process then progresses to complete acidophilic necrosis of the cells and invasion of polymorphonuclear leukocytes, which ultimately arrange themselves over the dead neurons to form a typical focus of neuronophagia. After several days the polymorphonuclear cells disappear and their place is taken by glial cells. This series of changes may be taken to represent the pathologic unit of poliomyelitis, and the resulting clinical signs may be regarded as depending on the number and location of neurons so affected. Associated with this there is also an interstitial

and perivascular infiltration with cells which vary with the stage of the disease, many polys being present early and mononuclears and glial cells predominating later. The cells present in the meninges actually represent an overflow from the perivascular spaces and are thus secondary to neuronal damage rather than the result of a true meningitis. The so-called signs of meningeal irritation, including the familiar spasm of muscles of the back of the neck and back, therefore, are more properly regarded as the earliest signs of neuronal damage than the result of a true meningitis.

"Although the disease may primarily affect one region, it may shortly be followed by typical pathological processes in another part of the central nervous system.....Where in the spinal cord the anterior horns are affected primarily, the posterior horns are not altogether spared, and the dorsal root ganglion almost invariably shows destruction of varying numbers of sensory neurons."

Further proof of the fact that the virus affects nerve cells primarily has been brought out in the experiments of Howe and Bodian (17) on monkeys. "Retrograde degeneration of most of the nerve cells of the left ventrolateral thalamic nuclear mass

was caused by ablation of their projection center in the cerebral cortex. One month later an emulsion of spinal cord containing potent poliomyelitis virus was inoculated directly into this susceptible region of the brain which had been denuded of nerve cells, but which was otherwise relatively normal. Although poliomyelitis occurred after five days, with typical pathological changes in the brain and spinal cord, the lesions were practically absent in that portion of the left thalamus which had undergone a previous retrograde nerve cell degeneration. This occurred despite the fact that the virus had been placed directly into the substance of this region. Such a result suggests that the characteristic response of the mesodermal-glial tissue in poliomyelitis, responsible for the non-neuronal lesions, does not depend on the presence of the virus alone but it is due rather to a resultant, perhaps chemotactic, of a virus neuron reaction." In addition, these two men proved that when the virus is introduced directly into a non-susceptible region of the brain (e.g. lateral geniculate body or occipital pole) mesodermal-glial lesions fail to

develop. It appears, therefore, that the virus of poliomyelitis is highly neuronotropic; this is, it has a selective affinity for the neuron itself.

How the virus is transmitted in the body from the portal of entry, wherever that may be, to the central nervous system, has been one of the problems of poliomyelitis during the first two decades of this century. Many theories were advanced, such as transmission by blood-vascular system or by the lymphatics surrounding the nerves. Suffice it to say, that it is definitely known today, that transmission of the virus is by axonal progression. This idea, although suggested before from time to time in the literature, was never given adequate proof until the classical work of Fairbrother and Hurst in 1930 (18). They found that following intracerebral injection the virus gradually disappeared from the site of inoculation, but would later reappear in the pons and the cervical and lumbar regions of the spinal cord. It was shown that the spinal fluid remained free of demonstrable virus, and took no obvious part in its transfer to the cord. It was their interpretation, therefore,

that transmission was by axonal cylinders of neurons to brain stem and cord.

This work has been corroborated. Perhaps that of Howe and Bodian (17) was most extensive and complete. In a series of experiments on peripheral nerves they made the following observations:

(1). Following section of the sciatic nerve of a monkey and moistening cut peripheral end with potent virus suspension, poliomyelitis invariably would result after an incubation period of four to six days. This result was not altered by previous nerve section or freezing the nerve peripheral to the point of inoculation two to six weeks previous to inoculation.

(2). Inoculation of the cut nerve immediately following momentary freezing central to the point of inoculation failed to produce the disease. The explanation given in this case was that physiological continuity of the nerve is not enough for transmission in the absence of living fibers.

(3). Following transection, this same fact was

found true for regenerating fibers that had not completed the process of regeneration.

(4). However when regeneration of nerve fibers was complete (after at least two months) inoculation of the central stump will result in poliomyelitis.

(5). Since nerve central to the point of interruption is morphologically intact as regards vascular supply, lymphatic connections, connective tissue sheaths, Schwann sheaths, and myelin sheaths, it can be concluded that it is axis cylinders that are rendered refractory to the virus.

(6). Evidence on reciprocal relationships between the number of nerve fibers and time of exposure suggests that the virus is not propagated in the nerve fiber by multiplying within it. They further believe that sympathetic nerve fibers are not especially susceptible to the virus; however transmission can probably occur over either motor or sensory fibers.

(7). The mechanism of transport of the virus in

the axis-cylinder remains a matter of speculation.

They further found that even spinal transection would not prevent the virus from passing around this artificial barrier, up or down the cord. It was assumed that the virus passed from the cord via the sympathetic fibers to the paravertebral chains and, after ascending or descending in them, would reenter the cord. This illustrates the possibilities for the movement of the virus through the peripheral nerves.

Not all parts of the central nervous system are equally susceptible to the virus of poliomyelitis. There is a difference quantitatively and in the severity of lesions, depending on their site. Goodpasture states that "the great motor cells of the cord, especially in the lumbar and cervical enlargements, are most susceptible, while cells of the cerebral cortex seem to be relatively so" (4). Apparently area four of Brodmann represents the most susceptible area of the cortex; some areas, as has already been mentioned, are refractive to the virus. This difference in susceptibility is an

important consideration; by it can be explained the curious distribution of lesions which sometimes occurs in this disease, and, the clinical symptomatology which would necessarily follow.

Explanation of the reason for so-called abortive or non-paralytic attacks of poliomyelitis was quite easy when the group believing in a primary inflammatory process held sway. It was their theory that with the relief or edema and pressure on the neuronal cells, those cells not "too far gone" would recover their function. However with advent and proof of a primary neuron-virus relationship, a new explanation had to be found. On close examination of lesions throughout the spinal cord and brain it was found that necrotic, degenerated nerve cells may lie side by side with normal or only slightly affected cells. It was also noted in experimentally produced poliomyelitis that the animals did not need all their anterior horn cells for apparently normal functioning. These findings have led Sabin to this conclusion: (1) "That even when active destruction of the lower motor neurons occurs the segmental distribution of the lesions may be so

spotty as not to affect the major innervation of a given muscle; and, (2) that the virus need not necessarily destroy all the affected neurons but can also produce only partial degenerative changes from which the cell may recover" (16).

From the above discussion it must naturally be assumed that the virus passes to the central nervous system by its peripheral nerve connections. Schultz and others believe this connection to be via the olfactory nerve (19); Toomey as a representative of the group believing in a gastro-intestinal portal of entry, promulgates the idea that the route of pathogenesis is from the gastro-intestinal tract to the cord and medulla by way of the sympathetic and parasympathetic nerve fibers (20). However, proof of route taken by the virus to the central nervous system is still in doubt.

Lesions found in non-nervous tissues are few, and as yet, no importance has been attached to them. Hyperplasia of lymphoid tissue throughout the body is usually associated. No pathological changes occur in muscles or peripheral nerves during the acute stage of the disease. Changes that occur

later in muscles are of the type following destruction of lower motor neurons--flaccid paralysis and atrophy (21).

The description of pathological processes in poliomyelitis given above belongs to what might be termed the orthodox or usual view. Most of the main symptoms listed under this heading can be given explanation. For instance flaccid paralysis is secondary to lower motor neuron lesions of spinal cord and would of course result in absent tendon reflexes often seen in this disease; pain is said to be of central origin--possibly due to lesions of posterior root ganglia or posterior roots themselves; deformities result from the fact that weakened muscles are overpowered by unaffected muscles with contractures occurring if the condition is untreated.

However with the advent of Sister Kenny and her recognition of many symptoms heretofore overlooked by the medical profession (muscle spasm, muscle incoordination, and "mental alienation") many pathologists have begun to reevaluate certain changes found in the central nervous system--changes which apparently have not been considered important

in the symptomatology of the disease.

Regions of the central nervous system most frequently affected are (16):

(1). Spinal cord and posterior root ganglia.

(2). Medulla--vestibular nuclei, reticular formation and nuclei of various cranial nerves.

(3). Cerebellum--root nuclei (fastigii and dentate) and vermis (hemispheres negative).

(4). Midbrain--periaqueductal gray, tectum and tegmentum.

(5). Thalamus

(6). Hypothalamus

(7). Globus pallidus

(8). Motor cortex--especially area four of Brodmann.

Considering these facts Bruce Brown makes the following statement: "Here we have the background not for a single simple picture of flaccid paralysis,. but for diverse symptoms and signs: for incoordination, for tremors, for variations in tonus, for automatic anomalies. Further, we have here a background for transitory signs and symptoms, for

temporary paralysis, possibly for that condition Miss Kenny has called 'mental alienation.'

"We are not even in a position to say that, beyond all doubt, the lesion in the cord can produce flaccid paralysis and flaccid paralysis only. In the first and most obvious place the lesions in the cord are patchy. One may see all the motor cells destroyed at one level and only a millimeter or two away there may be no visible destruction: equally one may find a great deal of destruction in the cord in cases with no clinical paralysis. To produce complete flaccid paralysis of a leg muscle or muscle group according to our standard concept of this disease would require complete or almost complete destruction of the cells of origin of its nerve; that is, almost continuous destruction process involving several segments of the cord. Pathologically such findings seem unusual. Then again one cannot help but wonder what the effect of the poliomyelitis lesion in the cord may be on the mechanism of reciprocal innervation. Recently two physiologists have again raised the question of the double innervation of muscle, innervation for movement and innervation for tone, suggesting that the seat of cells for

both the functions is in the anterior horns. How much then might apparent flaccid paralysis be paralysis of tone rather than, as we have ordinarily conceived it, paralysis of movement.

"From all this I simply make the point that we cannot use the known pathology of the disease to justify a refusal to re-examine the clinical picture of the disease.....Studies have not yet advanced very far, but what studies there are suggest that immobilization speeds up the process of muscle degeneration....The whole disease is in need of reassessment" (22).

Some of these ideas expressed by Brown are new, some of them are just old ideas placed in a new light. At any rate new explanations of certain symptoms are possible. For instance it is Sister Kenny's idea that the paralysis of poliomyelitis is spastic in type. By inference from Brown's article this may be due to either lesions of the higher centers or a "paralysis of tone." Pain according to Miss Kenny is of peripheral origin--due to muscle spasm, relieved when spasm is relieved. The characteristic "stiff neck" of acute poliomyelitis

is said to result from "spasm" of the posterior neck muscles.

Cole, Pohl, and Knapp (12) in their report said "the exact pathological significance of these symptoms (muscle spasm, muscle incoordination, and "mental alienation") is not well understood at present although physiological studies indicate that there are neuromuscular mechanisms which could produce the described symptoms....Probably this acute process in the muscle bears an important relationship to the deforming fibrotic changes in muscle common to the chronic stages of the disease when ineffectively treated in the acute stage."

Under the auspices of the National Foundation for Infantile Paralysis, Herbert Hipps has made studies of late pathological changes in muscles of patients afflicted with this disease (23). This study was made on patients who had had the disease two years or more--using living patients. Thus, in a general way it was possible to correlate muscle findings with muscle behavior.

Microscopically it was found that a gain in strength by a partially paralyzed muscle was through hypertrophy of remaining undamaged cells. Pathological

changes are brought on in two ways: (1) Primarily through denervation, and (2) secondarily from abnormal variations of tension in the muscle.

Cellular changes from denervation begin with atrophy and progress to degeneration, disintegration, and replacement changes. These same pathological changes occur in muscle cells in exactly the same way due to secondary factors. "Too much tension or overstretching results in minute tears, zonal degeneration, and subsequent fibrosis; while too little tension produces changes identical with denervation and rate of change is nearly as fast. These secondary abnormalities may produce just as much weakness in a muscle as primary denervation changes.

"Secondary changes following immobility and disuse seem to be more severe than those following overactivity."

Certainly changes of a primary nature are uncontrollable, but perhaps something of practical value could be done from the standpoint of these secondary changes. Perhaps Sister Kenny's method is the answer. Muscle changes in the acute stage

should be studied if possible.

In addition, other peripheral changes have been noted by Arthur Steindler (24) of a bony and ligamentous nature. "A complete washing out of bone shadow with some evidence of cortical thinning....a marked increase in the density of the shadow with extreme accentuation of longitudinal bony trabecullae.....a transverse band of epiphyseal absorption." This was attributed to circulatory changes due to sympathetic involvement. However this is not definitely proven. Definite loosening of ligamentous reinforcements of the severly involved joints was also observed.

Frank Ober (25) has summed up these points in the following manner: "It is impossible to see that pain, spasm, unexplained bone growth, changes and vascular disturbance in the extremities are all due to lesions in the anterior horns. In certain cases of poliomyelitis there are also sensory changes affecting bladder, bowel, sometimes skin which also cannot be explained by anterior horn changes. Neither does it explain continued pain often seen in non-paralytic extremities or muscle groups."

Since new emphasis is being placed on these peri-

pheral changes and on changes in the central nervous system heretofore passed over as unimportant, and certainly in the light of this new concept of the symptomatology advocated by Sister Kenny, the disease does need to be reexamined thoroughly with an open and unprejudiced mind.

TREATMENT

AND AN EVALUATION OF RESULTS

The treatment of poliomyelitis has not, of course, been limited to physiotherapy alone. Many drugs have been tried both in man and experimentally; drugs used have ranged from hexamethyamine to ergot to the sulfonimides. All have apparently failed. Injection of hypotonic and hypertonic solutions have been tried; results are inconclusive. Chemoprophylaxis has been proposed on the basis that the portal of entry lies in the olfactory mucosa; Rosenow still believes in the efficacy of an antistreptoccal vaccine and serum. However, physiotherapy has played a major role in the treatment of this disease, and since it is still in the foreground therapeuticly speaking, my discussion will be limited to this one factor.

The principle of, so-called, orthodox treatment was laid down about three decades ago by the classical work of Lovett. It is based on rest to the paralyzed muscles and prevention of deformity by means of immobilization. The best means to this end is a routine similar to the following:

(1). Rest in bed. The reason for this is explained by C. E. Irwin. "Without proper care muscle fibers can be stretched and ruptured causing a loss

of power of muscle contraction due to scar tissue formation. This break in continuity is usually due to overactivity (too deep massage, overexercise, inadequate support) and may cause a transverse zone of fibrosis. The early care of acute poliomyelitis, therefore, is absolute rest." (26)

The patient is usally kept in bed for a period of about eight weeks. The bed must have a firm mattress; a plywood board under the mattress is useful to prevent sagging (especially needed in cases with paralyzed back muscles). The bed clothing should be arranged not to press on the feet and thus cause foot drop. The patient may be kept on a Bradford frame. The patient should be kept in a recumbent position with body in as good an alinement as possible--attempt to avoid attitudes aggravating deformity.

(2). Immobilization. This is best maintained by removable, lightweight, well-padded splints or plaster of paris casts, according to the preference of the physician. With paralysis of the leg the foot should be held at right angles, knee straight and hip extended. In the arm a similar platform type of splint should be used; support the hand with the fingers straight and wrist slightly cocked, elbow at right

angles and shoulder abducted to a right angle. "These positions put the affected muscles at rest, avoid abnormal strain on paralyzed muscles, and prevent to a great degree development of deformities resulting from unequal muscle pull. If deformities do occur the extremities are held in the best position for function later on." (27) If wire splints are being used they can be corrected to fit the deformity and reapplied as the deformity diminishes.

(3). Treatment of sensitiveness. In addition to the comfort which immobilization is said to afford, this symptom is treated with hot packs applied to the sensitive areas. Moist heat is best; used at least two times daily. Tenderness under this regime is usually relieved in about three weeks. Where immobilization is by means of plaster of paris casts, such treatment awaits diminution of the acute symptoms. Tests for sensitiveness may be done by passive motion or deep slow squeezing or pressure on muscles.

(4). Massage and rubbing. This portion of the treatment schedule must not be given so long as deep muscle pain and tenderness exist. This applies also to any type of exercise.

(5). Sedatives may be given at the discretion of

of the physician if the patient is delirious or in great pain.

(6). With the beginning of the second or convalescent stage it is necessary to make a careful analysis, evaluating the loss of power in every muscle. This can be based upon a classification something like the following (28):

Gone--no contraction seen or felt.

Trace--tightens but does not move part.

Poor--muscle moves part with gravity.

Fair--muscle moves part against gravity.

Good--muscle moves part against gravity and some resistance.

Normal--muscle moves part against hard resistance ten times without fatigue.

(7). Passive and active exercise. Passive exercise should be started as soon as pain and muscle tenderness have diminished sufficiently (usually start with about twenty minutes and increase gradually). Fatigue is to be avoided. Exercise in warm water is said to be of value.

How soon active movements should be instituted is a matter for debate. Lovett, in his classic of 1916, (9) stated that in the convalescent stage "get

the patient in an upright position early." This was for the purpose of increasing circulation and giving stimulus to the muscle and nervous system. Early walking was to start with only a few steps at a time. Ober in 1938 states, however, that early walking is to be discouraged. (29)

At any rate most authorities are agreed that this phase of the treatment should be in the hands of a trained and competent physiotherapist.

(8). Braces and crutches. These are recommended especially if the patient cannot stand or walk unaided or walks in a position of deformity. The brace should be light and strong, support the extremity, prevent deformity and improve function.

(9). In the chronic stage, treatment is limited mainly to surgery--stabilizations, nerve and tendon transplantations, etc.

These are the main features of the "orthodox" form of therapy with perhaps one exception--and that is the use of the respirator. To most laymen "infantile paralysis" and "respirator" follow, one upon the other. However, James L. Wilson definitely states that there are certain indications and contraindications for its use (30).

The only true indication for the use of the respirator is with paralysis of the intercostals and diaphragm. Disturbance of respiration in the bulbar form of the disease can only occasionally be aided by the respirator. With paralysis of the pharynx causing a collection of mucous and vomitus around the glottis the respirator is rarely effective, and sometimes harmful. The function of the respirator is to provide physiologic rest for the muscles of respiration.

It is Wilson's idea that the respirator should be used for cases of only mildly paralysed respiratory muscles, rather than saving it for a few desperate cases. "Patients with only a moderate degree of paralysis of respiratory muscles, who are far from being cyanotic and show only slight dyspnea with perhaps only a little motion of the alae nasi on inspiration will often, on first being placed in the respirator, drop into profound sleep-- evidently a result of relief from necessity of prolonged conscious attention to respiration. "

The treatment of acute poliomyelitis according to the method of Sister Kenny is based on the three

main symptoms which she has recognized. And, like the symptoms, this treatment is practically opposite to that proposed by orthodox agencies. Her routine of therapy is ritualistic and many men proclaim that only Kenny-trained doctors and technicians should attempt its use.

According to this method treatment should be started as soon as a diagnosis is made. (12) Bed boards are used under the mattress the same as in the orthodox method of treatment. In addition a foot board is placed at the end of the mattress. This allows the patient to maintain normal standing reflexes during the time in bed and should not be considered as a splint. (14) The mattress should be separated from the footboard so the heel can rest against the board when the patient is lying on his back, and toes can project below the mattress when the patient is in a prone position. This is not a particularly new feature since this same principle was suggested by Peabody, Draper and Dochez in 1912. (11) The patients position, of course, should be in as good an alignment as possible.

The treatment of spasm is by hot fomentations. Woolen cloths are cut to fit the part accurately; a light waterproof covering is used then and the whole

composite is covered with a piece of dry woolen material. The first cloth is boiled and wrung from the boiling water twice through a very tight wringer at the bedside. It is then applied very quickly. As a rule the joints are not covered because of the resultant limitation of motion. These " hot packs " are renewed every two hours, or may be applied as often as every fifteen minutes in severe cases.

Early treatment of spasm is important. "A delay of treatment as long as three weeks may mean irrevocable harm to the delicate muscle substance.....with the Kenny treatment acute spasm and pain will subside within one week if the treatment is proper.....hot packs must be continued until the muscle is able to extend itself completely as evidenced by full range of motion of the joint concerned". (31) The aim of this particular treatment is to relieve muscle spasm, pain and tenderness, and thus to prevent contractures and overstretching of muscles. Immobilization of any type is avoided.

As soon as muscle spasm, pain and tenderness are relieved a program of muscle reeducation is reinstituted. The main purpose here is to overcome muscle incoordination and "mental alienation". A good example of incoordination is given by John Pohl (31): "Normally the hip is flexed by the iliopsoas. The

opponent is the hip extensors (hamstrings). Assume the hip flexor ceases to function. To flex the hip the patient may substitute the adductor. To gain mechanical advantage for these muscles in their new function, the hip is now externally rotated. Contraction of adductors is normally associated with relaxation of abductors (gluteus medius and minimus). It is unlikely that the hip extensor will pay out slack, at least smoothly, for contaction of the hip adductor if it is substituted for the flexor. Smooth motion is lost and incoordination is established. This condition may become permanent. Therefore, allow no voluntary effort in the presence of incoordinated muscle action".

The Kenny technique of muscle reeducation is started with passive motion under the direction of a trained physiotherapist. A careful analysis of the power present in all muscles is made first. The insertion of the affected muscle is stroked, the function of the muscle described, and the patient instructed to concentrate on this during the excercises. Thus a program is started to reestablish "mental awareness" of the particular muscles involved and the production of a normal rhythmic motion. Voluntary motions are allowed only when full range of passive

motions are attained and when no incoordination occurs. Fatigue is avoided.

This process of muscle reeducation is not completely original with Miss Kenny since Lovett in 1916 stated(9): "If a muscle is apparently without power, the patient should concentrate his attention on the attempt to accomplish movement while it is performed passively.....not until the muscles are capable of performing a movement through the whole arc should additional resistance be added--then a small amount from time to time.....perform excercises in slow rhythm, allowing complete recovery between efforts to avoid fatigue.....paralysed limbs should be completely uncovered during the performance".

Under the Kenny method "when a patient is no longer suffering from a fear complex, further hydrotherapeutic measures can be adopted by a gradual working up to an immersion in bath, and use of cold sprays and warm douche".(14)

The Kenny method does not permit the use of a respirator. Where the respiratory muscles are involved it is her belief that muscle spasm is the direct cause and the respiratory difficulties can best be relieved by hot foments. However, a dis-

turbance of respiration due to lesions of the bulb cannot be successfully treated in this manner; "her bulbar cases die just like everyone else's and there is no way of preventing it."(32)

On the use of artificial supports, Miss Kenny says "I would urge extreme caution in applying supports, and would give the patient every chance of walking without appliances."

According to Sister Kenny this is the difference in the clinical picture of the two methods.(14)

Orthodox:

1. Muscles affected are never spastic.
2. Affected muscles hang loosely like a hammock between two points of attachment.
3. Where coordination is never damaged, destroyed or disorganized.
4. Where the patient never loses power to control any function that may be left.
5. Where non-affected muscles contract involuntarily and shorten and stretch the weaker, supposedly affected muscles.
6. Where it is neccessary to apply splints in an attempt to prevent deformity.
7. Where stiffness is a troublesome feature and prevents patient's cooperation in re-education by orthodox or any other methods.

8. Where mental alienation of the patient does not exist.
9. Where muscles affected are pronounced to be definitely flaccid and need supporting.

Kenny system:

1. Muscles affected present muscle spasm.
2. Affected muscles become shortened through spasm.
3. Coordination is disorganized and incoordination is frequently seen.
4. Patient frequently loses power in non-affected opposing groups as well as affected.
5. The non-affected muscles refuse to contract being over stretched and bound in this position.
6. It is not necessary to apply splints, casts or frames to prevent deformities is this system. Deformities do not occur.
7. Stiffness does not exist--owing to the fact the symptoms causing stiffness are treated from onset.
8. Mental alienation is accepted as the most damaging symptom, as it leads to permanent paralysis of the part and deformity. Therefore, if treated early, spasm is reduced sufficiently.

9. When spasm is present the opposing groups are not supported owing to the fact that when the spasm is released in the affected groups the stretched non-affected muscles shall return to their normal resting place and mental awareness can be restored by this system, also a normal condition is registered.

Miss Kenny especially condems immobilization because it interferes with the treatment of muscle spasm, incoordination and "mental alienation", and tends to promote stiffness. In addition, she says that it interferes with nutrition of the skin, reduces circulation, does not prevent deformities, and gives the patient an adverse psychological outlook. She believes that her system aids in maintanence of full vitality of all tissues and is the best method for reestablishment of normal function to the affected parts.

There has been considerable controversy in the recent literature over the true value of the "new" type of treatment. Some men claim that it is much better that anything heretofore proposed. Others believe that it holds forth no greater chance for recovery than orthodox methods. The greatest argument seems to hover around two articles written by H. R.

McCarroll. (33 &34) McCarroll in his discussion presents a chart based on 245 cases of poliomyelitis (487 involved extremities) showing results with different forms of physiotherapy:

Results in extremities for each group regardless of degree of original involvement

Group and treatment	Total No.	Zero	Poor	Fair	Good	Normal
1. Immobilized 1-4 mos.; no physical therapy	53	19%	19%	15%	38%	19%
2. Immo. 1-3 mos.; phys-ther. 3-6 mos.	14	7%	14%	43%	36%	0%
3. Immo. 4-18 mos.; no phys-ther.	88	31%	18%	30%	19%	2%
4. Immo. 3-12 mos.; phys-ther. 8-24 mos.	70	23%	13%	24%	27%	13%
5. No treatment.	262	9%	12%	15%	47%	17%

From these figures McCarroll concluded that no particular form of physiotherapy produced outstanding results. In fact the group which received no treatment and began to walk without support as soon as they could get out of bed (Group 5) apparently had the greatest percentage of good results. It is McCarroll's belief that these results can be adequately explained by the underlying pathology--localized edema,

congestion, and cellular infiltration without actual destruction of the ganglion, but with an interference of their function. (This theory of pathological processes, however, as explained in the section on pathology, is probably not in accord with latest ideas on the subject.) Speaking of Sister Kenny's method of treatment he says: "From the standpoint of symtomatic treatment in the acute stage this method may offer a definite advantage, but it falls far short of supplying the answer to our problem in this disease. Sister Kenny prefers to consider the local muscle involvement as the primary pathological process and feels that the damage to the spinal cord is of seconday importance. She will admit, however, that spinal cord damage does exist in those patients who show persistent residual involvement. Muscles that have been previously considered paralyzed she prefers to consider as merely alienated because of the spasm existing in opposing groups as a result of their involvement, i.e., the muscles we have originally considered involved she considers satisfactory but merely alienated. She uses as proof the fact that these muscles later show evidence of return even though they may never attain more than a trace of power. She refuses to admit that this could be due to restitution

of function in a few of the ganglion cells. Since such ideas cannot be reconciled to our present knowledge of the proved pathological process in this disease, and since many of her patients also show residual paralysis and even flail extremities, I feel certain that this method in time will take its place among others offered by this field of physiotherapy as having been tried but found wanting.

"Physiotherapy will never prove to be the answer to our problem in this disease. The control of poliomyelitis will undoubtedly some day be brought about through prevention and not through its cure."

These articles brought immediate repercussions from many points. John Pohl wrote (35) "McCarroll has completely missed the point. Miss Kenny's discovery is definitely not that there is a cure or even a treatment for the paralysis of poliomyelitis but rather that there are muscle conditions which are far more damaging to the bodily mechanics if unrecognized than is paralysis. Dr. McCarroll could not possibly have found a satisfactory treatment for the disease of poliomyelitis if he has neglected to acquaint himself with the true symptoms of the disease."

A short time later Miss Kenny came to her own defence (36) with reports of the Orthopedic hospital,

New York (Dr. Robert Bingham) made in June 1942. Using the orthodox (12 patients) and the Kenny (24 patients) method the average duration of hospitalization was 16 months in the former and 6 months in the latter. In addition, patients with residual paralysis were 87% when treated by orthodox methods and only 33% with the Kenny method; patients with deformities were 75% by orthodox and 8% by Kenny method. To her these figures speak for themselves; however, it will be noted that this experiment was carried out on a comparatively few number of patients.

In 1937 Albert J. Schein made a study of results in 100 cases treated from the onset in orthodox fashion. (37) In this series 56% of trunk, 24% of lower extremity and 53% of upper extremity cases recovered entirely and nearly all patients somewhat. Only 4% of the cases were bedridden and totally non-ambulatory. It was his conclusion that careful after treatment (by orthodox methods) was of value in decreasing the incidence of deformities. Flail shoulder, scoliosis and foot imbalance were hard to avoid; gross hip and knee deformites almost entirely preventable.

Comparative studies of the two methods of treatment were made by Daly, Greenbaum, Reilly, Weiss, and Stimson. (38) Thirty-one cases of spinal paralysis

were treated by the orthodox method. Atrophy and deformities were noted in the second and third weeks. Seventeen were seen in 4-5 month check-ups and it was found that in every case there was a limitation of motion and muscle incoordination. Twenty-six paralytic cases were treated with the Kenny method and they found:

1. Spasm was relieved, return of unrestricted passive motion was weeks to months sooner than by the orthodox method..
2. Six to nine hot fomentations per 24 hours would give relaxation; three to four would not.
3. Tendency toward atrophy and deformity was minimal, condition of the skin was excellent.

Their comment was that "it is difficult to measure differences in amount of recoveries in the two groups of patients, but all those who have observed this group of patients and other groups treated in the orthodox manner are convinced that those who receive the Kenny treatment are better off in (a) comfort, (b) freedom from atrophy and deformity, (c) rapidity of recovery, and (d) possibly extent of recovery."

Arthur Steindler has made definite contributions

to the problem. (24) From 1940 to 1941 he treated 200 cases and found that in 25% contractions of some kind occurred. "In all these cases we found that the contractions developed in paralyzed muscles which had some form of prolonged immobilization, and they occurred as early as three and a half weeks or as late as eight months after onset of paralysis.....This same thing was true of patients receiving no treatment.....We found that return of strength is more likely to occur in a muscle which is kept at the physiologic optimum of circulation as well as muscle tone." This condition he points out should not be compared to long immobilization in a hip spica for Perthes disease, for instance; in this case stiffness disappears in a few weeks with active physical therapy and free motion in bed. In the case of poliomyelitis the condition is not due to involvement of periarticular structures, but rather due to a contracted muscle with pain and tenderness over its belly on flexion.

Steindler believes that splinting should still be used, but for shorter periods--only so long as pain and tenderness are present. He states that the "old idea of purely motor deficiency originating in the anterior horn cells must be changed. Motor dysfunction

seems to reach much higher and produce a state of confusion, which blocks the use of individual paralyzed muscles, which leads to adoption of complex substitutionary motions.....which become habitual and hard to overcome."

James M. Thompson (39) in speaking of the final analysis of the Kenny type of therapy says "true there will remain some cases with residual flaccid paralysis, but without stiff joints and contraction deformities. Kenny treated cases all have supple joints and remarkable flexability of their bodies. We did not see a single case of scoliosis in her group, nor were there any vasomotor changes so frequently observed with orthopedic treatment. Also, there will be some cases after two years of treatment that will, because of instability need braces for support and surgical stabilization. But, as Dr. Cole says, there are fewer cases of isolated paralysis than under the old form of treatment."

Phillip Lewin (32) tells the story of two patients sent to the hospital to go into the respirator. Sister Kenny asked for what was apparently the worst of the two cases. Her patient recovered. The other patient died within twenty-four hours. Similar stories have been repeated in the recent literature. Lewin states

that rigid splinting is not only "on its way out" but that it "is out." After two years of follow up studies on fifty-four cases he has observed complete recovery of the trunk, abdomen, back and neck.

Many other such glowing reports could be given on the treatment of poliomyelitis in the acute stage by the Kenny method. It is not easy to evaluate them at this time since her technique has been used for such a short time in this country and because of the comparatively few patients undergoing the treatment. Three thousand and two hundred cases of poliomyelitis have been reported for the year 1942 up to Dec. 12th. (50) Most of these were treated by the Kenny method or some modification of it. About 500 physicians, nurses and physical therapy technicians have been taught the Kenny method. In their opinion the "Kenny method is superior to immobilization. Figures are not available as to 'failures'."

There are other, perhaps more important factors to be considered in evaluating these reports. Statements like this can be found throughout the literature: Nonparalyzed cases represent at least 50% of the total incidence of the disease, and some men place the figure as high as 70%-80%. Wickman writes:

"Can we from any clinical data predict the course and termination of the disease in a given case? All clinicians agree that the intensity of the initial symptoms has no prognostic significance. Alarming initial symptoms may usher in a benign or an abortive attack; and a mild beginning may be the prelude of a stormy, perhaps of a fatal course."

In the survey of the international committee organized by Jeremiah Milbank this conclusion was reached. (2) "Probably not more than 25% of all cases of poliomyelitis that can be diagnosed develop paralysis. The case fatality is usually from 10% to 15%. As practically all fatal cases are in the paralyzed group, the number surviving with paralysis is much less than 25% of the total number of cases. Of the cases with paralysis a complete restitution of function will take place in a goodly number and a partial restitution in the remainder."

These facts again disprove the old adage that "figures never lie," for here we have variables that can affect the results of any type of treatment. Certainly the Kenny treatment will have to be tried many more times on a great many cases before its true worth can be made known.

Perhaps this method will take its place among

those that have been "tried and found wanting." However, no doubt, Sister Kenny has performed a service for humanity by shocking the medical profession out of its complacent attitude and in attempting to show its sins of commission and omission.

"Disease, being always an effect, is always in exact proportion to the sum of its causes, as much is the case of Spigelius, who dies of a scratch, as in that of the man who recovers after an iron bar has been shot through his brain. The one prevalent failing of the medical art is to neglect the causes and quarrel with the effect."

Oliver Wendell Holmes

Medical Essays, 1911

EPIDEMIOLOGY

This section on epidemiology has been included in this thesis, not because it applies particularly to what has been said so far, but because it is an interesting subject in itself and it aids considerably in an understanding of certain problems concerned with the disease.

This disease is sometimes classified under the title "Epidemic Poliomyelitis" serving to emphasize its epidemic nature. Although sporadic cases do occur from time to time, the diagnosis of "Poliomyelitis" should place the practitioner instantly on guard against an outbreak of the disease in the community.

The epidemiologic features of this disease are many and varied and might well serve as the main topic of discussion.

Before 1880 poliomyelitis was considered a curiosity among diseases of man, only a few sporadic cases occurred. But in this decade, by some quirk of fate, a change of character took place. It became a periodic scourge. (51) The first epidemic was reported in Sweden about this time. The first definite American epidemic occurred in the early '90s in New England.

Wickman is the name that stands out in this early period for his epidemiological studies. This man was an apt and most successful student of Medin. By careful study of epidemics in small villages, and by recognizing that so-called "abortive" cases could occur, he concluded that the disease was spread by person to person contact. He recognized the possibility of a healthy carrier; he also suggested and believed that this was a milk-borne disease. His contributions are significant as he was concerned only with the spread of the disease in man; the virus was as yet unknown and there was no experimental medicine.

Since the time of Wickman the etiological agent has been fairly well determined, the world has seen and had time to study many different epidemics, experimental medicine has added a great store of material; and the disease has been noted to have the following characteristics:

Geographical distribution. In general poliomyelitis is world wide in its distribution. (2) The most extensive epidemics have occurred in Scandinavia, Northern United States and Canada, Australia, and New Zealand. In general, it is s disease of the temporate climate.

Seasonal distribution. Poliomyelitis is now recognized as essentially a disease of summer and fall. The maximun case incidence is said to occur in August. (3) It is rather unusual for epidemics to occur before summer (usually about July) or to last beyond cold weather. Occasionally epidemics have been known to last on into the winter; and, a few winter epidemics have been reported although they are few in number.

Urban and rural distribution. Although there is some disagreement on this point by some investigators, most authorities believe that the disease is most severe or confined to small urban centers or rural districts, rather than the most densely populated areas. (52) It is, therefore, not only a summer disease but a rural disease--the explanation of which is by no means settled.

John Paul states (51): "One is still free to choose as to whether the influence of season or place exert themselves more as features which tend to spread poliomyelitis virus, or more as features which tend to affect the resistance of the host.....it seems logical that the rural prevalence of poliomyelitis, its summer prevalence and its prevalence in temperate zones may be linked together in some mysterious way.

The missing link or links probably represent environmental factors of considerable epidemiological importance. As yet one can only conjecture what they are, but it is logical to suppose that some of these factors have to do with facilitating the spread of the virus."

Age incidence of poliomyelitis. In the early years when this disease was first being described it was often, and still is, titled "infantile paralysis". This name is considered by many present day authorities as a misnomer; based on the fact that a goodly number of adult cases do occur and, in many cases, takes a non-paralytic or abortive form.

However, among all the controversial features of poliomyelitis one thing is agreed upon by one and all, that children are more susceptible to the paralytic form of the disease than are adults. (51) The reason for this is a controversial question, but it is considered by some to be the result of an "acquired" immunity during childhood. In support of this view Nicoll and others have shown statisticly that the more densely populated the community, the younger was the age of those affected. Thus it could be postulated that in cities opportunity for contact was high facilitating acquisition

of a subclinical immunity whereas opportunity for contact was low in the rural districts and, therefore, children did not have benefit of this type of immunity.

Ruhrah and Mayers stated in 1917 that adults are generally affected to the extent of about 10% in various epidemics--but in this there are great variations. (53)

Despite these facts that were brought out by early investigators there appears now to be a general increase in tendency for poliomyelitis to effect the higher age groups. It has also been noted that the age incidence of urban cases in New York City in the epidemic of 1931 was almost identical to that which had been noted in rural New York State in 1916. (2) This may be due to one or many factors such as (a) an increasing susceptibility, (b) inclusion of a greater number of abortive cases, (c) greater or better means of transmission.

Poliomyelitis has been known to frequently occur within the first year of life; 23 such cases out of 228 were reported in England and Wales in 1918. Cases have been reported as occurring 12 days, 3 weeks, and one month after birth. Two cases of suspected intrauterine infection have also been reported. (3)

The age of the patient often has an important influence on fatality rate. In a series of cases reported by Wickman the case mortality was 11.9% for children under 11 years and 27.6% for persons between ages of 12 and 32 years. (54)

Mortality. The statistics vary considerably with each epidemic, but according to McNalty (3) the average case fatality is from 10% to 20%, depending on the recognition of mild or aborted cases. However, the extremes show a marked fluctuation from 1% to 40%.

Incubation period. Experimentally this period has ranged from 3 to 46 days; the average is usually 11. In 1942 Albert Casey (55) made a study of the incubation period in 37 instances of epidemic human poliomyelitis. He found that the period varied from five to 35 days and averaged 12 to 11 days. The incubation period was calculated from exposure to onset of prodromal symptoms. He noted that these figures were compatible with the incubation periods in eleven cases taken from the literature; and, in addition with the incubation period in ten rhesus monkeys or chimpanzees inoculated with freshly isolated human strains. Here is another instance pointing out the similarity between the disease in man and that of the monkey inoculated experimentally.

Immunity and susceptibility. Immunity, of course, can be divided into three types: Natural, Active acquired, and Passive acquired. Considerable work has been done clinically and experimentally on this particular subject, and, as will be pointed out, from a therapeutic standpoint not much has been accomplished.

Natural Immunity may be defined as a state of resistance to infection not dependent upon a previous spontaneous or experimental contact with infectious agents or their antibodies. (51) All races of human beings are susceptible to poliomyelitis. Different species of monkeys have shown a difference in susceptibility and some lab animals have been considered refractory to infection by the virus (horses, sheep, calves, dogs, rabbits, guinea pigs, rats, mice, and chickens). Armstrong has adapted at least one strain of poliomyelitis virus to the Eastern Cotton rat (56). This work has been confirmed. Since the cotton rat is much cheaper for experimental purposes than the monkey perhaps this will lead the way to more extensive poliomyelitis research.

In most infectious diseases the patient is in a weakened condition--generally "run-down". Aycock

in 1937 pointed out, however, "the tendency of poliomyelitis to occur in children who are large, healthy, and well nourished." (57) Jungeblut attempted to show by experimentation on monkeys that Vitamin C deficiency would increase the susceptibility of the animal to poliomyelitis. (58) This fact, however, has not been corroborated and Sabin has recorded results of just an opposite nature. (59) Howe and Bodian (17) proved the virus had no effect on previously degenerated neurons which in normal state would ordinarily be susceptible. It seems, therefore, that the "disease is not influenced by states of malnutrition.....the virus is not only intimately associated with host cells, but it appears that they have adapted themselves to normal cells. " (51)

Active Immunity is defined as a state of resistance to infection engendered by a normal spontaneous attack of an infectious disease, by the experimental or intentional production of the disease or a modified form of it, or by injection of vaccines. (51) The blood serum of convalescents, both human and experimental, will neutralize the virus of poliomyelitis. At first this was taken as evidence of "acquired immunity" on the part of the individual; and Flexner and Lewis in 1910 (60) believed they

had proved that monkeys having had the disease were immune to further inoculation. This fact was sited as the reason why communities where poliomyelitis had occurred seemed to escape or were only slightly affected in subsequent outbreaks. Even as late as 1932 this statement can be found in one of the classical books on poliomyelitis ".....a relatively high degree of permanent immunity to the virus of poliomyelitis has been shown to follow infection with virus. This is evidenced by resistance to reinfection in the experimental, and rarity of second attacks in human cases; and by demonstration of power of blood serum of convalescent human beings and of monkeys to neutralize the virus in vitro." (2)

However, it was presently shown that this so-called protective power is present in the serum of individuals who have had suspected abortive attacks and sometimes in the serum of normal individuals, both those who have been in contact with poliomyelitis patients and those who are not known to have had such contact. "Within the last 15 years it has been shown that 75% to 85% of all normal adult population in various parts of the world, regardless of occurrence of obvious poliomyelitis cases, possess neutralizing antibodies against poliomyelitis virus."(51)

In addition, it was proven quite definitely by experimentation that monkeys even with high titer would usually succumb to subsequent inoculations intracerebrally. (61) It is only natural therefore that Howe and Bodian (17) should make the following statement: "As regards humoral immunity.....prevailing view that the virus of poliomyelitis is a poor antigen and that circulating antibodies, actively or passively produced, exert but little if any protective effect on the central nervous system." Doubt has been expressed by many authorities that these circulating antibodies are in any way a true measure of immunity to poliomyelitis. Another factor in this jig-saw puzzle, is that many strains of poliomyelitis occur, with a great many differences between individual strains immunologically speaking.

The presence of these neutralizing bodies in the serum of persons recovered from an attack of poliomyelitis forms the basis for convalescent serum therapy. However, there is no unamimity as to results or best method of administration and most physicians if they do use serum, do so because they feel it will do no harm. Occasional reports such as this one are made; "less than 2% of some 200 consecutive patients with poliomyelitis treated with convalescent serum had residual paralysis compared with 20% found among

untreated patients." (62) However, as a rule lack of good controls nullifys most of the work done with this means of therapy.

The problem of immunity has been thrown into greater confusion by several other factors. In the first place, although quite rare, it is known that second attacks occur in human beings. Lucas and Osgood in 1913 (63) recorded a case of poliomyelitis occurring two years after the initial attack. Other such cases have been reported in the literature as occurring three years, six years, and even sixteen years after the initial attack. In cases of a second attack after a very short interval it would seem that the original infection had flared up again. Just how long immunity may last following an attack of the disease has never been determined.

Older textbooks often make the statement that the disease seldom attacks more than one member of the family. For instance, MacAusland (5) states: "Occurence of more than one case in a family is uncommon. In the epidemic of 1916 only 4% of 8,635 families attacked suffered more than one case." John R. Paul represents a newer concept of the disease, however, when he states: "What the textbook authors implied was that the disease seldom paralyzes

more than one member of the family, for multiple cases within families are frequent and often explosive in character." By this he means that attack rates for poliomyelitis would be much higher if all epidemics were carefully scrutinized for cases of the abortive form--rates would perhaps even reach those of "highly contagious diseases."

However, wide variations in individual susceptibility occur, even within one family. Children occupying the same bed with one that contracts the disease often will show no signs or symptoms of infection. This, of course, brings up another point--means of transmission--a subject to be discussed later on.

Varying reports have been forthcoming on experimental attempts at active immunization by vaccines. John A Toomey summarizes the subject nicely when he says: "Artificial immunity may be established against poliomyelitis if the living virus is injected as the antigen; but many monkeys acquire the disease during this process of active immunization.....Since there is no way of distinguishing susceptible persons from those who are immune and since the factors which predispose toward the disease are unknown, it is obvious that vaccine antigens must

be safe. Thus they cannot be viable and paradoxically, if they are not viable they seem to have no value. In fact, not only may it be inadvisable from the practical standpoint to immunize actively with virus antigens but it has been suggested that such vaccines may even be dangerous to human beings." (61)

Passive immunity is a state of resistance to infection produced in a normal individual or animal by the parenteral administration of serum from actively immune individuals possessing circulating antibodies. (51) As suggested above, attempts to passively immunize individuals in field tests and experimentally in animals have, in general, been considered failures. The main explanation given in the literature for this fact is that antibodies, as a rule, do not enter the cells; they produce a humoral antibody effect probably, but no tissue immunity. This phenomenon was proven experimentally. A vaccine virus within cells multiplies and produces lesions, even though the infected tissues are cultivated in a potent antivaccine plasma. (64) A second possible explanation lies in the fact that there is, apparently, a barrier between brain and blood which prevents the passage of immune substances against poliomyelitis from the blood into the central nervous system. (51)

Portal of entry. The portal of entry of the poliomyelitis virus in man is another problem over which there is much discussion, but nothing definitely known. It has been found experimentally that a typical disease picture can arise from innoculations in the tonsilo-pharyngeal region, intracutaneously, subcutaneously, intravenously, or into the intestines, and intranasally. However, without any trauma whatever and by transitory contact alone, only the latter has yielded regular results. (65) Therefore, the intranasal route along with the gastrointestinal route, have been the only two "portals" seriously considered by most investigators---since they more closely represent what appears to be the normal.

The theory of an intranasal route of infection is supported by the fact that after olfactory tracts are cut monkeys no longer are infected by this particular route. (19) This fact has been confirmed by many workers. In addition, pathological changes can be detected in the olfactory bulbs. Knowing that the virus must pass via the medulla down the cord, if it enters by this portal, then why should paralysis often show up first in the legs? Either the portal of entry is by the intestinal tract or some other factor enters the picture. This factor according to

Schultz is a difference in tissue susceptibility. He states: "Virus may be present in the cord in fairly high concentrations even without paralysis .....Our investigations show the cervical cord may have high concentrations of virus in advance of its appearance in the lumbar cord. Therefore motor nerve cells of the lumbar cord are more susceptible for some reason". (19)

He goes on to say, "The propagation of virus in the central nervous system is by neurons. Therefore, why isn't the initial infection in the only place of the body where neurons are exposed to the external surface."

In man no definite lesions have been found in the olfactory bulbs. This is, of course, a point in favor of some other route of infection. However, it is possible, according to some authorities, that in man the lesions may have been missed. Another question that must be answered is whether it is possible that the virus may spread without leaving a "trail".

John Toomey is perhaps the most vociferous advocate of the theory that poliomyelitis is a gastrointestinal disease. He bases this idea on the following line of reasoning. (61) Poliomyelitis virus has

been consistently recovered from stools of convalescents. Section of olfactory bulb does not protect animals from inravenous innoculation. Chemoprophylaxis does not prevent poliomyelitis with innoculations either intravenously or by gastro-intestinal routes. The disease occurs at the same time of the year as typhoid, another GI disease. The disease can spread along sympathetic fibers from the gastro-intestinal tract. (66) In addition, the clinical symptoms of the disease can be best understood if spread is supposed to pass from the GI tract. Schultz believes that Toomey's experiments with this route of infection are much too drastic. "In general it is well understood that any method of forcibly introducing virus into the tissues may result in infection." (19)

It has been suggested that the presence of the virus in the GI tract may be the result of continued swallowing of virus contaminated secretions. Sabin adds this point: "Until it had been shown, therefore, that monkeys whose olfactory pathways have been blockaded by chemical means or interrupted surgically can develop poliomyelitis after feeding of the virus, There is no real experimental basis for considering the oral or gastro-intestinal routes as sites from which the virus invades the nervous system". (65)

Exactly this thing was done by Howe and Bodian (17) on chimpanzees--the animal contracted poliomyelitis. As a result of this experiment they made the following statement: "By analogy with the chimpanzee, which appears to react to poliomyelitis in a fashion practically identical with that seen in man, one can merely say that the virus in man probably gains access to the nervous system from the oral cavity, pharynx, or intestinal tract."

Despite work that has been done on sites of infection and the arguments for either intranasal or gastro-intestinal routes, it must be concluded that we have no direct data on actual portal of entry in the human body. In the light of a recognized association between bulbar paralysis and tonsillectomy the nerves of the superior pharynx may serve as a pathway to the central nervous system; this fact may be of further significance since the pharynx is one of the principle sites outside of the nervous system where the virus is found. (67)

Transmission. This factor, of course, depends on the portal of entry. If the nasal mucosa is the portal virus may be spread via droplet infection, dust, carriers, etc. If the GI tract is the portal of entry fecal contamination of food and water may

play a part. The idea has been expressed in the literature from time to time that poliomyelitis is a milk-borne disease.

Another possibility of transmission that has not been completely explored is by means of insects. In a recent study of outbreaks of the disease in Atlanta and Cleveland, Sabin and Ward have been able to demonstrate poliomyelitis virus in eight of fifteen batches of trapped flies. Incidentally, these flies were not caught in the vicinity of "privies". They concluded from their work, "A search for reservoirs in lower animals is worthwhile; but it appears the epidemiology of poliomyelitis is more like typhoid or dysentery in which the chief reservoir is infected excreta with direct and insect spread". (68)

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