Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Mechanism of Muscle Spasm In Poliomyelitis

Volume XIV | Friday, June 4, 1943 | Number 28

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XIV | Friday, June 4, 1943 | Number 28

INDEX

Published for the General Staff Meeting each week
during the school year, October to June, inclusive.

Financed by the Citizens Aid Society,
Alumni and Friends.

William A. O'Brien, M.D.

I. LAST WEEK

Date: May 28, 1943

Place: Recreation Room, Powell Hall

Time: 12:15 to 1:25 p.m.

Program: "Primary Resection for Lesions of the Colon, Rectosigmoid and Rectal Ampulla"
Owen H. Wangensteen

Discussion
O. J. Campbell
Irvine McQuarrie

Attendance: 100

Gertrude Gunn,
Record Librarian

- - -

II. TECHNICAL PERSONNEL IN MINNESOTA HOSPITALS - 1942

	Full Time	Part Time
Nurse Anesthetists	129	67
Laboratory Technicians	210	65
X-Ray Technicians	140	65
Dietitians	130	21
Physical Therapists	38	24
Pharmacists	42	18
Medical Record Librarians	64	32
Other Librarians	20	17
Medical Stenographers	108	28
Occupational Therapists	39	6
Dental Hygienists	15	17
Social Service Workers	61	3

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III. APPROVED SCHOOLS FOR CLINICAL LABORATORY TECHNICIANS IN MINNESOTA

Name & Location of School	College Affiliation	Maximum Enrolment
St. Luke's Hospital, Duluth	Hamline University	10
St. Mary's Hospital, Duluth	College of St. Scholastica	17
Minneapolis General Hospital, Minneapolis	University of Minnesota	18
Northwestern Hospital, Minneapolis		4
Swedish Hospital, Minneapolis	Gustavus Adolphus College	8
University Hospitals, Minneapolis	University of Minnesota	75
Ancker Hospital, St. Paul	University of Minnesota	6
Chas. T. Miller Hospital, St. Paul	Macalester College	8

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IV. APPROVED SCHOOLS FOR PHYSICAL THERAPY TECHNICIANS IN MINNESOTA

University of Minnesota,
Minneapolis, Minnesota

Mayo Clinic, Rochester, Minnesota

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V. THE MECHANISM OF MUSCLE SPASM IN POLIOMYELITIS

Herman Kabat
Miland E. Knapp

1. Clinical Description of Muscle Spasm

One of the fundamental innovations of the Kenny concept of poliomyelitis[1,2] is the emphasis placed upon "muscle spasm." This is a condition characterized by shortening of the skeletal muscles which appears in the acute stage of the disease and may persist for months or years. It may be identified in the early stages by characteristic deformities produced, by prominence of tendons, by flatness and narrowing of muscle bellies, by increase or decrease in the number and depth of skin folds or creases, by limitation of range of passive joint motion, and by other evidences of muscle shortening. The muscles in spasm are painful to stretch or pressure and may often be weak or even completely paralyzed. Spasm may be the direct cause of loss of function in opposing muscles because of the mechanical interference with motion produced by the shortened painful muscle. Relief of this condition may lead to increased active as well as passive motion and increased power in apparently paralyzed muscles by removal of this mechanical obstruction in permanent deformity as well as dysfunction of antagonistic and synergistic muscles.

This phenomenon, which is apparently present in every case of poliomyelitis is essentially an abnormally increased tonus of the skeletal muscles. It is not, however, a relative increase in tonus due to weakness of opposing muscles as is commonly thought. Clinical observations have shown that there is no constant relationship between spasm and paralysis caused by motor denervation. Examples have been seen where "spasm" is present in extremities where all active motor power has been lost in all muscles, both those that are shortened and those that are lengthened. Other cases show severe spasm in muscles whose opponents retain normal power.

Two types of "muscle spasm" may be identified.

A. Muscle hypertonus. This is a markedly increased muscle tonus which may persist without relaxation for months or years. Hypertonus not only causes limitation of joint motion but also frequently causes gross deformities. In the gastronemius-soleus group it causes so-called "foot drop," more accurately named "foot retraction." In the upper trapezius, it causes an elevated shoulder which may in turn be the cause of a scoliosis. In one quadratus lumborum it results in a unilateral lordosis with pelvic tilt and apparent leg shortening. Lordosis or scoliosis may result from hypertonus in the muscles of the back.

B. Hyperirritable stretch reflex. In this type, there is a limitation of the range of passive motion which is variable in degree. There is no resistance to passive stretch of the involved muscle until the point of limitation of motion is approached, when further passive motion becomes impossible due to the resistance of the strongly contracted, painful muscle. A slight decrease in stretch of the muscle, produced by a return of a few degrees toward the resting position, relieves the pain and terminates the strong contraction of the muscle. This appears to be a stretch reflex, whose "trigger mechanism" is set at a much lower level than normal. Since there is no resistance to passive stretch through a certain range of motion, this phenomenon differs from the rigidity in Parkinson's disease and the spasticity in hemiplegia.

Hyperirritable stretch reflexes are observed in poliomyelitis most commonly in the hamstring, quadriceps, adductor, pectoralis major and latissimus dorsi muscles. It may also occur in the triceps brachii, wrist flexors, and other muscles.

Muscle hypertonus and hyperirritable stretch reflex are closely related and may have the same fundamental mechanism.

They both appear to be dependent upon hyperirritability of the spinal mechanism for proprioceptice reflexes and muscle tonus.

The distribution of muscle spasm varies in different cases and may not be symmetrical bilaterally. It may be localized to a single muscle or group of muscles in a limb. It may involve both agonist and antagonist about a particular joint. Many of the milder cases of poliomyelitis show muscle spasm but no permanent paralysis or atrophy. On the other hand, spasm may be severe in muscles which remain paralyzed and undergo atrophy.

In a particular muscle, the spasm has a number of characteristic clinical features:

(1) There is limitation of the range of passive motion, variable in degree, but usually marked. This is observed in both "hypertonus" and "hyperirritable stretch reflex."

(2) The muscle is painful to stretch or pressure. While spontaneous pain usually disappears following the acute stage of the disease, the tenderness to stretch is present as long as the muscle spasm persists, frequently for many months.

(3) The shortening of the muscle in the acute stage may be increased by attempts at forced motion, rough handling, electrical stimulation, or anything that increases pain.

(4) Muscle spasm may be remarkably persistent. Untreated muscle spasm often persists for months or years. Even with active therapy beginning in the acute stage, spasm may not be relieved for many months. On the other hand, in milder cases, spasm may subside spontaneously.

(5) Deformity frequently results from marked hypertonus. Examples are: "foot retraction" from spasm in the posterior calf muscles; elevated shoulder from spasm in the upper trapezius; apparent leg shortening from spasm in the quadratus lumborum muscle, etc.

(6) If the spasm persists for a long period of time, irreversible contracture may result.

(7) The muscle in spasm is often weak and sometimes paralyzed. Voluntary contraction of the muscle may be decreased or absent. This is an interesting paradox: a muscle strongly contracted in tonus and proprioceptive reflexes which is weak or incapable of voluntary contraction.

(8) Many muscles in spasm do not undergo permanent atrophy and recover function completely with therapy. Muscles in spasm may, however, even after relief of the spasm, remain paralyzed and undergo marked atrophy.

(9) Spontaneous fasciculation is not characteristic of muscles in spasm.

2. The Neurogenic Basis of Muscle Spasm

In an attempt to determine the mechanism of muscle spasm in clinical cases of poliomyelitis, we carried out a number of experiments on patients at various periods after the onset of this disease.* Five patients were subjected to intravenous pentothal anesthesia in order to produce loss of consciousness and elimination of pain. Muscle spasm in various muscle groups was determined quantitatively by measuring the angle of limitation of motion with a goniometer (Table I.) In some cases, no significant change was noted. In other cases, a slight to moderate decrease in limitation of motion was observed. In no instance, however, was there complete relaxation of the muscle spasm under pentothal anesthesia. These results suggest that pain may be a factor tending to increase muscle spasm in some

*These experiments were carried out with the cooperation of Dr. Scott M. Smith, Dr. N. Sonnesyn and Dr. R. T. Knight of the Department of Anesthesia, University of Minnesota Hospitals. Their assistance and cooperation are gratefully acknowledged.

Table I

EFFECT OF ANESTHESIA ON MUSCLE SPASM IN POLIOMYELITIS

	Case	Time Since Onset	Type of Anesth.	Ham. R	Ham. L	Gastroc Soleus R	Gastroc Soleus L	Soleus R	Soleus L	Quad. R	Quad. L	Abd. Legs
												both legs
Before	1	2½ mos.	P	50	55	100	105					55
During	"	"	"	60	60	100	105					60
Before	"	"	S	45	50	100	105	95	90	75	75	60
During	"	"	"	75	70	95	95	90	95	60	60	70
Before	2	1 mo.	P	30	30	115	120	110	115	85	85	35
During	"	"	"	55	50	115	120	110	115	50	45	55
Before	"	"	S	30	25	125	130	115	120	80	85	35
During	"	"	"	70	75	100	105	95	95	*	*	70
Before	3	2 mos.	P	35	40	105	100	100	90	45	40	55
During	"	"	"	65	55	105	100	95	90	*	*	65
Before	"	"	S	45	40	110	105	100	95	55	50	45
During	"	"	"	80	80	100	95	95	95	*	*	70
												R L
Before	4	3 wks.	P	40	40	105	100	85	80	90	90	35 45
During	"	"	"	45	50	105	105	95	85	75	80	35 40
Before	5	2 mos.	P	35	35	115	115					25 25
During	"	"	"	30	30	105	110					25 25
												both legs
Before	6	14 mos.	S	60	50	100	95	100	95	120	120	45
During	"	"	"	60	60	100	95	95	95	115	120	50
Before	7	14 mos.	S	55	55	120	115	105	115	90	75	60
During	"	"	"	55	55	115	110	110	95	80	70	65

*Heel touches buttock

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cases, but that it is not of primary importance in its mechanism.

In five patients, spinal anesthesia was performed with injection of 100 to 150 mg. of procaine intrathecally. In acute and subacute cases, spinal anesthesia led to relaxation of muscle spasm to a marked extent (Table 1). On the other hand, in two cases fourteen months after the onset of the disease, spinal anesthesia produced practically no change in the limitation of passive motion (Table I). At our suggestion, Dr. Malcolm Cook, Department of Pediatrics, Washington University, St. Louis, carried out spinal anesthesia on two acute cases of poliomyelitis within two weeks after the onset of the disease and observed elimination of muscle spasm.

In two cases, measurements were made of limitation of passive motion at various periods after introduction of procaine into the spinal canal. With complete sensory anesthesia but incomplete block of the motor nerve roots, the relaxation of muscle spasm was not as great as was observed later with complete motor block (Table II).

In one case, Beta-erythroidine hydrochloride was injected intravenously 8½ months after onset of poliomyelitis. Marked relaxation of muscle spasm and of pain on passive motion was observed at the height of the drug effect. This drug has a curare-like action in blocking the myoneural junction. It is of considerable interest that in the same patient (DH) at 14 months after the

Table II

PROGRESSIVE DECREASE IN MUSCLE SPASM WITH MORE COMPLETE BLOCK OF MOTOR NERVE ROOTS FOLLOWING INTRATHECAL ADMINISTRATION OF PROCAINE

Patient Age 12 Time since onset of 2 mos	Time	Sensory Level	Ham R	Ham L	Soleus R	Soleus L	Quad. R	Quad. L	Abduction both legs
Before anesthesia	3:55		45	45	110	105	55	55	45
Intrathecal procaine 100 mg.	4:05								
Sensory anesthesia	4:12	T 10	60	60	100	100	*	*	65
Motor block	4:20	T 4	70	70	105	100	*	*	70
Motor block	4:32		80	80	100	95	*	*	70
Beginning recovery	4:44		70	75	105	105	*	*	70

*Heel touches buttock

onset of the disease, spinal anesthesia caused practically no change in limitation of passive motion. Correlated with this observation was the fact that hot foments had caused little improvement in the later stages in this patient. This suggests that changes in the muscle may be set up by persistent spasm, which eventually result in irreversible contracture.

These experiments indicate that the mechanism of muscle spasm in poliomyelitis is primarily neurogenic. Muscle spasm is apparently due to an increased discharge of motor nerve impulses from the spinal cord to the affected muscles. This is in keeping with the observation[3] that muscles in spasm in poliomyelitis exhibit increased electrical potentials. Elimination of this excessive discharge of motor impulses by spinal anesthesia or by block at the myoneural junction is effective in relaxing the spasm. While pain may be a factor of some importance in certain cases in increasing limitation of passive motion, it is not of primary significance in the etiology of spasm. It appears, rather, that the spasm produces the pain.

A neurogenic mechanism for muscle spasm is in accord with the accepted conception of poliomyelitis as a virus disease of the gray matter of the spinal cord. The virus is highly neurotropic and is selective in its attack on nerve cells of different types.[4] Further more, there is no inflammation or other pathological change in the muscle tissue in the acute stage of the disease and virus is absent from the muscles.

It is unlikely that the associated meningitis can explain the persistent muscle spasm observed in poliomyelitis. The meningitis in this disease is mild, apparently lasts for only a short time and virus is absent from the spinal fluid. Indeed, the suggestion has recently been made by Sabin[5] that the inflammatory cells in the spinal fluid in poliomyelitis may not represent a true meningitis but rather an overflow of cells from the parenchymal inflammation of the spinal cord. Can the muscle spasm in poliomyelitis, which persists for months or years and which is more widespread than in severe meningitis, result from the brief mild meningeal irritation in this disease?

One is led to consider, therefore,

that the muscle spasm as well as other signs and symptoms involving skeletal muscle in poliomyelitis may all be secondary to attack by the virus on neurons in the central nervous system.

3. A Theory Concerning the Pathological Basis of Muscle Spasm.

What is the pathological lesion in the spinal cord which is responsible for the production of muscle spasm? A so-called "irritative lesion" of anterior horn cells would be difficult to reconcile with muscle spasm persisting for months after the acute infection. The persistence of the spasm suggests that the mechanism is more likely to be a "release phenomenon": a release from inhibition producing hyperirritability of the spinal mechanism for muscle tonus and proprioceptive reflexes.

Most pathologists emphasize injury and destruction of the large motor neurons in the anterior horn by the virus as the characteristic lesion in the spinal cord in poliomyelitis. There are a number of reasons for believing that involvement of anterior horn cells is not the pathological basis of muscle spasm. In order to produce increased muscle contraction as in spasm, the anterior horn lesion would have to be "irritative," which is difficult to correlate with the persistence of spasm. Furthermore, destruction of anterior horn cells is invariably followed by denervation atrophy of muscle, while permanent atrophy does not occur in many muscles in spasm in poliomyelitis.

In order to determine the relationship of anterior horn cell pathology to muscle spasm, measurements were made of the chronaxie of muscles in spasm in 14 of our poliomyelitis patients by Dr. J. Moldaver, of the Neurological Institute, New York. (By measuring the chronaxie of muscles in poliomyelitis, one may determine the degree of denervation of the muscle and hence reconstruct the anterior horn cell pathology resulting from the virus infection.) It was found that many muscles in spasm in poliomyelitis have a normal chronaxie. Other muscles in spasm have a greatly increased chronaxie, suggesting marked anterior cell destruction. Still other muscles in spasm show partial neuro-muscular degeneration which is apparently reversible. One may therefore conclude that there is no correlation between anterior horn cell destruction and muscle spasm in poliomyelitis. If we make the logical assumption of a unitary mechanism for muscle spasm in poliomyelitis, then an anterior horn cell lesion is ruled out by the fact that many muscles in severe spasm appear to have perfectly normal anterior horn cells.

Since an anterior horn cell lesion does not form a reasonable mechanism for the phenomenon of muscle spasm, one must cast about for another explanation. An enlightening suggestion has come from animal experimentation.

Muscle spasm similar in many respects to that observed in clinical poliomyelitis has been produced in dogs by a temporary arrest of the circulation to the spinal cord.[6] Following arrest of spinal cord circulation for 45 minutes, the dog develops muscle spasm which persists unchanged for weeks. This muscle spasm is characterized by marked resistance to passive movement, pain on stretch of muscle and pain on deep pressure, as well as loss or decrease of voluntary and reflex movement. Muscle spasm was observed in the posterior neck muscles, back, hamstrings, quadriceps, pectorals, latissimus dorsi, biceps, triceps, trapezius, etc., and was greater in the proximal than in the distal muscles. Both agonist and antagonist were sometimes involved. In the forelimbs, reflex and voluntary movements were absent; in the hindlimbs, voluntary movement was absent but reflex movement was present. Sensation, autonomic functions and respiration were normal. Examination of the spinal cord revealed a lesion localized to the region between the anterior horn and the posterior horn with marked injury and neuronophagia of the small internuncial neurons. The large motor neurons in the anterior horn, the preganglionic sympathetic neurons, cells of nucleus dorsalis and of the posterior horn were unaffected. The internuncial cell lesion was more severe in the brachial enlargement than in the lumbo-sacral enlargement

of the spinal cord, correlating with the greater severity of muscle spasm in the forelimbs. Hypertonus has also been observed in cats as a result of a similar lesion.[7]

A lesion of internuncial neurons with relatively normal anterior horn cells would serve admirably as the pathological basis of muscle spasm. Most of the synapses on the motor neurons of the anterior horn originate from the internuncial neurons.[8] Nerve impulses in the pyramidal tract[9] and other tracts, as well as in many afferent nerve fibers are relayed to the motor neurons of the anterior horn through the internuncial cells. The internuncial neurons thus form the switchboard mechanism controlling the motor neurons. It is a reasonable hypothesis that a lesion of internuncial nurons may therefore produce a localized muscle spasm on the basis of release of anterior horn cells from inhibition. The characteristics of muscle spasm from an internuncial lesion would, however, differ from a typical upper motor neuron spasticity, because of the multiplicity of connections of the internuncial cells.

Thus far, it has been shown that muscle spasm may apparently be produced in animals by an internuncial cell lesion as a release phenomenon. One must still demonstrate that such a lesion occurs in human poliomyelitis and is correlated with muscle spasm. In analyzing the pathology of the spinal cord in poliomyelitis in man, one must keep in mind that only about 5 to 10% of the cases die in the acute stage and it is this restricted group of the most severe cases which have received most attention from pathologists. Even in these very severe cases, however, it has been possible to find some which show a lesion of internuncial cells with relatively little damage to anterior horn cells.[10] In going over sections of the spinal cord from cases that died of poliomyelitis in the acute stage at Minneapolis General Hospital, at the University Hospital, Ancker Hospital, and at the Mayo Clinic, twenty-six out of sixty-eight cases showed predominantly inflammation and neuronophagia in internuncial neurons with relative sparing of anterior horn cells. In the remainder of the 68 cases examined, marked destruction of anterior horn cells as well as internuncial cells was observed. In one case, which died in 1942, there was a record of severe muscle spasm correlated with a lesion of internuncial neurons and normal motor neurons in the anterior horn of the spinal cord.

Of considerable interest in this connection in the recent report of Minckler[8] on pathologic akterations in the synapses of the human spinal cord in various neurological diseases using special staining methods. He studied eight cases of poliomyelitis and observed marked degeneration of the synaptic endings on the surface of the anterior horn cells. He proved that injury to anterior horn cells themselves had no effect on synapses on their surfaces and concluded that the degeneration of synapses on the anterior horn cells was due to injury to the internuncial neurons. It is interesting to note that Minckler also reports two cases of tetanus in which the synaptic endings had practically disappeared from the anterior horn cells. Tetanus is, of course, characterized by severe "muscle spasm."

The observations of Minckler and our own studies on human pathology suggest that internuncial neurons appear to be frequently involved in poliomyelitis and that even in the severe cases which die in the acute stage, the internuncial lesion may be more marked in some sections of the spinal cord than the anterior horn lesion. One may, therefore, assume that in the majority of cases which survive the acute infection, the internuncial lesion may actually predominate. In accord with this view is the clinical observation that mild cases of poliomyelitis have muscle spasm but no paralysis, suggesting that internuncial neurons may perhaps be more sensitive to the virus of poliomyelitis than anterior horn cells in the spinal cord.

One question that remains is why the internuncial lesion should eliminate inhibitory mechanisms without blocking impulses for excitation of muscle tone and stretch reflexes. A recent observation of Lloyd[11] helps to explain this

phenomenon. He has demonstrated that the proprioceptive reflex arc of the extensor muscles in the cat is a two-neuron arc, with no interposed internuncial neuron. Therefore the nerve impulses for excitation of tonus and stretch reflexes would be conducted through the internuncial region only by nerve fibers. Since nerve cell bodies are susceptible to the virus of poliomyelitis but nerve fibers do not appear to be attacked directly by the virus, excitation of the proprioceptive reflexes would not be blocked by virus involvement of the internuncial neurons. On the other hand, since impulses for inhibition of tonus and stretch reflexes are conducted through the internuncial neurons, the attack by the virus on these neurons would lead to an exaggeration of proprioceptive reflexes, as a result of release from inhibition.

From these observations, a conception of spinal cord pathology emerges which is characterized by two types of lesions: an anterior horn cell lesion resulting in motor denervation with muscle atrophy; and an internuncial lesion resulting in muscle spasm. The two lesions show great variability in their distribution, so that one may see, in different muscles, muscle spasm with or without denervation atrophy and motor denervation with no muscle spasm. It appears probable that some functional anterior horn cells must remain to produce clinical muscle spasm.

Another possible mechanism for muscle spasm that suggests itself would be based on acetylcholine contracture. It is well known that denervated muscle is hypersensitive to acetylcholine.[12] Acetylcholine readily produces contracture in denervated skeletal muscle.[13] The possibility that acetylcholine contracture might play a role in muscle spasm in poliomyelitis has been investigated by the study of the effects of prostigmine on muscle spasm. This drug exaggerates the action of acetylcholine by inhibiting cholinesterase at the myoneural junction. If prostigmine resulted in an increase in hypertonus, one would have to consider acetylcholine contracture as a possible factor in the mechanism of muscle spasm. However, prostigmine produced relaxation of muscles in spasm in patients with poliomyelitis.[14] This observation suggests that acetylcholine contracture is not the basis for muscle spasm in this disease.

The relaxation of muscle spasm in poliomyelitis by prostigmine is apparently based on the action of the drug on the spinal cord, inhibiting the centers for proprioceptive reflexes.[14,15] There is evidence that the locus of the spinal action of these drugs is proximal to the anterior horn cells, presumably on the internuncial neurons.[16] The relaxing action of prostigmine on muscle spasm provides additional support to the theory that an internuncial lesion, by interfering with inhibition at synapses on the anterior horn cells, is responsible for muscle spasm in poliomyelitis.

Interference with the conduction of impulses through the internuncial switchboard to the motor neurons in the anterior horn may also help to explain the phenomenon of "incoordination" in poliomyelitis. This appears to be essentially a disorganization in the flow of impulses to anterior horn cells, resulting in involuntary contraction of antagonistic and synergistic muscles. There is also disorganization in discharge of motor impulses to muscle fibers within a single muscle, resulting in fascicular twitchings in voluntary and reflex contraction of the muscle. Incoordination is decreased by prostigmine[14] and by muscle re-education.

Summary

1. Muscle spasm in poliomyelitis is of two types:
   A. Hypertonus
   B. Hyper-irritable stretch reflex
2. The clinical characteristics of muscle spasm are described.
3. Muscle spasm is relaxed temporarily by spinal anesthesia or block of the myoneural junction in acute and subacute poliomyelitis. Intravenous pentothal decreases muscle spasm moderately in some cases and is in-

effective in other cases.

Muscle spasm in poliomyelitis has a neurogenic mechanism and is apparently the result of an increased discharge of nerve impulses through the motor neurons.

4. Evidence is presented to support the theory that the pathological basis of muscle spasm in poliomyelitis is a lesion of internuncial neurons in the gray matter of the spinal cord. The muscle spasm is produced as a result of release of proprioceptive reflexes from inhibition.

   A. Muscle spasm has been produced in the experimental animal by temporary arrest of circulation in the spinal cord. Correlated with this muscle spasm, the spinal cord showed a lesion localized to the internuncial neurons while the anterior horn cells appeared normal.

   B. In 68 cases of poliomyelitis, review of the spinal cord pathology revealed an internuncial lesion in almost every case. Twenty-six cases showed an internuncial lesion with relatively normal anterior horn cells.

   C. Measurements of chronaxie of muscles in 14 cases of poliomyelitis which exhibited marked muscle spasm demonstrated that there is no correlation of muscle spasm with anterior horn cell damage. Many muscles in spasm showed a normal chronaxie while other muscles in spasm showed more or less marked increases in chronaxie. These results suggest that anterior horn cell destruction is not the basis of muscle spasm in poliomyelitis.

   D. Prostigmine, acting on the spinal cord to inhibit proprioceptive reflexes, relaxes muscle spasm and may be of therapeutic value in poliomyelitis.

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VI. GOSSIP

In view of the anticipated increase in home canning this year we have condensed the following from the Minnesota Hospital Association Education Packet, May, 1943:

TREATMENT OF FOOD POISONINGS

Raymond N. Bieter, M.D.*

I. Bacterial Toxins.
1. Staphylococcus toxin
2. Botulism

Living bacteria
1. Streptococci, alpha type.
2. Salmonella organisms.

A. General Measures indicated in all.

1. Empty stomach
Stomach pump and gastric lavage.
Emetics:
Household Mustard, 1-2 tsp. or Copper Sulfate 0.5 Gm. in warm water.
Apomorphine HCl, 5 mg. Hypodermic only.

2. Decrease Intestinal Absorption and hasten Evacuation.
Saline Cathartic preferable to an irritant cathartic, followed by Saline or water by mouth or stomach tube if patient can tolerate fluid.

3. Rest to Gastro-intestinal tract.
Bismuth Subcarbonate, in 1, Gm. doses.
Camphorated Tincture of Opium, dosage to fit age of patient.

4. For excessive secretions, saliva, sweat, tears, etc.
Atropine Sulfate in appropriate doses.

5. For excitement, nervousness, convulsions.
Barbital Hypnotics, oral, hypo.
a. Pentobarbital sodium 0.1 Gm. or more.
b. Phenobarbital sodium 0.03 " " "
Intravenous administration for convulsions
Pentobarbital sodium in doses up to 0.5 Gm.

*Prepared especially for this packet at the request of the Committee in charge of the Subject-of-the-Month Program.

6. For Fluid and Chloride Loss (may be excessive).
   Especially where fluids cannot be tolerated by mouth use one or more of the following U.S.P. solutions parenterally, in doses of 500 to 1000 cc. or more as indicated.

   a. Sterile Isotonic Solution of Three Chlorides for parenteral Use. (Ringer's Solution)
   b. Sterile Isotonic Solution of Sodium Chloride for Parenteral Use.
   c. Dextrose Injection (5-10% most satisfactory).
   d. Dextrose and Sodium Chloride Injection (5% and 0.9% of each respectively).

7. If illness prolonged, add 1 or more of the following vitamin preparations (in ampuls), preferable to a NaCl injection above.

   a. Ascorbic Acid, in doses of 50 mg. or more.
   b. Thiamine Hydrochloride, in doses of 5 mg. or more.
   c. Injectable Vitamin B Complex Ampuls, or a B Complex preparation made, for example, from crude liver in doses of 1 or more ampuls.

8. Return to normal food habits slowly.

B. Specific Measures.
   1. Botulism.
      Botulinus Bivalent Antitoxin, N.N.R. (Jensen Salsbery Laboratories, Inc., 21st and Penn Sts., Kansas City, Mo.). Prophylaxis 2,500 Units or more by subcutaneous injection. Therapeutic, 10,000 units intravenous and repeat if necessary. Must be given early to be of value.

II. Mushroom Poisoning.
   1. Empty Stomach and decrease intestinal absorption and hasten evacuation as above.
   2. Atropine Sulfate in suitable doses, especially to control excessive sweating, salivation, lacrimation. The toxic alkaloid in mushrooms in Muscarine, which has an action very much like a stimulant of the parasympathetic nervous system.
   3. Stimulants as indicated.
   4. Barbital Hypnotics as above, if indicated.
   5. Morphine Sulfate for pain.
   6. For excessive Fluid, Chloride and Vitamin loss, administer parenteral fluids, etc., as above.

III. Poisoning from Contaminating Metal Salts. Arsenic, Lead, Chromium, commonest.
   1. Empty stomach and treat intestinal tract as above.
   2. Soluble metal salts are reduced and rendered insoluble and very much less toxic by Sodium Formaldehyde Sulfoxalate. If used early before appreciable absorption has occurred, this drug is life-saving. Administer 1-10 Gm. via stomach tube and repeat if necessary. Same dose via enema to protect colon. Same dose via intravenous injection; repeat if necessary. This salt plus fluids may institute a diuresis and thereby reduce kidney injury. Fluids parenterally as above if anuria is absent. In presence of anuria administer fluids cautiously.
   3. Symptomatic treatment as indicated. Barbital Hypnotics and Morphine Sulfate. Stimulants if necessary.
   4. Return to normal food habits slowly.