FROM THE ORTHOPEDIC SERVICE OF THE QUEENS GENERAL HOSPITAL, JAMAICA, N.Y. DIRECTOR: DR. FREDERICK COURTEN

INTENSIVE PHYSICAL THERAPY AIDED BY CURARE IN THE ORTHOPEDIC TREATMENT OF POLIOMYELITIS[1]

By

VICTOR H. RAISMAN, M.D., F.I.C.S.
Kew Gardens, N.Y.

and

FREDERICK COURTEN, M.D., F.A.C.S.
Richmond Hill, N.Y.

INTRODUCTION

The aims of the orthopedic treatment of poliomyelitis have always been the restoration of function and the prevention of deformities. In the United States, the treatment of most patients, until recently, was based on the theory that if a muscle were weakened or paralyzed, its unresisted normal antagonist would pull the part into a deformed attitude. After the onset, therefore, as soon as paralysis was evident, a plaster cast would be applied to the involved extremity or trunk to prevent overpulling by the normal muscle and to give its weakened antagonist a better chance to recover. When the acute phase was over, the cast would be removed, and increasing exercise permitted. If muscle power showed obvious inequality with the likely development of a deformity, a brace would be applied, and electrical stimulation and regulated exercises for the weakened muscles would be begun.

In 1937, Elisabeth Kenny startled the orthopedic world by advocating hot packs and early exercise, and the abandonment of casts and braces. Her treatment seemed to upset completely the theory on which the orthodox treatment had been based. She said that the overpulling muscle was not normal but was affected by the disease and in an abnormal state which she called "spasm".

[1] Presented at the Medical Congress in Oslo May 20, 1953, and before the Orthopedic Departments of several Scandinavian universities.

Aided by a grant from the National Foundation for Infantile Paralysis.

In further contradiction of accepted theories, she added that the paralyzed muscle was normal but, because of muscle incoordination and mental alienation resulting from the spasm of its antagonist, was unable to function (Pohl). These fundamentally contrary opinions forced a serious review of the concept on which the orthopedic treatment of poliomyelitis had been based. New studies disproved the Kenny theory, in part, by showing that what had been considered a paralyzed muscle was indeed a paralyzed muscle and not merely a muscle whose function was inhibited. They did show, however, that the overpulling muscle was not normal but was the subject of increased irritability which Miss Kenny had labelled "muscle spasm".

MUSCLE SHORTENING

Thus, a new concept was introduced into orthopedic thinking, namely, that the damage to the central nervous system caused by poliomyelitis included not only destruction of the anterior horn cells with resultant paralysis of the motor nerves but also injury to many more cells to which clinicians had previously paid little attention. Aside from the considerable involvement of the brain and the brain stem, interest was focussed on the internuncial nuclei in the cord. It is believed that their involvement is responsible for the development of the increased muscle irritability or spasm to which Kenny had called attention (Lowman).

This concept of increased skeletal muscle irritability in poliomyelitis has not been accepted very widely. While most orthopedic surgeons and physical therapy specialists have been content to overlook it in their treatment of patients, others have attacked it as though this were a problem for theoretical discussion rather than for controlled testing.

Schlesinger, for instance, stated that, since myanesin decreases spasm in upper motor neuron lesions but has no influence on the so called muscle spasm of poliomyelitis, there is no muscle spasm in poliomyelitis. He considers the tightness to be a combination of increased postural tone and meningismus, plus protective splinting. In a statement to the authors, however, he added that he did not treat poliomyelitis, as he was a neurosurgeon.

Pollock denies the presence of muscle spasm in acute poliomyelitis because the definition he and his co-workers use is: "Muscle spasm is a sustained reversible tetanic contraction of muscle or in lesser degree resistance of muscle to pressure." He and his co-workers do not accept the electromyographic proof of its existence.

Mitchell agrees with Pollock's definition. He found few patients in the early stage of polio in whom deforming factors which could interfere with the passive mobility of limbs were present. He found muscle resistance to stretch "only a transient complication".

Definitions

From the above references it is clear that our terms must be clearly defined. In this paper, "muscle shortening" has been chosen rather than "muscle spasm", in view of the fact that the term "muscle spasm" is linked in the minds of many with upper motor neuron lesions. They cannot accept its use for a somewhat different condition in poliomyelitis.

1. *Muscle shortening* means the involuntary partial or complete sustained contraction of a muscle, resulting in loss of its normal length. It is probably caused by lower motor neuron irritability. This condition is temporary and therefore reversible, so that normal length can again be attained by stretching.

2. *Contracture.* If muscle shortening persists for a long enough time, it becomes a true contracture and is no longer reversible. It must be treated surgically.

3. *Muscle tightness* refers to the state of loss of normal muscle. length, without differentiating between temporary reversible and permanent irreversible changes.

Electromyography

The existence of abnormal muscle irritability in poliomyelitis was first proven by Schwartz by electromyography. Ransohoff then began similar studies and showed that most, if not all, the skeletal muscles of the body demonstrate abnormal action currents in acute poliomyelitis even when at rest. This was true even in those muscles which appeared to be perfectly normal. He showed further that frequently when maximum efforts were exerted by a weak muscle, its antagonist was electrically more active than the muscle being tested. Finally, he showed that "after administration of curare aided by intensive stretching", the abnormal currents were diminished or obliterated and the disproportion in the degree of activity of muscles in relation to their antagonists did not occur. Furthermore, muscles with previously small action potentials showed increased potentials and the pathological stretch reflex, described by Schwartz in poliomyelitis, had disappeared.

Clinical Observations

Clinically, the experiences of Ransohoff, Paul, Boines, Hechman, the authors, and others, have proven beyond any doubt that involuntary muscle shortening is a constant finding in the acute stage of poliomyelitis. In marked cases, the following may be present: stiff neck, stiff back, marked limitation of straight leg raising, equinus attitude of the ankles and limitation of shoulder elevation. In some patients, the increased irritability may be less obvious, and in others, many more muscles may show involvement. If the tendency to shorten is overcome by stretching, aided by curare, normal muscle length will be re-established and can be maintained as long as stretching is continued.

Importance of Normal Muscle Length

Maintenance of normal muscle length has always been known to be important in the treatment of poliomyelitis. This is true for several reasons. If a muscle develops a contracture, owing to paralysis of its opponent, the latter is prevented from functioning. The more severe the muscle shortening, the more its antagonist is overstretched. Later, therefore, even if the innervation of the antagonist becomes normal, it is unable to regain power. Such a contracture can then only be treated by surgically lengthening the shortened tendon. The commonest such operation is tenotomy or lengthening of the Achilles tendon to overcome an equinus deformity. The purpose of lengthening the Achilles tendon by tenotomy is not only to correct the deformity but also to stop the overstretching of its antagonists. It is hoped that if they, namely the dorsiflexors of the foot, are no longer overstretched, they may be able to regain power, if there has been recovery of their motor nerve cells. In many patients this is exactly what happens, and an anterior tibialis muscle, which has previously appeared to be completely paralyzed, may develop very good to normal power when given the chance. Thus, prevention of muscle shortening means the absence of contractures and the elimination of operations for their correction. No overstretching of, nor abnormal tension on, any muscle can occur, and there is, therefore, no hindrance to recovery of power if re-innervation of the weakened muscle occurs.

Furthermore, maintenance of normal muscle length permits the complete maximum contraction of which a muscle is capable. To the extent, however, to which a muscle has already shortened, it has lost ability to contract, assuming its innervation is normal. Thus, a muscle which has become partially shortened is partially weakened.

To summarize, therefore, prevention of muscle shortening or maintenance of normal muscle length equals the maintenance of full strength of the involved muscle if its innervation is normal. It means that there is no restriction placed on the function of its antagonist. Therefore, if the latter had at one time been paralyzed and subsequently its innervation were gradually restored, there would be no obstacle to its gradual recovery of power. Thus, the best possible conditions are established for recovery of muscle function consistent with innervation.

TECHNIQUE OF STRETCHING

The treatment used in this study to overcome muscle shortening consists of stretching with the aid of curare. If possible, it is begun immediately after the diagnosis has been made. The idea of stretching is logical since it combats the involuntary shortening of the muscle by passively lengthening it. It is not a new concept. It has been used from time immemorial and for the treatment of many conditions in which involuntary muscle shortening occurs. Hippocrates used traction to overcome muscle shortening and permit reduction of fractures and dislocations.

Stretching, then, is a logical procedure for lengthening muscles in poliomyelitis and is already a tested method for overcoming muscle shortening in other conditions. Finally, electromyography has demonstrated that the abnormal action currents found in acute poliomyelitis are eliminated by stretching with the aid of curare (Ransohoff).

Standards for Stretching

The standards of normal muscle length for the most commonly involved muscles which were established by Ransohoff, are:

The neck must be so flexible that the chin can touch the sternum.

The back must permit flexion so that the face can be placed between the knees, which are held in extension with the legs together and with the entire back forming a nicely rounded curve (Figure 1).

The adductors must permit the hips to be flexed to a right angle and then externally rotated until the hips are in the frog position (Figure 2).

The hamstrings must permit flexion of the hip with the knee extended to an angle of 60°, or even 45° or 30°–45° beyond a right angle (Figure 3).

The gastrocnemius-soleus muscle group must permit dorsiflexion

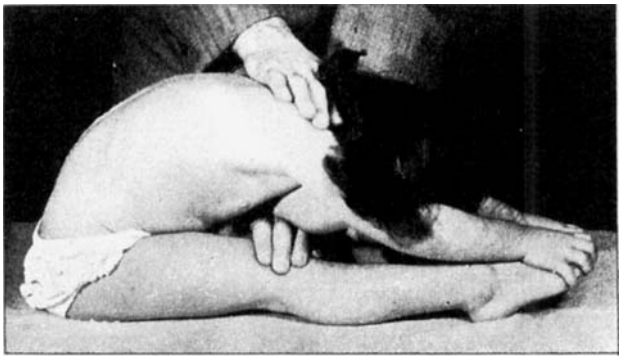

Fig. 1.
Stretching Back.

Fig. 2.
Stretching Hip Adductors.

of the ankle to 60°–65° with the foot in slight inversion and the knee in complete extension (Figure 4).

Motion of other parts of the body must be possible to a comparable degree (Figure 5).

No definite standards can be established for lateral and rotary stretching of the trunk which must be carried out as thoroughly as possible.

In some patients, mainly adults, 10°–15° less than these ranges have to be accepted as the maximum attainable.

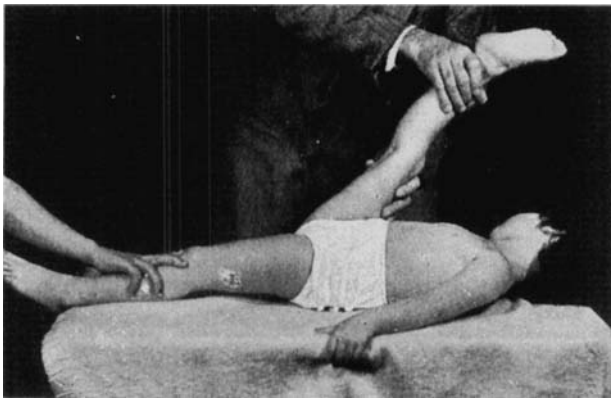

Fig. 3.
Stretching Hamstrings.

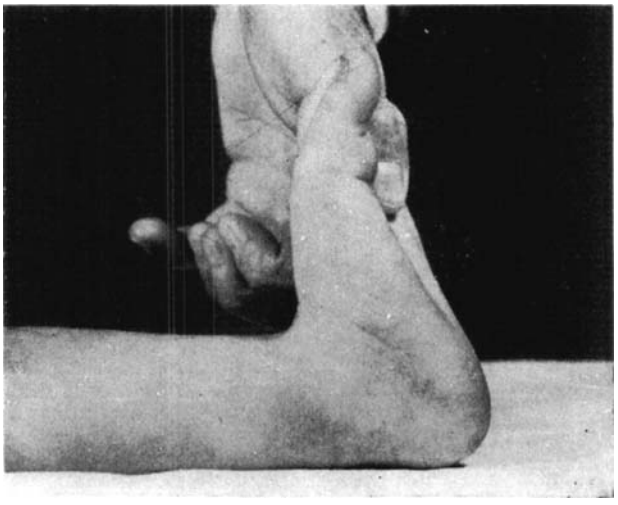

Fig. 4.
Stretching Heelcords.

These standards seem excessive to most orthopedists and physiotherapists, as they imply a far greater range of motion than is customarily considered normal. Our experience has shown, however, that satisfactory results are possible only by such maximum stretching as described above. For instance: if the hamstrings alone are under consideration, it is usually considered satisfactory to obtain straight leg raising to 90°. Consequently, a patient who can bend down with straight knees and touch his toes is considered to have fully relaxed

Fig. 5.
Stretching Hip flexors and Rectus Femoris.

back and hamstring muscles. When, however, this is analyzed more critically and the patient is examined sitting on the table with the knees in extension and the legs together, it will be noticed that he has little flexibility within the back. If the back itself is flexible, this can be proved by eliminating the tension on the hamstrings with external rotation and flexion of the hips and knees, after which the patient will be able to bend forward and practically kiss the table. In such a case, tightness of the hamstrings permits many to have persistent backache, abdominal muscle pain, and failure to recover power in the abdominal muscles. When stretching is carried to the degree outlined above, the backache and abdominal muscle pain disappear, and frequently some recovery of abdominal muscle power becomes evident.

These rigorous standards and ,in fact, the entire concept of stretching have been attacked by Lenhard, who stated that the muscles were being permanently damaged. As discussed in a previous article, careful observation of a series of patients in a continuing follow-up study over several years failed to demonstrate any evidence of such damage. Furthermore, the follow-up survey showed that as soon as stretching was neglected, the muscles began to tighten up to the range within which the patient used them.

CURARE TECHNIQUE

Curare is used because it relaxes the voluntary muscles and thus facilitates stretching (Prescott). Its physiological effect is to block nerve impulses at the myoneural junction of skeletal muscles (McIntyre). This results in their temporary partial or complete paralysis. When given intramuscularly, its effect becomes noticeable after a few minutes, rises to a peak at about one half hour, and then subsides. After one hour the effect is only moderate, and after four it has com-

pletely disappeared. At the peak of the curare effect, stretching can be carried much further before pain is produced.

Dosage

The patient is given curare (tubocurarine 3 mg. per cc) by intramuscular injection every eight hours, the amount being dependent on the body weight. It usually starts at 0.05 mg per pound body weight per injection and is gradually increased to a maximum of 0.1 mg. In patients with extensive paralysis, the initial dose may be as low as 0.02 mg and the amount is increased very slowly. One-half hour and ideally again one and a half and two and a half hours after each daytime injection, the patient is stretched by a physical therapy technician. No stretching is given after the midnight injection, which is not required by all patients. The patient is also encouraged to stretch himself and to assist in the stretching of others.

Safeguards:

Curare is given intra-muscularly, as rapid absorption is undesirable, both because of its equally rapid destruction in the body and because of the greater possibility of untoward reactions. If it is accidentally given intravenously or in an overdose, it may cause temporary partial or total paralysis of the respiratory muscles. Therefore, curare must not be given unless adequate treatment for such an eventuality is immediately available. It is, therefore, most desirable to carry out this treatment in the hospital. The following precautions are recommended for its safe use:

1. The plunger must be pulled back to insure that the needle is not in a blood vessel.
2. A syringe containing 1 cc of prostigmine 1: 2,000 or 1 cc of Tensilon must be on hand.
3. A positive pressure oxygen mask or anesthesia machine and a member of either the house staff or the nursing personnel, trained in their use, must be ready.
4. Close observation should be limited to three-quarters of an hour after each injection, since the peak effect subsides after that time. Infants under the age of two years should be watched more carefully in view of their inability to warn of impending respirator difficulty.
5. For patients previously treated in the respirator, a roentgenogram of the chest should be taken for atelectasis, before the injection of curare is begun; also, an otolaryngologic consultation to confirm the absence of throat paralysis is advisable.

6. In the presence of an acute intercurrent infection or unexplained fever, the dose should be lowered or curare temporarily stopped.
7. The initial injection is given intravenously in a dose of 0.1 cc to observe for a possible myasthenia-like reaction.

Owing to its effect on the muscles of deglutition, curare is always given after meals, namely, in the morning after breakfast, in the late afternoon after supper, and at midnight. No food is permitted for two hours after an injection.

The only complication to be feared is, as stated above, respiratory difficulty. It must be emphasized, however, that the danger following the onset of respiratory difficulty is measured in minutes, not in seconds. If respiratory difficulty should occur, an intravenous injection of Tensilon will overcome the curare effect in about a minute. Careful watching of the patient for about one hour thereafter is necessary as the duration of Tensilon action is one half to one-third that of curare. The injection of Tensilon may conceivably have to be repeated, if the symptoms of respiratory embarrassment recur.

Prostigmine becomes effective more slowly, particularly if given intramuscularly. If more than a minor reaction occurs, oxygen should be given. Extreme lethargy and weakness indicate the advisability of decreasing the dose, but double vision and increased temporary skeletal paralysis are physiological effects of curare and do not require a reduction of the dose.

Stretching is continued with the aid of curare until the standards for normal muscle length have been attained. The curare is then discontinued. The patient is not discharged from the hospital, however, until several days of stretching without curare have proven that no immediate loss of muscle length need be feared. If any tightness recurs, curare is resumed until normal muscle length is unequivocally obtained.

The patient is then discharged from the hospital with orders to report daily to the office or out-patient department for continued stretching. The patient's family is also instructed how to carry on the stretching and is expected to do it as vigorously as possible seven days a week at home. In a few cases where, for one reason or another, the patient was not brought back to the clinic or not stretched at home, muscle shortening promptly recurred. It was possible to readmit some patients to the hospital and to resume stretching with curare until a satisfactory range had been restored. The later this was attempted, however, the less the likelihood that normal muscle length would be obtained, although some improvement could be expected.

INTENSIVE PHYSICAL THERAPY

Although standards for only a few stretching manoeuvers have been mentioned, stretching should be carried out for all the joints of the body. The actual technique may be described as a gentle pumping action. The technician constantly strives to carry the part a trifle further, depending on the tolerance of the patient. Although stretching is painful in acute polio, the pain is greatly diminished when curare is used, owing to the increased resistance offered by the muscles. Despite the pain, the majority of patients have a sense of relief following the stretching and say they feel better for it. The intensity and duration of the pain is much less than when stretching is used to overcome adhesions or capsular contractions.

One difficulty encountered in this treatment is teaching the technicians. They are usually in the habit of stretching with much more force through a smaller range.

Stretching, which has been given major emphasis in this paper, is only a part of the routine called "intensive physical therapy". It is the predominant feature of the early phase of treatment until, with the aid of curare, complete relaxation has been obtained. Thereafter, as time goes on, and, as less and less difficulty is experienced in maintaining normal muscle length, the technician has time for other types of therapy: exercise, electrical stimulation, muscle reeducation, and, personnel permitting, progressive assistive or resistive exercises.

EARLY EXERCISE

Next to stretching, early exercise has probably caused the most objection on the part of physicians to this treatment. Instead of being kept in bed for months or allowed only guarded exercise with instructions "to walk only 25 steps in an hour", for example, he is urged to exercise all his muscles as soon as he can. He is given a regime of exercise to keep him busy whenever he is not being stretched. When tired, he is supposed to take a rest and then resume his exercises. He is gotten up as soon as the temperature is no longer elevated and is encouraged to sit, stand, or walk, even if it requires the assistance of two technicians. No attention is paid to muscle patterns. On the contrary, the patient is encouraged to use whatever muscles he can. As re-innervation occurs, muscle coordination becomes more and more nearly normal, so there is no need to worry over muscle substitution (Byrne). There is no concern over the harmful effect of fatigue. As shown by Hines, active use of a muscle assists its recovery, and forced activity of laboratory animals on a treadmill was "in no instance found

to be detrimental to neuro-muscular regeneration". Last, but not least, is the immense encouragement to the patient and his family derived from their ability to contribute to his improvement.

Ambulation in Children

Early ambulation is not permitted in very young children if there is still tightness of the gastrocnemeus-soleus muscles and the lower extremities are still weak. In such patients, early walking caused the development of a genu recurvatum deformity which took many months to overcome. They are, therefore, given exercise in other ways, as on a bicycle.

BRACES

As pointed out by Ransohoff, braces become unnecessary with this treatment. This seems hard to believe until their function is analyzed. Braces have been used for two purposes in poliomyelitis patients, first to support a limb, and therefore to improve weight bearing and stability, and secondly, in an attempt to prevent deformities (Broderick).

In the absence of muscle shortening and the maintenance of normal length, there is no tight muscle to act as a deforming force. Therefore, no external device need be employed to control it. An example of this, cited in a previous paper, is a patient with shortened gastrocnemeus-soleus and hamstring muscles in a limb with a paralyzed quadriceps. When he attempted to walk, his knee remained in flexion, because tension on the contracted muscles caused too much pain as he tried to lock the knee. After he had been stretched with the aid of curare, and despite the lack of any immediate return of quadriceps power, he was able to lock his knee without pain and could walk with stability. He no longer needed to keep his hand on the anterior surface of the thigh, the attitude usually associated with quadriceps paralysis, because his knee no longer tended to buckle.

The use of a brace for support of a limb is not required when muscle shortening has been eliminated, as there is no force in action other than gravity. This statement should not appear too unusual since similar observations have been made frequently in patients with a completely flail leg. Such a leg, despite lack of support, remains in a neutral position without deformity (Dunn). There is no deforming force since all the muscles are equally completely paralyzed. Similarly, the fully stretched limb passively assumes whatever position it is placed in, within the limits permitted by its joint capsules and ligaments. These structures absorb the generally mild strain and keep the

limb in a neutral position. If, however, muscle shortening were present, the strain would be much greater, and then lack of support would permit the development of a deformity. That is what occurred in the young children who developed genu recurvatum deformities.

The only patient who requires any type of external assistance is the one with a drop foot, who is fitted with an elastic device or steel spring to pull the forefoot out of the way so that it does not interfere with walking. The patient with a completely flail upper extremity whose arm subluxates at the shoulder joint, is also given a sling to help prevent overstretching of the capsule by gravity. Usually, this is soon discarded, and, in time, enough power is regained to prevent the subluxation.

CHRONIC STAGE

When this treatment was begun, it was assumed that if the patients were fully stretched and normal muscle length maintained for a period of about one year, it would be retained indefinitely. Unfortunately, time demonstrated the fallacy of this point of view. It was found that patients must continue to stretch indefinitely if normal muscle length is to be retained. In most cases, if there is no obvious paralysis, cessation of stretching brings about a degree of muscle shortening consistent with the range usually called "normal". The patient can bend down and touch his toes. Straight leg raising is possible to 90°. The heel cords are relaxed enough to permit dorsiflexion of the ankles to 85°, etc. In other words, the patient retains that degree of flexibility which he makes use of in the course of his normal life. Therefore, flexibility is greater in growing children, who are actively participating in sports, than in adults who work in an office sitting in a chair all day. It is therefore necessary to continue follow-up examinations of the patients for the purpose of encouraging them to persist in their stretching. If stretching is neglected in the presence of a weak muscle, its antagonist tightens up to the degree permitted by the strength of the weak muscle. Further recovery of the weak muscle is then prevented, and, in fact, a deformity may result.

Thus, persistent stretching, perhaps for the rest of the patient's life, is a desirable goal. While superficially this may seem to be an intolerably dreary future, it is less annoying than wearing a brace for the rest of one's life. Moreover, many surgical procedures for the relief of deformities, for instance, frequently become unsatisfactory with time and the persistent pull of the so-called deforming element so that further surgery is required. It should not be too unreasonable, therefore, to expect a patient to be willing to continue stretching for

15 minutes a day. It should become a daily habit on a par with washing one's face or brushing one's teeth.

Fixed Deformities

All the patients, who remained relaxed and continued stretching, remained free from contractures or deformities. No equinus deformity developed despite persistent paralysis of the dorsiflexors of the foot. No calcaneus deformity was noted regardless of permanent paralysis of the gastrocnemeus-soleus muscles. Furthermore, in most patients such persistent paralysis was not noted. On the contrary, a gradual return of function would occur in most of the paralyzed muscles, and such improvement could be observed in progress even five years later (Anonymous).

Deformities of the Trunk, including Scoliosis

Shortening of the muscles of the trunk is not so easily treated as that of the extremities. The major back muscles are extensors of the trunk and are stretched by passive forward flexion. This manipulation, however, does not affect the smaller muscles of the back, which pull in an oblique direction or cause its lateral deviation. Their passive stretching can be carried out only indirectly by pressure on the ribs and shoulders, particularly in the dorsal spine. Moreover, there is no absolute standard for their complete relaxation, so that it is understandable that muscle shortening may not always be completely eradicated, or prevented from recurring in the subacute and chronic stages.

Thus, although a lordotic deformity can be prevented despite paralyzed abdominal muscles, mild fixed scoliotic curves may develop in the chronic stage, particularly in the dorsal spine, despite maintenance of complete relaxation in the rest of the body.

Cyanosis and Vascular Changes

Cyanosis and vascular changes are common complications in the paralyzed limbs of poliomyelitis patients (Stenport). As pointed out in a previous article, these changes were not encountered in our patients. There was one exception that proved the rule: One patient had severe involvement of both lower extremities. Within six weeks after the onset, he sustained fractures of the shaft of the tibia and fibula of the weaker leg. After two months of immobilization in plaster for the fractures, the leg remained for some time cold, painful, and cyanotic

when dependent. Subsequently, however, with improved exercise, these complaints disappeared.

Inasmuch as neither stretching nor curare could affect the peripheral circulation per se, it must be assumed that the early exercise and ambulation unhampered by plaster casts and braces, and facilitated by stretching, were responsible for the absence of these complications.

Limb Length Inequality

Another complication of poliomyelitis is the development of limb length inequality (Gullickson). Analysis of the literature fails to reveal any conclusive correlation of the causative factors. It has been usually associated with the extent of the paralysis. Another factor known to hamper growth of a limb is immobilization. Analysis of a series of our cases (see previous article) points to the importance of tightness of the lower extremity muscles, such as the hamstrings, adductors, and gastrocnemeus-soleus muscles. All of the patients in that series in whom limb length inequality was found, had such tightness except two who had required plaster immobilization, the one mentioned above for a fractured leg and the other post-operatively following a tendon transplantation to the foot. Thus, the authors believe that unequal leg length following polio is related to persistent tightness and/or immobilization. Stretching and exercise tend to maintain equal leg length.

Tightness Syndromes

If there are no subjective complaints, tightness does not appear too significant. But Kuhns and Bingham have described syndromes both in children and in adults which were characterized by tightness and resulted in functional disability. Although Kuhns did not recognize the poliomyelitic etiology, he gave an excellent description of the very same tightness which has been the subject of this paper. Moreover, he confirms the fact that exercises which stretch the tight structures are of value in these cases. Bingham in his investigation of a group of military recruits thought to be "gold brickers", found contractures and shortening of the back, hamstring, and other muscles which were disabling them. Even though they were apparently in good physical condition and had no deformities, they were unable to carry out their duties as combat soldiers. Further study revealed rather clearly that their disability was due to polio. Thus, his observations represent a very late follow-up study on poliomyelitis patients who were con-

sidered to have recovered completely, but actually had a minor disability.

The concept of muscle tightness is attacked on the ground that there are normal variations of muscle tone. Therefore, it is said to be unrealistic to insist that everyone should be equally flexible. This point of view is in accordance with the definition found in "Applied Physiology" by Wright (page 582): "skeletal muscle tone is a state of reflex contraction concerned with maintaining posture ... and due to low frequency asynchronous discharge from ventral horn cells producing a partial tetanus."

However, in a recently published paper, Ralston and Libet raised the question of the very existence of active muscle tone. By delicate electromyographic studies they showed that normal skeletal muscles, when at rest or in a position of easy standing, are not in an active state of contraction. On the contrary, the muscles are in a relaxed state without any electrical activity except, occasionally, for a few brief moments for maintenance of balance, etc.

Their studies bolster our clinical observations that the so-called natural variations of flexibility are not "natural", but are the residual effects of poliomyelitis and that maximum flexibility is restricted by muscle tightness. Furthermore, practically all infants and many young children are extremely flexible and meet our standards. We believe, therefore, that tightness or lack of flexibility cannot be produced by normal muscles in a state of increased tonus. On the contrary, tightness is an abnormal state, probably the end result of a poliomyelitic infection.

"NORMAL" RANGE OF MOTION AND TIGHTNESS

During the past few years in which this treatment has been carried out, the authors have become more and more convinced that the so-called normal range of motion is merely an average range of motion drawn from the experiences of orthopedists, physiotherapists, etc. Contrary to the usual assumption, it is our impression that recovery from acute poliomyelitis, even if there is no obvious weakness, is practically always accompanied by residuals in the form of tightness, unless treated by stretching.

Recent epidemiological surveys have demonstrated that the great majority of the urban population has had an inapparent poliomyelitis, and thus is probably in the chronic stage. Therefore, the so-called normal range of motion is the range of motion of people who are in the chronic stage of poliomyelitis. It need occasion no surprise, there-

fore, to learn that that is the same range of motion which is found in patients who have apparently made a complete recovery from acute poliomyelitis. Proof of this opinion should be forthcoming in the next twenty years or less, if active immunity is induced successfully on a large scale, as appears likely. There will then be a large body of children who have been protected against poliomyelitis and who should demonstrate the true normal range of motion, equivalent to our standards for maximal stretching, and now found only in babies and some young children. (Moskowitz and Kaplan in a follow-up study of so-called non-paralytic patients found that a large percentage had paretic muscles or complained of general fatigability.)

Postural Deformities

Two common postural conditions, usually labelled "idiopathic", are, we believe, the result of untreated muscle tightness, caused by poliomyelitis:

1. *Adolescent round back* is, in our opinion, the end result of contractures of the back and hamstring muscles. The patient's attempts to bend forward failed to cause stretching of the contracted soft tissues but instead resulted in compression of the bodies of the mid-dorsal vertebrae with consequent development of a spinal deformity.
2. *Anterior metatarsalgia* with callus formation on the sole of the forefoot, in our experience, has been consistently associated with dorsiflexion contractures of the 2nd, 3rd, and 4th metatarso-phalangeal joints. The consequent loss of forefoot flexibility causes improper weight bearing, which in turn produces calluses and so-called anterior metatarsalgia. Unless significant arthritic changes can be blamed for the contractures, it is our belief and experience that these contractures were caused by poliomyelitic shortening of the extensor digitorum brevis muscles.

ECONOMIC ASPECTS

The economic aspects of the treatment of poliomyelitis are of considerable importance, as the incidence of recognized cases has increased so greatly. Most attention is being centered on the acute phase, without sufficient consideration being given to the costs of chronic invalidism to the patient and country.

Treatment by intensive physical therapy with the aid of curare may not lessen the hospitalization time for the acute attack, and may not

even save money by requiring less personnel, although both economies are claimed by Paul, Boines, etc. An obvious saving is, however, made by eliminating the need for braces, brace shops, and their personnel. Much more important, however, is the lessened need for later hospitalization. This is owing (1) to the almost complete elimination of deformities and consequent lack of surgery required for their correction, and (2) to the elimination of all obstacles to functional recovery of the weakened muscles, resulting in a much higher percentage of natural rehabilitation. Thus, in the long run, this treatment promises much less invalidism and therefore a lower economic cost to the patient's family and to the nation.

SUMMARY

The non-operative orthopedic treatment of poliomyelitis is based on early and persistent stretching for restoration and maintenance of normal muscle length with the aid of curare.

This method of treatment depends on the fact that muscle shortening is an integral part of the pathology of acute poliomyelitis and persists indefinitely to a greater or a lesser degree unless treated. Without treatment, such muscle shortening tends to weaken the involved muscles, prevent recovery of power by their antagonists, and produce deformities. Normal muscle length is obtained by stretching to a maximal degree to standards set by Ransohoff. Curare is required in the great majority of patients, as without it, stretching is too painful. The dosage, technique, and necessary precautions are described.

With this treatment, muscle recovery is facilitated, deformities of the extremities are not encountered, vascular changes and cyanosis do not occur, and limb length inequality is minimized.

Minor scoliotic deformities may occur owing to the difficulty of completely stretching the back.

Early exercise is an important part of the treatment, including early ambulation, except in certain groups of children. Muscle substitution is disregarded.

Braces are unnecessary, a double advantage:

1. They are unnecessary either for support or stability.
2. Their absence gives the body more freedom to exercise and recover function.

Patients with unrecognized poliomyelitis, as well as those not treated in this manner develop muscle tightness, which is recognizable, even when complete recovery is believed to have occurred. Muscle tightness thus makes the diagnosis possible in the acute as well as

the chronic phases. The definition of "normal range of motion" requires reconsideration. Muscle tightness is probably the chief etiological factor in the development of adolescent round back and of anterior metatarsalgia.

RESUME

Le traitement non opératoire orthopédique de la poliomyélite est basé sur l'extension précoce et persistante en vue de la restauration et du maintien de la longueur normale des muscles, à l'aide du curare.

Cette méthode de traitement se base sur le fait que le raccourcissement du muscle est une partie intégrale de la pathologie de la poliomyélite aiguë et persiste indéfiniment à un degré plus ou moins grand, à moins d'être traité. Sans traitement, un tel raccourcissement de muscle tend à affaiblir les muscles impliqués, empêche le recouvrement de force de leurs antagonistes et provoque des déformités. La longueur normale du muscle est obtenue par extension à un degré maximum d'après des standards établis par Ransohoff. Le curare est requis par la grande majorité, car l'extension est trop douloureuse sans cela.

Description du dosage, de la technique et des précautions à prendre.

Avec ce traitement, la guérison du muscle est facilitée, les déformités des extrêmités sont évitées, les modifications vasculaires et la cyanose n'apparaissent pas et la longueur inégale des membres est minimisée.

Des scolioses mineures peuvent être observées en raison de la difficulté qu'il y a à étendre complètement le dos.

Une partie importante du traitement consiste à pratiquer très tôt des exercices, y compris l'application ambulatoire du traitement, sauf pour un certain groupe d'enfants. La substitution du muscle n'est pas prise en considération.

1. Ils ne sont nécessaires ni pour le support, ni pour la stabilité.
2. Leur absence donne au corps plus de liberté d'action ce qui est important pour le recouvrement de la fonction.

Chez les malades atteints d'une poliomyélite non reconnue, aussi bien que chez ceux qui n'ont pas été soumis à ce traitement, il se manifeste une raideur des muscles que l'on peut constater alors même que le malade est entièrement guéri. La raideur des muscles rend donc le diagnostic possible aussi bien dans les phases aiguës que chroniques. La définition « portée normale du mouvement » demande à être reconsidérée. La raideur des muscles est probablement le facteur étiologique principal dans le développement du dos rond des adolescents et de la métatarsalgie antérieure.

ZUSAMMENFASSUNG

Die konservative orthopädische Behandlung der Poliomyelitis hat zur Grundlage die frühzeitige und beharrliche Streckbehandlung mit dem Ziele die normale Muskellänge mit Hilfe von Curare wiederherzustellen und aufrecht zu erhalten.

Diese Behandlungsmethode ist auf der Tatsache begründet, dass ein wesentlicher Teil der pathologischen Erscheinungen bei der akuten Poliomyelitis in Muskelverkürzungen besteht, ein Zustand, der sich mehr oder minder für eine unbegrenzte Zeit aufrecht erhält, wenn er nicht behandelt wird. Ohne Behandlung ruft die Muskelverkürzung eine Schwächung der befallenen Muskeln hervor, verhindert die Wiederherstellung der Kraft der Antagonisten, und erzeugt Deformiteten. Die normale Muskellänge erhält man mittels maximaler Streckung gemäss Normalmassen wie sie von Ransohoff aufgestellt wurden. Curare wird in den meisten Fällen benötigt, da die Streckung ohne dasselbe zu schmerzhaft ist.

Dosierung, Technik und Vorsichtsmassnahmen werden beschrieben.

Mit Hilfe dieser Behandlung gelingt es die Erholung der Muskulatur zu erleichtern, Deformiteten zu vermeiden, Gefässveränderungen und Zyanose werden nicht beobachtet und Ungleichheit der Gliedmassen wird wesentlich verringert.

Geringe Scoliosen können, wegen der Schwierigkeit der vollständigen Streckung des Rückens, auftreten.

Frühzeitige Übungsbehandlung, mitinbegriffen zeitiges Gehen, ist, abgesehen von gewissen Kindergruppen, von wesentlicher Bedeutung. Vom Muskelersatz wird abgesehen.

Apparate sind unnotwendig – ein zweifacher Vorteil:

1. Sie werden weder zur Unterstützung noch zur Stabilisierung benötigt.
2. Ihre Abwesenheit gibt dem Körper mehr Bewegungsfreiheit und damit grössere Möglichkeit der Wiederherstellung.

Patienten mit nicht erkannter Poliomyelitis, entwickeln, ebenso wie solche, die nicht in der oben beschriebenen Weise behandelt wurden, Muskelhärten, die selbst dann diagnostiziert werden können, wenn man eine vollständige Wiederherstellung vor sich zu haben glaubt. Muskelhärten ermöglichen daher die Diagnose sowohl in der akuten als auch in der chronischen Phase der Erkrankung. Die Definition des „normalen Bewegungsumfanges" erfordert neuerliche Erwägung. Muskelverhärtungen sind wahrscheinlich die Hauptursache für die Entwicklung des jugendlichen Rundrückens und der metatarsalgia anterior.

BIBLIOGRAPHY

Anonymous: Poliomyelitis. Lancet, 2: 582–583, 1948.

Bingham, R.: Muscle Fibrodystrophy. J. Bone and Joint Surg., 29: 85–96, 1947.

– Muscle Fibrodystrophy in Children. West. J. Surg., 58: 288–295, 1950.

Boines, G. J.: Treatment of Acute Poliomyelitis. Delaware State Med. J., 22: 270–271, 1950.

– Muscle Spasm and Paralysis in Diagnosis of Poliomyelitis. M. Rec., 163: 204–216, 1950.

Bower, A. G., Huddleston, O. L. and *Hovsepian, D.:* Use of Curare in Treatment of Anterior Poliomyelitis. Am. J. Med., 8: 160–168, 1950.

Broderick, T. F. Jr., Reidy, J. A., and *Barr, J. S.:* Tendon Transplantations in Lower Extremity. J. Bone and Joint Surg., 34 A: 909–14, 1952.

Burman, M. S.: Curare Therapy for Release of Muscle Spasm and Rigidity, etc. J. Bone and Joint. Surg., 20: 754–756, 1938.

Byrne, E. and *Roberts, A.:* Polio Epidemic in the New Castle Area. Med. J. Australia, 39/2: 10–13, 1952.

Dunn, N.: Calcaneo-cavus and its Treatment. J. Orth. Surg., 1: 711–721, 1919.

Gullickson, C. Jr., Olson, M., and *Kottke, F. J.:* The Effect of Paralysis of One Lower-Extremity on Bone Growth. Arch. Phys. Med., 31: 392–400, 1950.

Hines, H. M.: Effects of Immobilization and Activity on Neuromuscular Regeneration. J.A.M.A., 120: 515–517, 1942.

– *Thompson, J. D.,* and *Lazere, B.:* Physiologic Basis for Treatment of Paralyzed Muscle. Arch. Phys. Ther., 24: 69–73, 1943.

– and *Wehrmach, W. H.:* Physiologic Factors Involved in Atrophy and Regeneration of Skeletal Muscle. J. Iowa M. Soc., 34: 142–145, 1944.

Kuhns, J. G.: Tightness of Ligamentous Structures. Arch. Ped., 61: 179–183, 1944.

Lenhard, R. E.: Prognosis in Poliomyelitis. J. Bone and Joint Surg., 32 A: 71–79, 1950.

Lowman, E. W.: Preventable Deformities in Poliomyelitis. Arch. Phys. Med., 28: 455–61, 1947.

McIntyre, A. R.: Curare, Its History, Nature and Clinical Use. Chicago, Univ. of Chi. Press, 1947.

Mitchell, G. P.: Deforming Factors in the Early Stage of Poliomyelitis. Lancet, 263/2: 451–454, 1952.

Moskowitz, E. and *Kaplan, L. I.:* Follow-up Study in 75 Cases of Non-paralytic Poliomyelitis. J.A.M.A., 152: 1505–6, 1953.

Paul, W. D. and *Couch, O. A.:* Preliminary Report on Treatment of Anterior Poliomyelitis with Exercise and Curare. Arch. Phys. Med., 30: 277–284, 1949.

Pohl, J. F.: Kenny Treatment of Anterior Poliomyelitis. J.A.M.A., 110: 1428–1433, 1942.

Pollock, L. J.: Absence of Spasm during onset of Paralysis in Acute Anterior Poliomyelitis. Tr. Am. Neurol. A., 72: 57–62, 1947.

Prescott, F.: Clinical Pharmacology & Use of Curare. J. Belge Neurol. et Psychiat., 47: 599–612, 1947.

Raisman, V.: Orthopedic Treatment of Acute and Subacute Poliomyelitis with Curare and Stretching. N.Y. State J. Med., 52: 1147–49, 1952.

– and *Courten, F.:* The Importance of Stretching in the Treatment of Acute and Convalescent Poliomyelitis. N.Y. State J. Med., 52: 3005–09, 1952.

– and *Schneiderman, J.:* Orthopedic Treatment of Acute and Subacute Polio-

myelitis by Early Stretching with the Aid of Curare. J. International College of Surgeons, 19: 93–102, 1953.

Ransohof, N. S.: Curare and Intensive Physical Therapy in Treatment of Acute Anterior Poliomyelitis. Bull. N.Y. Acad. Med., 2nd Series, 23: 51–54, 1947.

– Treatment of Acute Anterior Poliomyelitis with Curare and Intensive Physical Therapy. Bull. N.Y. Acad. Med., 2nd Series, 23: 661–669, 1947.

Ralston, H. J. and *Libet, B.:* The Question of Tonus in Skeletal Muscle. Am. J. Phys. Med.,32: 85–92, 1953.

Schlesinger, E. B.: Neurological Changes in Anterior Poliomyelitis and Their Implications in Treatment. Arch. Neur. Psych., 66: 600–605, 1951.

Schwartz, R. P., Bowman, H. D. and *Smith, W. K.:* Significance of Muscle Spasm., J.A.M.A., 126: 695–702, 1944.

Stenport, K.: Treatment of Cold Limbs after Poliomyelitis. Nord. Med., 38: 1217–1219, 1948.

Wright, S.: Applied Physiology. 9th Edition, New York, Oxford University Press, 1952.