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Ever since the Kenny concept and treatment of acute anterior poliomyelitis was exploded into existence by its author, Miss Elizabeth Kenny of Brisbane, Australia, it has been the source of continuous orthopedic debate. Since the statistics concerning this subject vary so markedly, and are interpreted so variably, it seemed necessary to establish or condemn this concept on a more stable basis. Hence, as is so often done in medical progress, we look to the experimental laboratory. This paper does not involve the origin of the Kenny concept and treatment. Neither does it contain the prejudiced arguments for or against the Kenny process. This paper will not cite or deal with statistics in any form, but will attempt to present in an orderly fashion all pertinent experimental data to date.

Before such consideration, it is imperative that the Kenny concept and treatment be fully understood. This is quite adequately described by Miss Kenny and Dr. J. F. Pohl of Minneapolis.(1) The concept maintains that muscle spasm is the primary lesion, and the entire disease may be broken down into three main points:

1. Muscle spasm
2. Mental alienation
3. Incoordination

The concept is described as follows:

1. The affected muscles are painful, hyperirritable, and in spasm.
2. The flaccid muscles are normal. Loss of ability to contract these is due to functional dissociation (alienation) from the nervous system.
3. Ability to voluntarily contract the nonfunctioning muscle returns only after releasing spasm in the opponents and carefully restoring the physiological continuity of the nerve conduction paths back to the muscles.
4. Paralysis due to nerve cell death occurs but it is not a common condition. Most supposed weakness is due to untreated spasm and to disuse in the dissociated muscles.
5. Incoordination of muscle action appears in the untreated case.
6. Deformities do not occur. Those resulting from the old methods were due to untreated muscle spasm.

With this concept in mind, her treatment is essentially one directed towards the muscles, since herein lies the primary lesion. As with any type of physical therapy, there are certain objectives to be gained. These are:

1. Salvage(2) neuromuscular units.
2. Minimize musculo-skeletal deformity.
3. Establish patterns of functional motion.

The initial process consists of hot packs in a definitely prescribed manner[1] to relieve the muscle pain, irritability, and spasm. No casting whatever is done. The body is placed on a flat mattress and maintained in a position simulating the desired standing position. The feet are placed on a foot-board, thereby holding them at a right angle to the legs. Both knees are supposed to point directly to the ceiling. Any apparent weakness is supported. This is the technique designed to salvage damaged neuromuscular units, and secondarily minimize musculo-skeletal deformity by relieving the spasm.

With the resolution of muscle pain, physical therapy is begun. This is a process of making the patient "mentally aware" of definite muscle groups, and encouraging the action of these muscles to perform their given tasks. This is accomplished by pointing out the insertions of such muscles, stroking this area of insertion, enabling the patient to concentrate on the act. If other muscle groups come into play, the act is immediately discontinued to avoid incoordination. The patient is never exercised to the point of fatigue, and movements beyond the limits of pain are discouraged. Hereby muscle strength and function are improved to the point of usefulness.

This is but a brief account of the Kenny principles. To study the question adequately, it will be better to take each phase of the presented concept, and examine the evidence gleaned from experimental work on that particular point.

First, we shall consider "muscle spasm" which is designated as one of the initial symptoms of acute anterior poliomyelitis. According to the Kenny concept, within the muscle lies the precipitating lesion. The disease[3] is primarily spasm of the muscles, their fascial coverings, skin, and subcutaneous tissues. Changes in the central nervous system occur secondarily. This, then, is a peripheral condition.

In essence, there are two types of spasm.[4] [5]

1. Muscle hypertonus - this may persist without muscle relaxation for years.
2. Hyperirritable stretch reflex - which limits the range of passive motion. There is no resistance to passive stretch within this limit, so it differs from Parkinson's disease and spasticity in hemiplegia.

There have been multiple theoretic causes for muscle spasm proposed.[6] [7] It may be an inflammatory or toxic change present in the muscle. Or possibly there may be circulatory changes leading to a localizing anoxia in a muscle or group of muscles. Irritative cord lesions may be a factor in the etiology of spasm. Then, too, it may be due to hypersensitive denervated muscle reacting to acetylcholine from uninvolved motor, sensory, and autonomic nerve endings.

An associated pathologic picture is presented upon autopsy of patients succumbing to the disease, or other causes, in this

early stage. Bodian[8], in an extensive investigation, found that all poliomyelitis victims have brain lesions, which are most prominent in the brain stem. However, severe lesions occur regularly only in the vestibular nuclei, the reticular formation, and the roof nuclei of the cerebellum. The motor centers most commonly involved in experimental poliomyelitis are: motor cortex, globus pallidus, substantia nigra, subthalmus, midbrain, tegmentum, cerebellum, vestibular nuclei, reticular formation of pons and medulla, motoneurons of cranial and spinal nerves, internuncial neurons in intermediate and posterior columns of the cord, and proprioceptive cells in sensory ganglions. Most nerve cells are apparently destroyed during the acute attack or largely recover their normal appearance in about one month or less. There are several means whereby the nerve cells may be afflicted:[7]

1. Edema.
2. Perivascular infiltration.
3. Petechial hemorrhages.
4. Direct effect of the virus on motor neurons.

The first three alter the nutrition of the nerve cells but the effect is only temporary. The effect of the virus may be permanent, or possibly recovery may take place in some cases. In patients dying of respiratory failure early in an attack, autopsy frequently reveals extensive damage of the anterior horn cells throughout the cord, even though there may be no clinical evidence. Wright[9] describes the process as a swelling, clumping and granulation of Nissl substance

in the cytron, chromatolysis, cytolysis with distortion and disintegration of the cell-body and axis-cylinder, cytolysis with surrounding edema, cytolysis with phagocytosis, coagulative necrosis of the motor cells with neuronophagia, and round cell infiltration. Surrounding these cells are other cells which retain their appearance of normal cytrons but are encircled by disturbed tissue spaces, areas of perivascular infiltration and inflammatory products.

Besides the effect on the anterior horn cells, there is a similar destruction of the internuncial neurons.(5) Furthermore, it has been found that following arrest of the spinal cord circulation for forty-five minutes in dogs, lesions appear in the internuncial neurons with marked injury and neuronophagia.(10) The dogs developed muscle spasm which persisted for weeks. The loss of these internuncial neurons results in a marked degeneration of synaptic endings on the surface of the anterior horn cells(11), and herein may lie the mechanism of muscle spasm.

The muscle spasm of poliomyelitis is relaxed temporarily by spinal anesthesia, so it is a tetanus caused by excessive repetitive discharges of the lower motor neurons.(5) (12) The motor neuron can receive three separate groups of impulses.(13)

1. Impulses giving voluntary contractions.
2. Inhibitory impulses.
3. Excitatory impulses from the short reflex area.

It is the internuncial cells proximal to the dendrites and motor cell body, and the physiologic state of the motor cell which determine the

transmission or rejection of impulses.(14) Pain is not of primary importance in the mechanism of spasm since under pentothal sodium anesthesia the limitation of motion remains almost unchanged.(5) However, as before stated, spinal anesthesia usually produces relaxation. Complete motor block is found to yield more relaxation than complete sensory block with incomplete motor block, and curare-like drugs produce marked relaxation. Therefore we may say that the mechanism of muscle spasm is apparently a neurogenic phenomenon, predominantly on the motor side, with the internuncial neurons playing an active role.

Supplemental support of this proposed fact is offered by electromyographic studies of muscles in spasm. It is found that muscle spasm is far more generalized over the body than anticipated, and may be present in muscles demonstrating no clinical evidences of spasm whatever.(13) (14) (15) (16) Also, action current potentials can be recorded from the flaccid antagonists of spastic muscles as well as from the spastic muscles themselves. This will be expounded further later in this paper.

The spasticity is evidently reflex in nature because it is evolved by stretching or contraction of the antagonist.(15) It seems that in poliomyelitis there is a dissociation between the voluntary and the reflex excitation of the weakened muscle. Spasticity often yields stronger contraction than the voluntary contraction that the muscle is able to perform. This indicates that voluntary impulses

from proximal centers may be more effectively blocked than the sensory impulses which are stimulated by the stretch reflex. The stretch reflex of a normal lower reflex always produces a characteristic action current record.(14) In the acute stage of poliomyelitis, the stretch reflex usually produces a record indicating the prolonged contraction of muscle fibers, as related to both time and degree. Evidence of spasm is never present when a muscle fails to give any action current response to the stretch reflex.

An interesting study has been conducted by Moldaver(17) who investigated the chronaxia of muscles affected by infantile paralysis. Chronaxia of normal muscle is .06 - .50 milliseconds, depending upon the muscle. Chronaxia is found to increase with the degree of injury to lower motor neurons. Denervated muscle gives slow wormlike movements upon electrical stimulation, and its chronaxia may increase up to 120 times its normal figure. In paralysis from upper motor lesions, or in muscle atrophied from disease, chronaxia changes practically not at all.

Moldaver found that spastic muscles, as detected by myograph, have a normal chronaxia and therefore no element of neuromuscular degeneration. He states that in a normal person there is an increase of the stretch reflex in antagonist muscles unopposed by completely paralyzed protagonists. This was evidenced in a man with a severed right peroneal nerve whereby action potentials of high voltage were evoked in the right gastrocnemius muscle by dorsiflexion of the right foot, So it is the denervation which

gave rise to the spasm in this case. He feels that spastic muscles of poliomyelitis are basically normal, spasm being induced by meningeal irritation, then as the result of posterior root inflammation which lowers the reflex threshold, and lastly is due to an increase of normal tonus in the strong muscles.

Bouman and Schwartz[13] found the reflex nature of spasm is a cyclic event, one muscle being activated by another, and this one activating others, so that a vicious cycle occurs that can be broken only by disappearance of spasticity during the course of the disease, or when the muscle is completely paralyzed.

Van Rynberk and Ten Cate[19], experimenting with dogs, isolated a segment of the spinal cord by destroying three segments, both above and below the selected area, leaving it with no neural connections to other spinal cord segments. The skin area innervated by this segment could be stimulated and skin reflexes obtained stimulated those of the usual type of spinal cord animal. The muscle reflexes from the muscles innervated by this area, however, are changed. If one of these muscles is stimulated by deep pressure or direct muscle stimulus, a reflex contraction occurs in all muscles innervated by the segment.

Further work, localizing the etiology of spasm, is offered by the treatment of this acute phase with prostigmine. Prostigmine works by inhibiting cholinesterase, allowing acetylcholine to accumulate at synapses, parasympathetic nerve endings, myoneural junctions, and in the blood. Kabat and Knapp[20] have found that

prostigmine decreases spasm in poliomyelitis patients, gave evidence of decreased incoordination, and occasionally increased active muscular contraction. The range of passive motion is increased significantly. Atropine given simultaneously doesn't alter the muscular effect of prostigmine, so its muscular action is not secondary to a general parasympathetic effect. Nor is it the result of prostigmine on the myoneural junction, for increased acetylcholine would induce more spasm, not decrease it. It is probably the result of its inhibitory action on cholinesterase affecting synaptic function in the spinal cord, for intraspinal prostigmine produced a greater effect of hypotonus and produced it sooner than other parasympathetic effects due to the injection. The effects on muscle function preceded significant absorption of the drug in the general circulation, ruling out an effect on the myoneural junction. Sollmann[21] tells us that, with increasing doses of prostigmine in animals, the drug first affects the spinal cord.

If this condition, then, is due to an altered reflex activity initiated by pathology of the internuncial cells in the spinal cord, the question may arise as to why sensory and proprioceptive senses are not impaired. It has been found[22] [23] [24] that both sensory and proprioceptive senses have two neuron reflex areas, the impulse traveling directly from the sensory to the motor side, meeting only one synapse and involving no internuncial cells. So internuncial lesions may eliminate inhibitory mechanisms without blocking impulses for excitation of muscle tonus and the stretch

reflex.

Then there is the question as to how spasm can be picked up by the electromyograph when these muscles present no pain or tenderness. First we must attempt to account for the pain and tenderness. Moldaver[17] explains the pain as possibly being due to referred pain from lesions of the sensory protoneurons. Inflammation of the dorsal-root ganglia, the posterior horns, or the posterior roots is not found at exactly the same level as the lesions of the anterior horn cells. Therefore, it is not surprising that some affected muscles are not painful, while normal groups may show marked pain.

At this point, it seems almost mandatory that the Kenny concept of muscle spasm be discredited. We have found that muscle spasm is not the initial lesion, since the pathology found in the central nervous system occurs first to adequately account for this phenomenon. However, we do find that, once spasticity is present, it serves to precipitate spasticity in other areas, but only following internuncial cell destruction in portions of the spinal cord innervating these areas.

The rational of the Kenny treatment brings up another point. It seems established that moist heat will relieve muscle pain and soreness in the acute stage of infantile paralysis.[25] This was first proposed by Buzzard in 1910.[26] There have been several theories as to the mechanism involved, but nothing definite can be said. Hot applications may possibly excite warm receptors

in the skin and inhibit other motor neuron discharges, so voluntary activity, even in a normal person, should be weaker.[12] Wright[9] believes that the hot packs have a sedative effect and thereby reduce the number of irritating afferent sensations reaching the cord which is abnormally sensitive due to inflammation in the cord, nerve roots, and peripheral nerves. The general circulation is increased, and the patient tends to relax, the relaxation allowing more normal circulation, metabolism, and rhythmic tonic muscular movement. Products of effusion from the inflammation and irritation in the nervous system and muscles, tend to be absorbed before the exudate causes further damage either by pressure on neuromuscular units or by organization of fibrinous exudate into scar tissue. Heat with gradual cooling is best, for constant heat causes an undesired passive congestion, and loss of vasomotor tone results.

Position in bed may be an important factor in this stage, since it is noted that there are certain positions in which spastic muscles may be placed wherein they give off no action currents at all.[15] [18] These action potentials are evoked by passive movements. The resting position may be altered from day to day, thereby contraindicating splinting the extremity. This is compatible with the Kenny method of treatment which ignores splints and advocates complete rest in this early stage.

The next phase of the concept to be considered is "mental alienation." In accord with the concept[1], flaccid muscles are

normal. Loss of ability to contract these is due to functional dissociation (alienation) from the nervous system. Alienated muscles appear toneless and incapable of voluntary contraction, but are never painful or tender indicating that they are not the muscles involved directly by the disease. Basically, the Kenny concept of muscle alienation is founded upon the Sherrington reciprocal innervation phenomenon. This states that when one group of muscles contracts, antagonistic groups relax, so motion is facilitated. In other words, spastic muscles are contracted, yielding flaccid antagonistic muscles which are termed "alienated", and are not paralyzed at all.

This is sound reasoning, but is not borne out by electromyographic studies.[13] [14] [15] [16] By recording simultaneously the actions of antagonistic muscles in poliomyelitis, it was found that both are innervated at the same time. Also, when the right and left gastrocnemius, and right and left anterior tibial muscles were recorded simultaneously, stimulation of the weakened left gastrocnemius caused all four sets of muscles to register spasm. This was evident by both stretch reflex and voluntary contraction of the weakened muscle. Voluntary contraction of a muscle free from weakness gives more response than the same weakened muscle on the other extremity. With stretch reflex, the normal muscle gives a low magnitude spasm, whereas the weakened muscle yielded spasm greater in degree than its response to voluntary effort. Therefore, we have

a reversal of Sherrington's reciprocal innervation in poliomyelitis.

Moldaver[17] found that alienated muscles showed evidence of neuromuscular degeneration or complete denervation. Chronaxias often reached 100 times normal. Stimulation of motor nerves to these muscles yielded the wormlike movements of degeneration, and partial failure to respond. This work indicates that spastic muscles are normal, and alienated muscles show degeneration. This is precisely contrary to the Kenny concept.

This is further confirmed by the work of Watkins, Brazier, and Schawb[16]. They found that when one muscle is functionally weaker that its antagonist, the weaker muscle will show the greater abnormality electromyographically. That weakness is not due to "alienation" but to greater involvement by the disease process is substantiated by loss of electrical excitability in the weak muscles. Irritability is correlated with weakness and not with the degree offshortening, tenderness, or other clinical signs of spasm.

Muscle weakness may conceivably be partially due to changes in the nervous system which do not actually destroy the cells or fibers but do cause loss of conduction power and interference with normal neuromuscular action.[27] This is best postulated on a vascular basis. We know there must be some vascular changes in spastic muscles, for strong contraction of muscles is known to diminish or completely stop the flow of blood through the muscle.[28] Bulbring and Burn[29] at Oxford University have presented a good

deal of experimental work on vascular changes affecting the transmission of nervous impulses. They discovered that when vascular tone decreases, muscle reaction to nerve stimuli decreases also. This was proven not to be a fatigue mechanism, for direct muscle stimulation, after the decrease of excitability to nerve stimulation occurred, showed an unaltered reaction. When vascular tone was increased by adrenaline or pituitary extract, muscular reaction likewise increased. The rise in arterial pressure due to adrenaline played no part, as evidenced by one experiment wherein the blood pressure was kept constant by utilization of an overflow method when the adrenaline was perfused into the system. Not only at the nerve ending, but along the course of the nerve fiber itself, adrenaline and sympathetic stimulation can improve the transmission of impulses. These workers conclude that sympathetic impulses or adrenaline act in three ways:

1. Augment contractions of fully curarized muscle, so it must act directly on the muscle.
2. Improve neuromuscular transmission, probably by augmenting the number of impulses which become effective.
3. Improving excitability and conductivity of motor nerve fibers by action on circulation or other means.

If this vascular factor is significant, the Kenny method of treatment is in good standing. The hot packs would tend to facilitate a more adequate circulation in the muscles, thereby increasing

vascular tone and indirectly aiding nerve impulse transmission. It is known that under the Kenny treatment, strength of contraction increases and spasticity decreases. This is the result of a larger number of fibers becoming active, as shown by an increase of action current spikes, and strength increases.(15)

Sister Kenny's main point in this phase is "muscle reeducation", or making the patient "mentally aware" of the weakened muscles. This was first proposed by Dr. Lovett in 1917.(30) The patient is called upon to perform a definite act with a designated muscle or group of muscles, which is aided by pointing out and stroking the insertion of these muscles or by passively doing the desired motion first.

This method has foundation upon the fact that passive movements, following the relief of spasm, act as a mechanism of setting up a barrage of proprioceptive impulses to facilitate the motor pathway and proper pattern of response.(31) Since a muscle is generally supplied by motor neurons from several segments of the spinal cord, total degeneration is not so frequent as partial degeneration(17), thus lending avenues for these impulses to reach the muscle. Loss of strength would be expected ultimately, the degree of loss being quite variable. However, the Kenny group feel that improving function is far better than increasing the strength of the muscle involved.(3) An increase of muscle strength is made in the majority of cases with approximately

two-thirds of the increase occurring during the first eight months after onset.(32)

At this point we may venture to conclude that although the Kenny concept of "muscle alienation" has been proved erroneous, her method of therapy seems quite plausible and efficient.

"Incoordination" is the final point of the Kenny concept. The function of weaker muscles is compensated by the action of stronger muscles.(1) Sister Kenny wishes to distinguish between "substitution" and "incoordination".(4) She states that substitution is a conscious effort to make a strong muscle aid a weak one. Incoordination, on the other hand, is a "misdirected impulse". Incoordination may be of two types:

1. That due to the 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 the involved muscle 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 muscle.

We can follow this concept rather well. The pathology in the spinal cord, with internuncial cell destruction, offers an excellent etiology for this condition. As stated before, simultaneous recording of antagonistic muscles in poliomyelitis has

shown that they are both innervated at the same time, one group not being inhibited as found in normal persons. This was found in all patients, including those being trained to avoid this"incoordination".(16)

Watkins, Brazier, and Schawb(16) have determined periods of synchronous discharge of antagonistic muscles in poliomyelitis patients, especially with voluntary action, but sometimes in resting muscle. This was found in recent patients and those ten to fifteen years after their attack. This was unrelated to the presence or absence of spasm. It is a phenomenon seen in cases of nerve regeneration, and never found in normal controls. So, evidently, this misguidance of impulses is a chronic condition.

This is rather conclusive evidence of the presence of "incoordination", and is the only one of the Kenny concepts substantiated by experimental study.

Sister Kenny claims that incoordination occurs only in untreated cases.(1) We have just found that this is not true. In fact, her methods of muscle reeducation have proved futile in remedying the situation; incoordination remains. Therefore, it might well be said that, although the concept is substantiated, her method of therapy is fruitless. It is actually the substitution, not the incoordination which is aided by her treatment.

S U M M A R Y

This is a physiological appraisal of the Kenny concept and treatment of poliomyelitis based on pertinent experimental evidence, and does not deal with clinical statistics. Each phase of the concept, muscle spasm, muscle alienation, and muscle incoordination, is considered in a separate manner.

The concept of "muscle spasm", as advocated by the Kenny group, with the initial lesion in the muscle, has been proven erroneous. Instead, this spastic condition has been shown to be caused by pathology of the neurons in the central nervous system, especially the internuncial cells. These cells may be impaired by:

1. Edema.
2. Perivascular infiltration.
3. Petechial hemorrhages.
4. Direct effect of the virus on motor neurons.

Spasm, as detected by electromyograph, is found to be quite generalized and present at times without clinical evidence. Chronaxia experiments have shown spastic muscle to be normal in innervation. A hyperirritable stretch reflex and blocked inhibitory impulses precipitate the spasm which is cyclic in nature, giving rise to spasm in other muscles, and these, in turn producing spasm in others.

The Kenny therapy directed toward relieving this spasm is apparently beneficial. The hot fomentations act by minimizing

impulses initiating the hyperirritable reflex and by enhancing circulation to relieve the destructive factors affecting central nervous system neurons. Position and support of weakness are also important in reducing hyperirritable impulses, since certain positions will decrease or eliminate action currents from spastic muscle.

The concept of "mental alienation" finds no support from physiologists. Sister Kenny bases this alienation or weakness upon Sherrington's phenomenon of reciprocal innervation and declares the weak muscles to be normal. Instead, there is actually a reversal of this phenomenon, and chronaxia experiments yield evidence of neuromuscular degeneration in the weak muscle opposing spastic groups. This weakness has two contributing factors, blocked voluntary impulses and impeded impulse conduction peripherally due to reduced vasomotor tone.

The Kenny treatment of "mental alienation" is judged physiologically adequate. The effect of hot foments aid this stage by improving circulation, in much the same fashion in which they are believed to aid muscle spasm. Destructive factors are reduced and vasomotor tone increased. Thereby more voluntary impulses may reach the muscle and the hyperirritable stretch reflex is lessened. Muscle reeducation facilitates the directioning of these impulses by stimulating proprioceptive pathways.

"Incoordination" is the only point of the Kenny concept which is supported by experimental work. This condition also originates

from the neuronal pathology in the central nervous system, and is readily manifest by electromyographic studies, showing antagonistic muscles affected by poliomyelitis to be stimulated simultaneously instead of one group being inhibited. But the therapy of this condition is at fault, for the same electromyographic findings are present in muscles being trained to avoid this incoordination. It is the substitution of muscles, not the incoordination, which is benefited.

CONCLUSIONS

1. The Kenny concept of "muscle spasm" is erroneous, but the therapy of this condition is seemingly well founded.
2. The Kenny concept of "muscle alienation" is inaccurate, but her treatment is adequately supported.
3. The Kenny concept of "muscle incoordination" is substantiated experimentally, but the treatment is ineffective.

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