Round Table Discussion

NATURE, RECOGNITION AND MANAGEMENT OF NEUROMUSCULAR DISABILITIES IN CHILDREN

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Chairman Perlstein: In neuromuscular disabilities there are many problems to consider. However, about half of these comprise that group of diseases we know as cerebral palsy. We will start by orientating ourselves toward cerebral palsy, and in the discussion taking up the question of differential diagnosis and related subjects.

Cerebral palsy is not a single disease but a group of conditions which have in common a disorder of the motor system. It can be paralysis, incoordination, tremors or excessive motions, due to involvement of the motor centers of the brain. In other words, palsy and cerebral are defined. The important thing about cerebral is that the motor centers must be involved. If the problem is lack of development because of mental deficiency, you can exclude it. Likewise, spinal palsy, polio, and other causes of motor defects not due to involvement of the motor center are excluded. In schools for children with physical handicaps of all types, about half the children are cerebral palsey. So, from the viewpoint of frequency, cerebral palsy is the most frequent of the disabling conditions.

Much confusion exists in the terminology and classification of cerebral palsy. The term "Little's disease" has often been used as a generic term to cover all forms of cerebral palsy. Actually, Little described only one of the many types. Likewise, the term "spastic" is often used by the doctor and laymen to cover all categories. This tendency is conducive to loose thinking. A logical classification of the types of cerebral palsy would help to clarify this confused subject. Classification may be according to: (1) anatomic site of lesion; (2) the qualitative nature of the clinical symptom present; (3) the topographic involvement; (4) the degree of tonicity, (5) severity of involvement and (6) etiology. These naturally overlap.

CLASSIFICATION ACCORDING TO ANATOMIC SITE

1. Pyramidal
   Clinically spastics
2. Extrapyramidal or basal nuclear
   Clinically athetoids, tremors or rigidities
3. Cerebellar
   Clinically ataxics

The pathologic classification is not too helpful since it presumes knowledge we do not have. Actually, cases in whom extrapyramidal rigidity has been diagnosed have been found to have cerebellar aplasia. Likewise, cases in which the clinical diagnosis was athetosis were found to have normal basal nuclei and only cortical changes.

CLASSIFICATION OF CLINICAL EVALUATION

1. Spastic—"upper motor neuron lesion"—stretch reflex
2. Dyskiniesias (motion and tone)
   (a) Chorea—involuntary, unpredictable, uncontrollable with excess motion—little tone
   (b) Athetoid—same with more tension
   (c) Dystonia—same with marked tension
   (d) Tremor—pendular, agonist-antagonist
   (e) Rigidity—greatest resistance to slow motion
      (1) lead pipe
      (2) cogwheeling
3. Ataxia—dyssynergia, loss of balance sense

The clinical classification is perhaps the most useful. The term spastic is used in the Sherrington sense of indicating a lesion of the pyramidal tract. There is an increase in the stretch reflex. This is an exaggeration of the physiologic property of normal muscles to contract when they are passively stretched. It is elicited by a rapid stretch of the muscle and eliminated by a slow stretch. It is more common in the antagonists to the antigravity muscles. This is to be differentiated clinically from rigidity, which is elicited by slow motion and eliminated by rapid motion, and which is most marked in the antagonists to the antigravity muscles.

It is characteristic of the spastic muscle that the motion of the antagonist is interfered with. Thus, a spastic biceps muscle has no trouble in flexing the elbow, but contraction of the triceps will cause a stretch of the biceps and thus interfere through the stretch reflex with extension of the elbow. This stretch reflex travels to all the muscles of an extremity so that mass motions of the complete extremity are elicited.

The second general group are the hyperkinesias. The muscle itself is normal, but 2 factors cause abnormality of motion: namely, the tendency for the motion to be involuntary, uncontrollable, unpredictable and often purposeless, and the tendency toward occurrence of tension. A choreiform motion is one in which the extraneous motion predominates, there being little tension. This type of motion is rapid and jerky, unpredictable and purposeless. If there is greater tension in it, it becomes a slower, wormlike motion and is called an athetoid motion. When the tension becomes so great they cannot move, it is referred to as a dystonia. Such individuals develop positional attitudes; there is so much tension that it may hide the extraneous motion. In dystonia the axial muscles are more involved.

Tremor is somewhat different in that it is a predictable and always the same. It is a pendular motion due to alternate action of the agonist and antagonist muscles. The direction of movement is predictable but the extent of movement not. The movement is involuntary.

Rigidity is characterized by decreased motion. In a rigidity there is disturbance in the innervation that can be found electromyographically, and when the agonist contracts, the antagonist will not relax. As a result, there is difficulty in the motion in both directions. The characteristic of this motion is greatest resistance to slow motion. In moving slowly there is a continuous resistance involving both the agonist and the antagonist muscles. It is different from the stretch reflex, which occurs primarily in a rapid motion. Rapid movement will cause the resistance of the rigidity to disappear just like it causes the resistance of the stretch reflex to appear. This is a clear-cut differential between the two. Another difference is that whereas the spastic muscle involves primarily the anti-gravity muscle, the rigidity muscle involves the anti-antigravity muscle.

Ataxia is a balance loss, which does not necessarily involve the cerebellum alone; lesions in the frontal cortex may also result in ataxia. Thus it is better to describe it clinically as ataxia than to try to project it into pathology as a cerebellar lesion.

CLASSIFICATION ACCORDING TO TOPOGRAPHIC INVOLVEMENT

1. Paraplegia—legs only (spastic)
2. Diplegia—legs mainly, arms slightly (spastic)
3. Quadriplegia—legs more (spastic)
   arms more (athetoid)
4. Hemiplegia—arms more (spastic)
5. Triplegia—both legs, one arm (spastic)
6. Double hemiplegia—arms more (spastic)
7. Monoplegia—rare (usually spastic)

Cases in which both legs are involved alone or to a greater degree than the arms are almost always spastics. Thus, paraplegics, diplegics and quadriplegics with greater involvement of the legs are generally spastic. Likewise, hemiplegics are generally spastic, although in this type of spasticity the arm is more involved than the leg. Quadriplegics with greater involvement of the arm are generally athetoids. In the exceptional case the quadriplegic patient with greater involvement in the arms is found in whom the diagnosis is definitely spasticity. For this type of spasticity the term double hemiplegia is employed. Generally, spasticity or athetosis exists alone and not together in the same case. In hemiplegias, mixed involvement is frequently seen, with evidence of athetosis as well as spasticity, primarily in the arm.

Monoplegias are about the rarest type of cerebral palsy. When they are seen, usually they are spastic and generally one leg is involved. In most instances where the diagnosis of monoplegia is
made, a careful examination will show involvement of either the homolateral arm or the contra-
lateral leg. To my knowledge, in infantile hemiplegia both arms are never involved alone, although
injuries of adults such involvement is found.

CLASSIFICATION ACCORDING TO TONICITY

Hypertonic or tension types
Normotonic
Hypotonic or "atonic" types

The tone of a patient may be increased, in which event they are referred to as hypertonic or
tension forms, or it may be decreased, in which case they are referred to as hypotonic or atonic forms.

CLASSIFICATION ACCORDING TO SEVERITY OF INVOLVEMENT

1. Mild—ambulatory and self-helpful—needs no special treatment
2. Moderate—difficult speech, self-help and/or ambulation—needs treatment
3. Severe—fully incapacitated and bed-ridden—treatment may not rehabilitate

The classification by severity of involvement is generally empiric and follows the outline given
above. Many subdivisions may also be included, such as moderately severe or moderately mild, or
any number of subclassifications.

CLASSIFICATION ACCORDING TO ETIOLOGY

I. Anoxia

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Clinically</th>
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<tbody>
<tr>
<td>Breech</td>
<td>Athetoids, tremors and/or rigidities</td>
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<td>Narcosis</td>
<td>Quadruplegias</td>
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<td>Maternal hypotension</td>
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<td>Cord kinks</td>
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<td>Placenta abruptio</td>
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<td>Atelectasis</td>
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<td>Mechanical obstruction, etc.</td>
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II. Trauma and Vascular

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<tr>
<th>Etiology</th>
<th>Clinically</th>
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<tbody>
<tr>
<td>Hemorrhage</td>
<td>Spastic paraplegia, hemiplegia or quadriplegia</td>
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<td>Prematurity</td>
<td>Spastic paraplegia</td>
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<td>Forceps or dystocia</td>
<td>Spastic quadriplegia or hemiplegia</td>
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<td>Toxemia</td>
<td>Spastic hemiplegia</td>
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<td>Precipitate or cesarean</td>
<td>Spastic or mixed</td>
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III. Specific

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<th>Etiology</th>
<th>Clinically</th>
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<tr>
<td>Rh factor</td>
<td>Athetoid with deafness and supraversion palsy</td>
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<tr>
<td>Internal hydrocephalus</td>
<td>Spastic paraplegia or diplegia plus ataxia</td>
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<tr>
<td>Strumpell's encephalitis</td>
<td>Spastic hemiplegia</td>
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<tr>
<td>Lethargic encephalitis</td>
<td>Rigidities, tremors</td>
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On the basis of the above classifications, one may therefore describe a patient according to each
of the areas mentioned above. Thus, one may speak of a severe tension quadriplegic athetoid due to
kernicterus, or of a moderately severe atonic spastic diplegia associated with prematurity. Rather a
large number of descriptive subclassifications may thus be used.

Some of these clinical correlates can be stated very positively, but of others there is less certainty.
Anoxia, as indicated, is likely to cause an extrapyramidal syndrome—athetosis, tremors or rigidity.
Because there is a specificity in the predilection of certain noxae in certain areas of the brain, it is
obvious that specific clinical syndromes can be expected. Generally, those you see are pure cases.
Either the child is just an ataxic or just a spastic. Combinations are not common. They are pure
syndromes because they have specific etiology. If the etiology is causing severe enough involvement
to involve all areas, it is likely to be fatal. The exception to the rule that cases are pure instead of
mixed is in the group of dyskinesias, in which it is the rule that athetosis, rigidity and tremors may all co-exist in the same patient.

Among the causes of anoxia we feel that breech delivery ranks high. In breech deliveries the time between the delivery of the navel and the delivery of the head is a time of potential anoxia, and if it is over 8 or 10 minutes the child will be asphyxiated rather than injured mechanically. Statistics on this show that in a large series of children with cerebral palsy, who were born by breech, the majority were found to be either spastic paraplegias or athetoids. Those which are spastic paraplegias may often be explained on the basis that the spastic paraplegia was prenatally present and the loss of the normal kicking ability in the legs made it impossible for the child to kick a presenting breech out of the pelvis. In a series of 42 children with cerebral palsy, in whom delivery was made by elective version and extraction, 40 were athetoids.

Where trauma or hemorrhage are the cause of the injury, it is most likely to involve the pyramidal tracts, since either the hemorrhages are submeningeal over the motor area on the cortex or involve blood vessels in the region of the internal capsule. Right hemiplegias are more common than left hemiplegias, possibly due to the fact that L.O.A. deliveries are more common than R.O.A. deliveries. It was interesting to note that in a series of children with spastic hemiplegia, the incidence of right hemiplegias was 2 to 1 in children weighing over 3.6 kg. at birth, and 1 to 1 in children weighing less than this at birth.

Spastic paraplegia is most commonly associated with a history of prematurity, presumably due to the fact that hemorrhages from the small veins entering the longitudinal sinus will be more likely to involve the leg areas than the arm areas.

In toxemias of pregnancy, the mother may give birth to a normal child or to a mentally defective child or an epileptic child, but if it has cerebral palsy it is likely to be a hemiplegic. This may be due to the fact that the child develops a prenatal stroke. What evidence have we of a stroke? Unfortunately insufficient autopsy material is available but we do have pneumoencephalograms to show tremendous hydrocephalus e vacuo and sometimes absence of a whole half of the brain. One case with a calcified hematoma on that side has been seen.

The Rh factor causes a specific type of athetoid. In kernicterus, of course, the name is derived from the fact that Schmorl noted that the basal nuclei were stained with bile. We feel that the staining of the basal nuclei occurs only in those cases where the basal nuclei have been previously injured by some other factor such as anoxia. This is indicated by the fact that newborn children with atresia of the bile ducts, where the icterus may be much deeper than erythroblastosis, do not as a rule develop kernicterus. Also, Zuelzer has shown that kernicterus is seen in premature babies who have been anoxic, and in whom jaundice later developed, in the absence of erythroblastosis.

In an attempt to determine when the result in erythroblastosis occurs, we are doing tooth ring analysis on children who have recovered from erythroblastosis and who have sequelae. Since Dr. Shour has shown that it is possible to identify the time of birth in a tooth by the neonatal ring, it is possible to show that enamel defects occur in the teeth even before birth. This explains why the deciduous teeth of these children are generally poor, while their permanent teeth are good. This is corroborative evidence for the thinking that the brain injury in erythroblastosis begins even before the child is born.

A typical clinical picture occurs in the child who has had kernicterus. They are athetoid children with greater involvement of the neck and arms. About 30% of them have some degree of deafness, generally in the higher ranges. Over 50% of them have supranuclear lesions of the eye muscles, particularly affecting voluntary supraversion. Similar supranuclear lesions occur in the tongue muscles so that although protrusion and retraction of the tongue are generally normal, lateral motion and tongue tip raising are generally involved. That these are supranuclear lesions can be shown from the fact that synkinetically these motions which are voluntarily absent can be elicited.

(Three clinical cases were shown to illustrate the clinical syndromes of (1) kernicterus, (2) spasticity and (3) amyotonia congenita.)

Question: Do chicken pox or mumps during pregnancy cause cerebral palsy in the offspring?

Chairman Perlstein: We have had cases where there is a history of the mother having chicken pox or mumps, but certainly it doesn’t occur with the same frequency that it does in German measles. It is not just the type of virus but the time of involvement. It would have to occur in the first trimester. One child, whose mother fell downstairs in the third month, has a congenital heart, deafness, spastic paraplegia, catalepsy and an anomaly of the brain, a syndrome which is typical of what occurs.
when the mother has had German measles. The temporal factor may be more important than the specific factor. In German measles there is not a humoral immunity but a tissue immunity. The mother is able to transmit the virus in her blood without having to actually have German measles.

**Question:** Do the equine encephalitides have the same effect as measles?

**Chairman Perlstein:** The children of equine encephalitis mothers seem to have a variety of things. Some have hemiplegia but the sequelae are not the same as seen in lethargic encephalitis. During the acute phase these patients act as though they had extraneous motions, but I have not seen enough of the sequelae to speak authoritatively.

**Question:** I am from the San Joaquin Valley where we had quite an epidemic. Victims were nearly all small babies or adults. Quite a few of the adults died but we have not seen any sequelae in the babies so far.

**Chairman Perlstein:** In the lethargic group sequelae may take years to develop. As far as I know, in equine encephalitis the sequelae are immediate.

**Question:** Isn't one of the purposes of the exchange transfusion to prevent kernicterus?

**Chairman Perlstein:** According to Dr. Diamond, the reason that an exchange transfusion is beneficial is because the injury to the brain has not occurred until after birth. Diamond has also stated that his criterion for kernicterus is the development of a stiffness or back-arching in the first week. I have seen children who have not had kernicterus but have had edema of the brain or hemorrhage of the brain with the same symptoms. This might also be due to an allergic reaction from Rh antibodies. It has not been my experience that every child who shows this early clinical picture has sequelae. We all have seen children who look as though they were going to have sequelae and didn't have it. Likewise, we have seen children who had no immediate symptoms and who developed sequelae later on. We believe that the damage has already occurred before birth, though it may be true that replacement transfusion may prevent further damage from occurring. In fact, I have seen sequelae in 8 children who had replacement transfusions. You rarely see an erythroblastic infant whose mother has 3 or 4 children after that, and the reason for the lesser incidence of sequelae is the effect of public education. Previously we would see a mother who had had 8 pregnancies and each child kept getting worse. Education is a tremendous factor. Giving transfusions is important. The point is that in the child with erythroblastosis you may not be able to recognize the sequelae for over a year because athetoid motions do not appear in these children at birth. The identifying factor in early infancy is increased tension rather than the athetoid motion.

**Question:** Would you comment on drug therapy in cerebral palsy?

**Chairman Perlstein:** I would like to say that drugs are no substitute for a training program in cerebral palsy. At best they would have only adjuvant value. The type of drugs which are used in cerebral palsy might be classed as those being of general effect and those of specific effect.

Of the drugs with general effect, one would include such things as vitamins and anti-epileptic drugs. The indication for vitamins or for anti-epileptic drugs in cerebral palsy is the same as it is for normal children. If a cerebral palsied child has convulsions, then these drugs are indicated; otherwise they are of no value. Sedative drugs, such as phenobarbital, are indicated for the same reasons that they are in normal children.

Of the drugs that are said to have specific effect on cerebral palsy, I should state the best drug that I know is alcohol. If an individual is tense, the best thing to relax him is alcohol. Many years have been spent in teaching children how to relax voluntarily and in my experience it has been a waste of time. Wine or beer may be given them. They have a specific effect in relaxing these children. The same thing may occur following anesthesia. A child who had ether for one reason or another may walk better and talk better for a week afterwards. Tridione may be effective in about 50% of the athetoid children but is less effective and more dangerous than alcohol.

Prostigmine, which is effective in myasthenia gravis, does not have too much value in cerebral palsy. In myasthenia gravis its benefit is dependent on its action on the myoneural junction; in cerebral palsy its benefit is limited to its effect upon the imagination. If an individual thinks he is going to be helped by prostigmine, he will be helped by it whether he is given the drug or a placebo. It has been proved that no better results are obtained from prostigmine than from milk sugar. In our experience it is effective in 5% of the cases, which is a very low average for a specific type of drug. Tolserol is not even as good as prostigmine. It has very little effect on an individual with cerebral palsy. This is true of the other related drugs.

Curare will actually release tension but clinically is not a good drug to use since it has a low
before an adequate speech therapy program can be planned. Several of the newer drugs which are now under clinical trial show promise of being more helpful. Carbon dioxide (30%) inhalations also have a transient relaxing effect.

**Question:** In a very young baby, what is indicative that it might be a cerebral palsied child?

**Chairman Perstein:** The very first thing that you would see would be a hypertonic infant. If a child has had colic, vomited and has been constipated, without any allergic cause for it or any anomaly of the gastrointestinal tract, cerebral palsy must be considered. Next you observe the child closely. How does the child hold its head up? Does this child have a smile that is differentiated? Does the child move its legs in the proper manner? Does it move one more than the other? Does it have increased tension with attempts to make voluntary movements? Actually the ability to diagnose cerebral palsy in the very young infant is directly proportional to the experience and acumen of the clinician. Since it is imperative that the clinician have an excellent knowledge of what is normal before he can recognize what is abnormal, it is obvious that the pediatrician would be best qualified to make the diagnosis early.

**Dr. McDonald:** To understand the many ways in which cerebral palsy might affect speech we should review briefly the processes in which normal speech is produced. Actually, normal speaking consists of nothing more than the making of a lot of noises, Man has learned to listen to these noises and to give them meaning. The process of making the noises is called "speech."

A study of the mechanism used in speaking shows that speech is an overlaid function and that we have no speech "organs" per se. A careful analysis of the physiologic processes which contribute to speech production indicates, however, that not only are basic biologic acts greatly modified for speech, but some functions appear to have been developed largely for speech production. Such an analysis indicates that at least 6 types of physiologic activity contribute to the speaking act: respiration, phonation, resonation, articulation, integration and cerebration.

The motive power for speech is supplied by respiration. The air which has been inhaled is held in the lungs and gradually exhaled though the larynx, where the second major physiologic event takes place. For the production of certain sounds, the vowels and voiced consonants, the vocal folds are adducted, and the outgoing air stream sets them in vibration, producing what is called "phonation." In the production of other sounds, the vocal folds are abducted and phonation does not occur; hence, these sounds are classified as voiceless sounds. The sound patterns set up at the laryngeal level are modified by resonation which occurs predominantly in the pharyngeal, oral and nasal cavities. Resonance is not only the chief factor which distinguishes one vowel from another but it affects all of our speaking by giving our voices their characteristic quality. Articulation takes place in the oral cavity where the tongue alters its position to place varying degrees of obstruction in the way of the outgoing air stream, in the case of some sounds, or to modify the size and shape of the resonating cavities in the production of other sounds. The integrating function of the central nervous system coordinates the actions of this complex neuromuscular system to produce the consonants, vowels and diphthongs [the noises] of which speech is comprised. The function of higher level processes makes it possible to impart meaning through these speech sounds.

Cerebral palsy may affect any one or various combinations of these physiologic activities which operate to produce speech. In fact, it has been estimated that about 75% of all cerebral palsied children have speech defects. Many of these children will respond to speech therapy; however, it is necessary to understand how the neuromuscular disturbance interferes with each physiologic activity before an adequate speech therapy program can be planned.

In normal respiration inhalation results from the simultaneous increase in all dimensions of the thoracic cavity. This is accomplished primarily by the elevation of the rib cage and the depression of the diaphragm. Exhalation occurs when these processes are reversed, i.e., the rib cage is lowered and the diaphragm returned to its former level. In normal quiet breathing these events alternate rhythmically about 20 times per minute with the duration of each exhalation being only slightly longer than that of each inhalation. In normal speech breathing this pattern is markedly modified. Inhalation is very rapid, occupying on the average only about one-sixth of the total respiratory cycle, and exhalation is greatly prolonged. This controlled exhalation is essential for continuous speech, i.e., speech which consists of more than one word. Cerebral palsy might produce any of several aberrations of the breathing pattern. Some children breath at a too rapid rate to be able to indulge in the vocal play which is an antecedent of speech. A too rapid rate might also prevent the prolongation of exhalation which is essential to speech production. Two types of irregularity in the breathing pattern...
are often found. The child may initiate an expiratory movement of the rib cage, for example, only to have it interrupted by an inspiratory movement. This would, of course, interfere with speaking. Another type of irregularity often seen is asynchrony between the diaphragmatic-abdominal and the thoracic musculatures. Normally in inhalation the thoracic and abdominal circumferences are simultaneously increased and in exhalation they are simultaneously decreased. Sometimes the thorax and the abdomen seem to be working at counter-purposes, i.e., the rib cage may be elevating thus increasing the volume of the thoracic cavity while the abdominal musculature is pushing the diaphragm upwards thus decreasing the volume of the thoracic cavity. When this occurs the child may be unable to inhale the air necessary for speech or, should it happen during exhalation, he would be unable to produce the air stream which is the motive power of speech. Many children are observed who appear to make little or no use of the thoracic cavity for quiet or for speech breathing relying almost totally on the movements of the diaphragm for both inhalation and exhalation. It has been estimated that movements of the diaphragm alone account for only about 50% of the volumetric changes during inhalation and it is thought that a breathing pattern which does not include thoracic activity is inadequate for speech.

High speech motion pictures of the larynx, the structure which produces phonation, have shown that in normal respiration the vocal folds are abducted thus creating a triangular area through which air passes easily. For phonation the folds are adducted and placed under tension by the contraction of the laryngeal musculature. It has been demonstrated that the degree of tension is related to both the intensity and the pitch of the voice. Involvement of the laryngeal musculature may be manifest in several ways. If the vocal folds are adducted with extreme tension the child may be unable to initiate phonation. If adduction is inappropriately interrupted by abduction aspirate speech may result. Variation in tension of the folds resulting from incoordination might produce sudden changes in the pitch or intensity of voice.

The most important resonators for speech production are the cavities of the larynx, pharynx, nose and mouth. Since the size and shape of several of these cavities can be modified by muscular action it is natural to expect that disordered resonation might be a result of cerebral palsy. While it is true that cerebral palsied children often present unpleasant voice qualities this problem does not usually receive the direct attention of the therapist but rather is treated along with some of the other more serious problems.

The tone produced by respiration, phonation and resonation would be variable only in pitch, intensity and quality and would be of only limited communicative value. Articulation is the process by which this tone is modified and new sounds created to give speech intelligibility. The lips, mandible, tongue and various other intra-oral structures play important roles in articulation. For some sounds the lips are momentarily approximated and suddenly separated, for others the lips may be pursed, retracted or neutral. For 2 sounds the lower lip is brought into contact with the upper teeth. It is not enough that the child be able to perform these movements in isolation. He must be able to perform them as parts of the movement sequences which characterize continuous speech. In connected speech the mandible is rarely lowered as much as 20 ml. from its rest position yet it moves rapidly and frequently within this range to assume positions appropriate for the production of different speech sounds. Many cerebral palsied children seem to hold their lower jaws in markedly depressed positions and often they have difficulty closing their mouths. Others can open and close their mouths slowly but cannot carry out this act at the rate required for producing connected speech. Of all the articulatory structures the tongue is the most important. Consisting as it does of several muscles the tongue is able to alter its size, shape and position. Because of this facility the tongue plays the dominant role in the articulation of most of the speech sounds. Three major types of lingual involvement are seen in cerebral palsy. Of these, inability to elevate the tip of the tongue is encountered most frequently and probably has the most adverse effect on speech since it has been estimated that approximately 85% of the consonants used in continuous speech are produced by adjusting the tip of the tongue to the roof of the mouth. Athetoid movements of the tongue frequently interfere with the performance of voluntary movements and distort the speech sounds. For producing continuous speech one of the most important characteristics of the tongue is its ability to perform overlapping movements, e.g., while the tip of the tongue is being elevated the back of the tongue may be lowered. In cerebral palsy this ability to perform patterns of movement is sometimes disturbed.

It will be recognized that speaking cannot be divided into several separate activities. Respiration,
phonation, resonance and articulation are really but different aspects of the speaking process. Since speech has so many aspects it has been described as the most complicated neuromuscular act which the body performs. For the production of even a short phrase more than 50 muscles must be made to work together with precise coordination. Oftentimes in cerebral palsy this timing is upset and the patient is unable to integrate the several activities which help produce speech as a functional whole.

In the normal individual higher mental processes function to provide content for the speaker and to give meaning and coherence to his utterances. Two disturbances of these higher mental functions, both of which affect speech, are sometimes seen as concomitants of cerebral palsy. Recent research has shown that about 45% of cerebral palsied children must be classified as mentally defective. This condition not only retards speech development in the cerebral palsied but may prevent the patient from developing the level of speech proficiency of which he is neuromuscularly capable. Some children suffer a disturbance in their ability to manage the symbols of which language is comprised. The speech of these cerebral palsied children will reflect the receptive, expressive or mixed nature of their dysphasia.

The cerebral palsied child’s chances of developing socially adequate speech are greatly enhanced by early treatment. It is unfortunate that the speech pathologist often does not see the cerebral palsied child until it has become obvious to the parents that the child’s speech is retarded or defective. As was pointed out earlier, speech is an overlaid function which emerges from the breathing, chewing, sucking and swallowing activities of the infant. If, during early infancy, the speech pathologist could evaluate the degree to which the neuromuscular disability has involved the structures which will later be expected to produce speech he can often outline training procedures which will prepare the way for speech production. Proper seating and exercises to strengthen the head, neck and trunk muscles may improve the breathing pattern. Phonation and breathing may be improved by blowing and humming, for example. Adequate lip, tongue and jaw movements may be developed by using modified feeding procedures to improve the chewing, sucking and swallowing activities of children. Early evaluation will also provide an opportunity for the speech pathologist to advise the parents concerning proper technics for stimulating the child to speak. When early and proper attention are given to preparing the child psychologically and physiologically for speaking later attention may be given to training in the production of specific speech sounds. It is sometimes thought that it is important for the speech therapist to know with what type of cerebral palsy child she is working, i.e., a spastic or an athetoid. In our opinion it is much more important for her to know specifically what the child can do and what he can’t do. Teaching the child to do those things which are important for speech production should be the objective of therapy.
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MEYER A. PERLSTEIN and EUGENE T. MCDONALD
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