A review of the literature reveals confusion in nomenclature and classification in the field of cerebral palsy. Using as a basis the majority opinion from questionnaires sent to the members of the American Academy for Cerebral Palsy during 1953 (all members were polled; 64 per cent replied) by the Nomenclature and Classification Committee, the following classification for cerebral palsy is presented.

A complete diagnosis should include one or more titles from each of the main headings of this nomenclature. The motor classification is given precedence. Next, there should be a statement as to topography of the disease (body parts involved). There also should be a statement as to the etiology of the disease. A statement of the capabilities of the patient should include the pertinent motor, sensory, intellectual, emotional, visual, speech, and hearing status, and should be made under each heading in the supplemental classification. If the pathology in the brain has been determined accurately at necropsy or by means of electroencephalograms, pneumoencephalograms, or surgical exploration, a statement of structural disease should be made (neuroanatomical diagnosis). Finally, a statement of the functional and therapeutic classification of the patient completes the list. Thus, a comprehensive diagnosis will demand a careful consideration of every aspect of the patient and will afford a sound basis for prognosis, neuroanatomical correlation, and treatment.

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CEREBRAL PALSY DEFINED

"The term, cerebral palsy, constitutes five main classes of handicapped children, of which the older term, cerebral spastic paralysis, is only one of the group. It is frequently a combination of both motor and sensory involvement. It was formerly considered as involving only a motor handicap, and on this basis confusion arose among those treating such cases, since the other associated handicaps were not recognized and difficulties arising from treating these children were attributed to their being mentally defective. However, it is very difficult to explain how mental deficiency can be associated with pure athetosis or ataxia. The brain is a very complicated organ and any injury or lack of development in it gives rise to unusual disturbances of function, both along the line of motor control and sensation. This places cerebral palsy in a group by itself, being made up of more than one entity depending on the area injured, diseased, or defective." (From lecture notes of Winthrop Phelps.)

In Cerebral Palsy: Its Individual and Community Problems by Denhoff, as a standard definition, reference is made to Perlstein who is quoted as stating that cerebral palsy is generally defined as a "condition, characterized by paralysis, weakness, incoordination, or any other aberration of motor function due to pathology of the motor control centers of the brain." A limited definition cited by Denhoff is one wherein it is conceived as "a condition in which interferences with the control of the motor system arises as a result of lesions occurring from birth trauma." The practical definition, according to Denhoff, warrants serious consideration: "One component of a broader brain damage syndrome comprised of neuromotor dysfunction, psychological dys-
function, convulsions, and behavior disorders of organic origin.

"The characteristics of the cerebral palsied child are paralysis, weakness, incoordination, or any other aberration of motor function due to malfunction of the motor centers of the brain.

"He may also have other symptoms which reflect a damaged brain. There may be convulsions, mental retardation or deficiency, vision, hearing or perceptual problems, as well as speech, behavioral and emotional disturbances.

"The underlying cause of these symptoms—brain damage—is not a specific type. Rather similar pathological findings are found in such clinical entities as mental deficiency, epilepsy, and behaviour disorders or organic origin. Thus originates the term 'brain damage' syndrome which includes cerebral palsy along with other conditions mentioned.

"Cerebral palsy is the neuromotor component of the 'brain damage' syndrome. It must always be kept in mind that the cerebral palsied child may suffer from any other component or components of the syndrome along with the neuromotor handicap.

"The concept of the 'brain damage' syndrome has many practical applications, particularly from an educational and 'parent understanding' viewpoint. It negates the propaganda that the cerebral palsied child presents a problem unique from that of other similarly handicapped children."

John F. Pohl defines cerebral palsy as follows: "Cerebral palsy is the term used to designate a group of neuromuscular disorders in which there is impairment or loss of muscular control due to a lesion of the brain."

C. L. Balf and T. T. S. Ingram, in a recent article, state as follows: "Cerebral palsy is a descriptive term, applied to a group of motor disorders of young children, in whom full function of one or more limbs is prevented by paresis, involuntary movement, or incoordination. Tacit agreement has been reached that progressive diseases and those characterized by transient motor disturbances should be excluded as well as those primarily the result of spinal cord lesions."

Cruickshank and Raus, state: "As a disease, cerebral palsy is not progressive nor is it contagious or epidemic in form. As a neuro-physical and neuro-psychological deviation, however, it is basically a disease. Glidden Brooks has correctly spoken of cerebral palsy as a 'long-term, non-fatal, non-curable disease.' He considers it a disease in the sense of the present discussion. It is non-curable, but oftentimes amenable to therapy and training. It is non-fatal per se."

Cerebral palsy comprises those motor and other symptom complexes caused by a non-progressive brain lesion (or lesions). The characteristic thing about cerebral palsy is that it is a well defined entity with a variety of etiologies and pathologies. When clinical syndromes become better delineated with respect to known etiologies and pathological changes, they too can be removed from the general category of cerebral palsy, and placed in their own categories. For example, it is not unlikely that bilirubin encephalopathy will someday be removed from the category of cerebral palsy and given its own name. In the aging, arteriosclerosis or parkinsonian disease can cause a change in the patient who has had cerebral palsy since infancy or childhood, but these are morbid entities superimposed on the original lesion causing the cerebral palsy.

The decision of the American Academy for Cerebral Palsy to delete neoplastic brain diseases and the progressive neurological degenerative diseases of the brain has several important advantages. This decision reduced the field of cerebral palsy to a more logical and manageable unit. The treatment of brain neoplasms is frequently surgical or radiological and the treatment program depends on the type of neoplasm. It is a great mistake to treat a brain neoplasm as cerebral palsy. Obviously, the correct diagnosis should be made and the proper treatment program carried out.
After the tumor is removed and there is no danger of recurrence, the patient may then be accepted to the ranks of those with cerebral palsy to be treated symptomatically for residuals of the lesion. The same is true for a child with hydrocephalus, traumatic or infectious damage to the brain. Under the new definition, the patient is not accepted as cerebral palsy until a progressive nature of the etiology has been eliminated. This does not mean that treatment technique suitable for cerebral palsy cannot be used on patients with progressive brain lesions, but rather that attention should be concentrated on the treatment and elimination of progressive damage to the brain as long as this is a possibility.

**Classification of Cerebral Palsy**

**I. Physiological (motor)**

A. Spastic
B. Athetotic

1. Tension
2. Non-tension
3. Dystonic
4. Tremor

C. Rigidity
D. Atonic (rare)
E. Mixed
F. Unclassified

**II. Topographical**

A. Monoplegia
   - Involves one limb; condition is rare; should be checked closely to determine if you are not dealing with a paraplegia or hemiplegia.

B. Paraplegia
   - Involves the legs only and practically always of the spastic or rigidity type.

C. Hemiplegia
   - The lateraled one-half of the body is affected and it is usually spastic, although pure athetoid hemiplegias are occasionally seen, as are pure rigidity hemiplegias. There is often sensory involvement in the areas of proprioception to point discrimination and form perception. Aphasia appears more frequently in right than in left hemiplegias and are much more common in the acquired than in the congenital cerebral palsy.

D. Triplegia
   - Involves 3 extremities, usually both legs and one arm, usually spastic. This may represent hemiplegia plus paraplegia, or incomplete quadriplegia. In the latter case, both arms will be equal or nearly equal in length. In the former, the involved arm will be shorter.

E. Quadriplegia
   - Involvement of all 4 extremities. Patients with the greatest involvement of the legs are usually spastic, and patients with greatest involvement of the arms are usually the dyskinetics, including athetoids.

**III. Etiological**

A. Prenatal
   1. Hereditary—Genetically transmitted and may involve racial or familial predilections and often sex-linked. These are often classified as "cerebral agenesis." The symptoms are often present at birth and generally do not progress. Examples: Hereditary athetosis, familial tremor, familial spastic paraplegia.
   2. Acquired in utero
      a. Prenatal infection (toxoplasmosis) rubella, or other maternal infection.
      b. Prenatal anoxia—carbon monoxide, or strangulation of mother, maternal anemia, hypotension, e.g., following spinal anesthesia, placental infarcts, or placenta abruptio, kinking, knots or prolapse of the cord.
      c. Prenatal cerebral hemorrhage—maternal toxemia, direct trauma, maternal bleeding diathesis.
      d. Rh factor, Klinefelter due to Rh factor (controversial).
      e. Metabolic disturbances, diabetes.
      f. Gonadal irradiation, harmful exposure to X-ray.

*The form of the following classification, in general, is patterned after that which has been successfully used for some years by the American Heart Association.
MINEAR – CLASSIFICATION OF CEREBRAL PALSY

B. Natal
1. Anoxia
   a. Mechanical respiratory obstruction.
   b. Atelectasis.
   c. Narcotism (due to drugs).
   d. Placenta previa or abruptio.
   e. Maternal anoxia or hypotension.
   f. Breech deliveries with delay of the after-coming head.
   g. Bleeding in the first trimester (see Eastman).

C. Postnatal
1. Trauma—Subdural hematoma, skull fractures, wounds and contusions of the brain (accidental).
2. Infections—(more common in children than adults) meningitis, encephalitis, brain abscess.
3. Toxic causes—Lead, arsenic, coal tar derivatives, streptomycin, etc.
4. Vascular accidents (more common in adults than children) congenital aneurysms, circle of Willis, hypertensive encephalopathies, emboli due to bacterial endocarditis or fat embolism, cerebrovascular thrombosis, in debilitated infants, sudden pressure changes.
5. Anoxia—Carbon monoxide poisoning, strangulation, high altitudes, and deep pressure anoxia, hypoglycemia.
6. Neoplastic, or late development defects—Brain tumors, brain cysts, internal hydrocephalus, hydrocephalus.

IV. Supplemental
A. Psychological evaluation
   1. Degree of mental deficiency, if any.
B. Physical status
   1. Physical growth evaluation (Wetzel Grid or other)
   2. Developmental level (Gesell)*
   3. Bone age
   4. Contractions
C. Convulsive seizures
D. Posture and locomotive behaviour patterns
E. Eye-hand behavior patterns
   1. Eye dominance
   2. Eye movements
   3. Eye postures
   4. Fixation
   5. Convergence
   6. Prehensory approach
   7. Grasp
   8. Manipulation
   9. Hand dominance
F. Visual status
   1. Sensory
      a. Amblyopia
   b. Field defects
2. Motor
   a. Conjugate deviations (33% of motor defects)
   b. Fixation defects
   c. Spasmus fixus (1%)
   d. Strabismus fixus (1%)
   e. Esotropia (51%)
   f. Exotropia (9%)
   g. Hypertropia
   h. Hypotropia
   i. Nystagmus
   j. Pseudopalsy of the externi (22%)
and progressive neurological diseases from the cerebral palsy classification. (2) Co-operate with the Editor of Standard Nomenclature of Diseases and Operations to prepare a classification and coding based on the accepted classification along with the following recommendations: (a) Delete the term “cerebral spastic infantile paralysis” and substitute “cerebral palsy.” (b) Assign code numbers for the 6 motor types of cerebral palsy, and a code number for “cerebral palsy, type unknown” (Note: The mixed type may be coded using two or more of the type code numbers).

POINTS FAVORING THE MOTOR CLASSIFICATION AS FIRST CHOICE

The consensus giving the motor classification first choice instead of the etiological or neuroanatomical classification is as follows:

1) The treatment is primarily the treatment of symptoms, not the etiology or the portion of the brain involved. Etiology frequently cannot be determined, but it is highly important to include it in the diagnosis when it can be determined (even when determined, the treatment will be symptomatic). Since the treatment of cerebral palsy is symptomatic, a classification based on major presenting symptoms is useful and practical. Moreover, if etiology is known, the eventual pathology and pathogenesis can better be studied.

2) The neuroanatomical classification based on position, extent, and character of the brain lesion, is highly desirable from a scientific point of view, but is presumptive without necropsy, although electroencephalogram and air studies give important information. When the Brain Registry of the American Academy for Cerebral Palsy has studied more brains and correlates lesions with primary presenting symptoms, we will be able to evolve a neuroanatomical diagnosis for cerebral palsy. Even then, the diagnosis in the living will be based on inference and the treatment will still be the treatment of symptoms.

3) The decision of the American Academy for Cerebral Palsy to include the manifestations of nonprogressive (static) brain lesions as cerebral palsy was probably the most important single decision. The field of cerebral palsy is now clearly limited to a more logical and manageable size. Furthermore, progressive brain lesions should be classified so as to place emphasis on etiology.

SCOPE OF FUTURE CLINICAL WORK OUTLINED

For the time being, the clinician had best be practical and resolve to make a careful symptomatic analysis of patients with cerebral palsy using all clinical tools available to him; treat the total patient symptomatically and co-operate with the Brain Registry in collecting, studying and correlating the symptoms with lesions. This then is the primary work of the clinician: Let him use a classification that fits his purposes in a practical way—Let him concentrate on the phase of cerebral palsy which, by training and opportunity, he is best able to make, then he will at least be doing his part well. It appears that we, as clinicians, are missing a lot in our clinical evaluation of the child with cerebral palsy, especially in the realm of sensory involvement. Probably this is true because we are just beginning to work out techniques to treat the sensory losses. We have concentrated on the motor and intellectual phases of cerebral palsy and neglected the sensory phase. If the student of medicine can grasp and understand the clinical manifestations of cerebral palsy, he will have broadened his knowledge of medicine considerably.

TYPES OF CEREBRAL PALSY DEFINED

Cerebral palsy comprises those motor and other symptom complexes caused by a nonprogressive brain lesion (or lesions). The various sub-types may be defined as follows:

A. Spasticity—Characterized by a lower threshold of the stretch reflex, an enlarged reflexogenic area, augmented responses with clonus, and an abnormal electromyographic
record (synchronization of discharge rate in various parts of the spastic muscle). The pathological stretch reflex must be present to make a diagnosis of spasticity. There is a tendency toward greater involvement and contractures, affecting the antigravity muscles.

B. Athetosis—Characterized by an abnormal amount and type of involuntary motion, normal reflexes, normal electromyographic findings, uncontrolled, involuntary and inco-ordinate motions with varying degrees of tension. Description of the various clinical types by Winthrop Phelps2 follows:

I. Rotary
   A. The most common type.
   B. Involves muscles which can take part of rotary motions.
   C. The rotary motions are usually rather slow, sometimes extremely slow.
   D. The feet describe a circular motion; the hands pronate and supinate; the shoulders internally and externally rotate.
   E. There may be varying degrees of tension.

II. Tremor (tremor-like)
   A. Almost as common as the rotary type.
   B. There is an irregular and uneven type of involuntary contraction and relaxation which involves flexor and extensor, and adductor and adductor mechanisms.
   C. Rotary motion is not seen. Involuntary rotary motion is not seen. The motion is not a true tremor.

III. Dystonic
   A. The extremities assume distored positions held involuntarily for periods of from a few seconds to a few minutes.
   B. The dystonic motions may involve neck, trunk, arms, and legs.
   C. An entirely different distorted position may be assumed within the passage of a few minutes.

IV. Shudder
   A. Closely resembles the shudder that any normal person may experience.
   B. The shudder may be violent, causing patient to fall on the floor or simply a mild shudder-like motion going through the arms and legs.

V. Flail(ing)
   A. A rare type of athetosis.
   B. Arms and legs are thrown around rather violently from the axial shoulder and hip joints.

C. There is usually little involvement in the hands, fingers, wrists, or knees.
D. Arms and legs describe these flailing motions, usually fully extended, sometimes flexed.
E. Doctor Phelps has never seen flail(ing) athetoids live beyond maturity. Apparently there is some progression of the disease between 10 and 14, with gradual weakening of the muscles of breathing followed by flaccidity and death.

Note: This resembles one of the rare progressive neurological diseases and since it is progressive, would be excluded from the new classification. The cause of death in this type of athetosis is not known. Doctor Phelps prefers to retain this entity for the sake of completeness.

VI. Tension
   A. A state of muscular tension in an anthetoid (rotary, tremor or dystonic) hiding the characteristic motions of the types.
   B. When the tension is relieved the true nature of the athetosis (rotary, tremor or dystonic) is revealed.
   C. Must be distinguished from spasticity, in which a stretch reflex must be present and from rigidity in which the lead-pipe-like resistance of the muscle is found.
   D. The state of tension is not constant, and can be shaken out by the examiner.
   E. The athetoid is only classed as tension athetoid when the tension is the outstanding characteristic and masks the rotary, tremor, or dystonic features.

VII. Non-tension
   A. This is also a transient state and might mask other types of athetosis.
   B. In infancy it is often mistaken for the rare atonic type of athetosis.
   C. Involuntary motion must be present.
   D. Non-tension athetosis is frequently seen in small babies and is usually the first identifiable symptom. As the child grows, one of the first five types mentioned above can be identified.

Note: It must be understood that both “tension” and “non-tension” are temporary classifications of patients under treatment.

VIII. Hemiathe'thosis
   A. This is strictly a topographical classification and is made when rotary, tremor-like, dystonic, or shudder athetosis involve one side of the body.
   B. Doctor Phelps has not seen a hemi-flail(ing) athetosis.

IX. Neck and Arm.
   A. This type is limited to the head, neck, and
shoulder girdle, with little or no athetosis in the legs.
B. Some of these learn to typewrite or pick up objects or write with their toes.
C. There may be or may not be a speech defect.
D. The motion may be rotary, tremor-like, dystonic, or shudder, but most of them are dystonic.
E. Doctor Phelps thinks it advisable to use the separate classification of neck and arm athetosis for determining the best plan for placement and treatment.

XII. Emotional Release
A. Usually the rotary or tremor-like athetosis is present, combined with release of the laughing, crying and anger mechanisms. A very slight stimulation for laughter or crying will produce the whole picture of this emotion without any particular background of feeling of the emotion.
B. The patient does not like the emotional displays, but depending on the tension associated with the athetosis present, the reaction may be more or less marked.

Note: Not all these types of athetosis have been accepted by the Academy, but are listed for the sake of completeness.

C. Rigidity—A disturbance of the agonist-antagonist relations with resistance to slow passive motion of both agonist and antagonist muscles. If the resistance to passive motion is continuous, it is referred to as the “lead-pipe” rigidity—if discontinuous, “cog-wheel” rigidity. The resistance is greater to slow than to rapid motion, whereas, in spasticity, there is greater resistance to rapid motion. In rigidity, the antagonists to the antigravity muscles are most involved. Total motion may be decreased. The main characteristic is hyper-tonicity, normal or diminished reflexes, no clonus, no stretch reflexes and no involuntary motion.

D. Ataxia—Ataxia is primary inco-ordination due to disturbance of kinesthetic or balance sense, or both. Characterized by disturbance in the sense of balance and equilibrium, dyssynergias, and the patient often exhibits the “rebound phenomenon” with astereognosis and depth perception involvement. Atonia and hypotonia may be present.

E. Tremor—Which may be intentional, non-intentional or constant, uncontrollable, involuntary motions of a rhythmic, alternating, or pendular pattern due to alternate agonist and antagonist contractions.

F. Atonia—Lack of tone, and failure of muscles to respond to volitional stimulation. The muscle lacks the firmness or turgor of the normal relaxed muscle. Weak stretch reflex may be obtained as well as increased deep reflexes, but no involuntary motion is present. This distinguishes it from non-ten-
sion athetosis (just a matter of degree). This is rare in pure form, but may be the initial symptom in any of the forms of cerebral palsy. It deserves special mention because it may be the outstanding initial symptom. The atonic form of cerebral palsy, in most instances, does not describe a type, but the outstanding symptoms of a type.

G. Mixed—The mixed class need not be used often, as the predominant motor symptoms determine the classification.

STATISTICAL DATA

Types of Athetosis: The majority of the members of the American Academy of Cerebral Palsy approved of Tension, Nontension, Dystonic, and Tremor-like types of athetosis. The others were rejected for various reasons. Possibly, some of the other types would have been accepted if understood by the members, each type needs further study. (See outline of Athetosis, herein.)

DISCUSSION

The majority of the members of the American Academy for Cerebral Palsy accept a motor classification, listing 6 separate types (7 with the mixed type), followed by the topographic involvement and etiology when this can be determined. The chief advantage of this classification is that it aids the clinician in planning treatment of the patient with cerebral palsy. Proponents of this classification point out that the best a clinician can do is describe the symptoms carefully so that, eventually, anatomical study of post-mortem material can be correlated with the clinical symptoms. Despite its obvious faults, the motor classification is practical for the clinician and for those engaged in the various phases of treating cerebral palsy. We cannot escape the fact that the treatment of cerebral palsy is, largely, the treatment of symptoms, not the treatment of the etiological factor or the treatment of some specific area of the brain. The site of brain lesion is, of course, added whenever this can be determined accurately, by autopsy, surgery, or other methods.

The evaluation of functional capacity of the patient with cerebral palsy, as presented in this paper, is a rough estimate and is subject to many individual variations. In order to make the evaluation more scientific, a careful study should be made similar to the work of McBride on "Disability Evaluation." The details of this type of evaluation should be worked out by specialists in each field in order to insure proper numerical evaluations.

The majority of members returning questionnaires wanted to accept a classification based on motor symptoms. A few members with a good deal of experience in the field of cerebral palsy strongly urge a simple classification, e.g., (1) Pyramidal; (2) Extrapyramidal; (3) Ataxic; and (4) Mixed. One supporter of this classification feels that we are likely to do more harm than good by accepting one more complicated, and that we must not let our classification outrun our understanding. Many are opposed to this type on the premise that the clinician presumes that he knows the approximate site of the lesion.

Most members feel that certain well recognized neurological syndromes such as the progressive neurological diseases and neoplastic diseases should not be included in a cerebral palsy classification, but only the static (non-progressive) neurological states. The field of cerebral palsy is now limited to a more logical and manageable size.

Those who prefer the descriptive or symptomatic (motor) classification must accept a stern word of warning that neurological signs and symptoms change as the nervous system matures and that one must be extremely cautious in making a final descriptive diagnosis in infancy. (Those concerned are urged to read the paper by Byers.4) For example, some infants with cerebral palsy may undergo a sequence of hypotonicity, hypertonicity, and, finally, athetosis, or some other extrapyramidal dyskinesia as the nervous system matures. It is only when the symptoms become static, following maturation of the nervous system, that the descriptive classification is of
value. A further study of changing neurological patterns during childhood and infancy is urgently needed.

As it has been demonstrated that the motor classification must be applied with caution, until there is some degree of maturation of the nervous system, we may profit by making a survey of children with cerebral palsy, followed from early infancy to school age or by comparing initial with final neurological descriptions from first to fifth year of life or later, to determine the pattern of changing neurological symptoms.

Those who have examined large numbers of children with cerebral palsy over many years have had the disconcerting experience of making a diagnosis of "atonia" only to find, in later years, that the child was

<table>
<thead>
<tr>
<th>Type of Athetosis</th>
<th>Approve</th>
<th>Do Not Approve</th>
<th>No Answer</th>
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<tbody>
<tr>
<td>Rotary</td>
<td>16</td>
<td>22</td>
<td>7</td>
</tr>
<tr>
<td>Adduction-adduction</td>
<td>28</td>
<td>12</td>
<td>5</td>
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<tr>
<td>Dystonic</td>
<td>27</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>Shudder</td>
<td>15</td>
<td>23</td>
<td>7</td>
</tr>
<tr>
<td>Flail(ing)</td>
<td>19</td>
<td>18</td>
<td>8</td>
</tr>
<tr>
<td>Tension</td>
<td>30</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Non-tension</td>
<td>22</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Hemiathetosis (topographical)</td>
<td>21</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Neck and arm (topographical)</td>
<td>19</td>
<td>20</td>
<td>6</td>
</tr>
<tr>
<td>Deaf athetoid</td>
<td>18</td>
<td>20</td>
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</tr>
<tr>
<td>Balance release</td>
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<td>23</td>
<td>7</td>
</tr>
<tr>
<td>Emotional release</td>
<td>11</td>
<td>24</td>
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</tr>
</tbody>
</table>

Classification Based on Motor Symptoms
27 of the members preferred listing the types separately, e.g., spastic, athetoid, tremor, rigidity, ataxic, atonic, and mixed.
15 of the members preferred the classification listing 3 main types of cerebral palsy: (1) Spastic (a) Atonic; (2) Dyskinetic (a) Athetosis, (b) Rigidity, (c) Tremor-like; (3) Ataxic; and (4) Mixed.
3 members expressed no preference.

Classification based on Etiology
13 members prefer including progressive neurological diseases.
28 members prefer deleting progressive neurological diseases.
16 members wish to include neoplastic diseases.
26 members wish to delete neoplastic diseases.

Supplemental Classification: These were checked as important enough to be included in the diagnosis by the following number of members (out of 45):
33—intelligence quotient 36—Major sensory disturbances
32—Developmental level (Gesell) 20—Physical status (comparative)
38—Convulsive seizures 24—Posture and locomotive behavior
24—Eye and hand behavior patterns 17—Additional behavior terms
32—Visual status 35—Auditory status

The returned questionnaires indicate the following choices as a basis of classification for cerebral palsy:
First choice: Motor Symptoms
Second choice: Topographic Involvement
Third choice: Etiology
Fourth choice: Anatomical Site
Fifth choice: Severity of Involvement
Sixth choice: Degree of Muscle Tone
Seventh choice: Supplemental Data
athetoid or spastic. Atonia may be an initial symptom of any one of the major types of cerebral palsy. One must look carefully for involuntary motion which, if found, would indicate athetosis. Some feel that the atonic phase may be an initial symptom of all the major types of cerebral palsy. Some children may go through rigidity with opisthotonos, flaccidity and athetosis. Therefore, we must accept the fact that the motor classification in infancy is no more than a tentative appraisal.

Conditions resembling cerebral palsy include such conditions as the “high cervical syndrome” described by Fay. These should probably be grouped under “Spinal Palsy” and the entire neurological group then referred to as “Cerebrospinal Palsy.” The term “Cerebrospinal Palsy” is probably better than cerebral palsy for the whole grouping, because it has been demonstrated that spinal lesions exist alone or in combination with brain lesions in certain types of so-called “Cerebral Palsy.”

The introduction to Standard Nomenclature of Diseases and Operations, now in use by medical record librarians, states:

“The method of classification is based on two elements: The portion of the body concerned (topographic) and the cause of the disorder (etiologic). These two elements are designated by code number separated from each other by a hyphen. The first three digits describe the topographic site; the last three, following the hyphen, describe the etiologic agent. Combined, they form a complete diagnostic code number.”

The above method of coding and classification for cerebral palsy is impractical, and, in some cases, impossible, since the etiology and the anatomical area of the brain can only infrequently be determined accurately. The present edition of Standard Nomenclature of Diseases and Operations can be used for the spastic type of cerebral palsy if it is caused by a disease due to prenatal influence (see page 392, line 4), however, if the spasticity is due to other causes, e.g., postnatal influence, or in other types of cerebral palsy, the coding is unsatisfactory.

In a letter dated October 20, 1954, Ada-line C. Hayden, Associate Editor of Standard Nomenclature of Diseases and Operations, states: “... I would like to state that we expect to have a complete revision of the section on Neurology for the 5th edition. At this time, the conditions which you present would be coded as follows: ... All cerebral palsy is coded cerebral spastic infantile paralysis 933.4-076 with the supplementary terms.”

This would indicate that all types of cerebral palsy are caused by underdevelopment of the brain due to prenatal influence (see pages 49-50, Etiologic Classification, Standard Nomenclature of Diseases and Operations). Given the following clinical diagnoses, the record librarian, using the present edition, would code as follows:

**Diagnosis:** Cerebral palsy, spastic hemiplegia, etiology brain hemorrhage at birth. **Coding:** 933.4-076; 94x-050 Birth injury of extrinsic arteries of the brain, and 969 hemiplegia.

**Diagnosis:** Cerebral palsy athetoid quadriplegia, dystonic type, etiology unknown. **Coding:** 933.4-076; 9211 Athetosis; 96x Quadriplegia, tetraplegia; 9216 Dystonic movements.

**Diagnosis:** Cerebral palsy, rigidity quadriplegia, etiology cerebral anoxia at birth. **Coding:** 933.4-076; 96x Quadriplegia; 540 Anoxemia.

**Diagnosis:** Cerebral palsy, ataxic type, etiology unknown. **Coding:** 933.4-076; 271 Atonia.

**Diagnosis:** Cerebral palsy, atonic type, etiology unknown. **Coding:** 933.4-076; 272 Atonia.

**Diagnosis:** Cerebral palsy, tremor type, quadriplegia. **Coding:** 933.4-076; 96x Quadriplegia; 9228 Tremor.

The proposed classification system, including coding, is as follows:

**Cerebral Palsy “X”**

1. **Physiological (motor)**
   - A. Spastic ......X(S)*
   - B. Athetotic ......X9211
   - C. Rigidity ......X(R)*
   - D. Ataxic ......X271
   - E. Tremor ......X9228
   - F. Atonic ......X272

* Above, indicates that code number is to be assigned.
AMERICAN ACADEMY OF PEDIATRICS – SPECIAL ARTICLE

G. Mixed ........X (plus any combination above)
H. Unclassified ....XY00

II. Topographical
   A. Monoplegia ......X ...-948 (Indicate proper physiological code number)
   B. Paraplegia .....X ...-941
   C. Hemiplegia .....X ...-969
   D. Triplegia ......X ...-978
   E. Quadriplegia ...X ...-96x

III. Etiological (Add to diagnosis when determined)
   A. Prenatal .................0
   B. Natal Anoxia .............-048
   C. Postnatal
      1. Trauma ....................-4
      2. Infection ..................-1
      3. Toxic causes ..............-3
      4. Vascular accidents .........-50
      5. Anoxia ....................-421
      6. Neoplastic ................-8
(When coding etiology, above, indicate agent by proper digits).

IV. Supplemental (may be coded from 'supplementary terms' in nomenclature book)
   A. Psychological evaluation
   B. Physical status
   C. Convulsive seizures
   D. Posture and locomotive behaviour pattern
   E. Eye-hand behaviour pattern
   F. Visual status
   G. Auditory status
   H. Speech disturbances

V. Neuroanatomical (select code number from “Brain,” pp. 38-39-40’)

VI. Functional Capacity (degree of severity)
   Class I
   Class II
   Class III
   Class IV

VII. Therapeutic
   Class A
   Class B
   Class C
   Class D
(Note: VI and VII, above, need not be coded by librarian, but added to the final diagnosis.)

CONCLUSIONS

The majority of the members of the American Academy for Cerebral Palsy voted to exclude progressive neurological diseases and neoplastic diseases of the brain from the classification of cerebral palsy. The lesion left by the removal of a brain tumor, however, is still considered one of the etiological factors of cerebral palsy.

Cerebral palsy comprises the motor and other symptom complexes caused by a non-progressive brain lesion (or lesions).

The nomenclature and classification questionnaires indicate that the members of the American Academy for Cerebral Palsy wish to accept a motor classification, listing each type of cerebral palsy separately: Spastic, Athetoid, Tremor, Rigidity, Ataxic, Atonic, and Mixed.

The following choices as a basis for classification of cerebral palsy were made by the American Academy for Cerebral Palsy:

First choice: Motor Symptoms
Second choice: Topographic Involvement
Third choice: Etiology
Fourth choice: Anatomical Site (of lesion)
Fifth choice: Severity of Involvement
Sixth choice: Degree of Muscle Tone
Seventh choice: Supplemental Data

The first 3 choices above should be used by the medical record librarian and by doctors discharging patients with cerebral palsy from hospitals or institutions so as to establish a common understanding and uniformity to hospital records. It is understood that the neuroanatomical classification (Fourth choice) is to be used when it can be proven, but not by presumption.

The majority of the members approve of tension, non-tension, dystonic, and tremor-like types of athetosis. The other types were rejected for various reasons. Probably, some of the other types would have been accepted if understood by the members. Each type is described and defined herein. There is a general lack of agreement on the various terms used in cerebral palsy. Definitions of these terms are now being made by a committee for a meeting in 1955.

There is a good deal of evidence that neurological signs and symptoms change in the child with cerebral palsy as the nervous system matures and that one must be cautious in making a final descriptive or symptomatic diagnosis in infancy. The pattern of
MINEAR – CLASSIFICATION OF CEREBRAL PALSY

changing neurological symptoms from infancy through childhood should be studied.

The high cervical syndrome described by Fay is being confused with cerebral palsy. This syndrome needs further study. It should be determined whether the term cerebrospinal palsy would not be more appropriate for the entire neurological group.

The American Medical Association's *Standard Nomenclature of Diseases and Operations (Fourth Edition)*, commonly used by medical record librarians, is not suitable for the classification of cerebral palsy. In this edition, all cerebral palsy is coded "Cerebral spastic infantile paralysis" with supplementary terms added to denote various types.

A complete classification for cerebral palsy is presented, using the majority opinion from questionnaires sent to the members of the American Academy for Cerebral Palsy during 1953 as a basis.

REFERENCES

SPECIAL ARTICLE: A CLASSIFICATION OF CEREBRAL PALSY

W. L. Minear

Pediatrics 1956;18;841

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