PAEDIATRIC ASPECTS OF CEREBRAL PALSY

INGRAM

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BY

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E. & S. LIVINGSTONE LTD.

EDINBURGH AND LONDON

1964

PREFACE

THIS book describes the results of a study of children suffering from cerebral palsy born between 1938 and 1953 who were living in Edinburgh on 1st January 1953. The study was suggested and initiated by Professor R. W. B. Ellis, Department of Child Life and Health, University of Edinburgh. Its aims were to ascertain the prevalence of cerebral palsy in Edinburgh and to study its causes and its effects on patients and their families.

During the investigation it became increasingly apparent that the currently defined categories which comprised " cerebral palsy " did not allow for the accurate classification of patients by neurological findings. Gradually a new classification of cerebral palsy by neurological findings was evolved. This classification is described in the first chapter.

Methods of ascertainment. It was decided that an attempt to ascertain directly and indirectly all patients suffering from cerebral palsy born between 1938 and 1952 should be made (Ingram, 1955 *a*). Three methods of case finding were used. Firstly, all doctors and organisations likely to see or have patients suffering from cerebral palsy were asked to refer them for examination. School medical officers and nurses, doctors and health visitors of the Maternity and Child Welfare Department and paediatricians, neurologists, psychiatrists, neurosurgeons, and orthopaedic surgeons working in hospitals and clinics in the city were contacted and asked for help in ascertainment.

Secondly, the case notes of patients who had suffered from conditions likely to result in cerebral palsy or to be associated with it were inspected. As many records as could be obtained were studied of patients who had suffered from birth injury, rhesus incompatibility, head injury, meningitis, encephalitis, cerebral thrombophlebitis, or abscess, mental retardation, epilepsy, overactive behaviour, talipes, and visual and hearing defects. Thanks to the excellent co-operation of consultants in local hospitals and the medical and nursing staff of statutory authorities and voluntary organisations it was possible to examine approximately 8,500 case notes and records.

The third means of ascertainment was by the personal inspection of selected groups of children in which a relatively high prevalence of cerebral palsy might be expected. Schools for mentally defective children and a number of local authority and private schools for normal children were visited. Children were watched at play and teachers and attendants questioned about any particularly clumsy or ungainly children in their care. Approximately 4,000 children were inspected in this way.

Other possible cases of cerebral palsy were sought in a wide variety of hospital and local authority clinics, nurseries and departments of physiotherapy and occupational therapy, which were visited personally every few weeks during the course of the investigation.

As a result of these methods of ascertainment it was possible to prepare lists of children who merited further examination to confirm or refute the diagnosis of cerebral palsy.

Methods of examination. Whenever possible the first examination of children classified as possible cases of cerebral palsy was undertaken in clinics which they were already attending. When it was convenient, children attending child welfare clinics were first examined there and children were seen at the time of routine medical inspection if this could be managed. In these circumstances, it was often possible to exclude the possibility of the child suffering from cerebral palsy without worrying the parents about the need for special examinations of their offspring.

When children were suspected of suffering from cerebral palsy, either on the basis of inspection or as a result of preliminary examinations, arrangements were made for history taking from the parents and a detailed examination of the child. Whenever possible the history was obtained in stages at a number of interviews, so that mothers' memories could be given time to work.

During the interviews with parents a detailed family history was obtained ; mothers were asked about their reproductive history ; full details were requested about the birth of the affected child, his motor, linguistic, adaptive and social behaviour at different ages and the extent of his disabilities ; questions were asked about his illnesses and there was direct questioning about convulsions, abnormalities of play, sleep and feeding behaviour.

Questions were also asked about progress at school and in particular about any specific learning difficulties which had been encountered. Histories obtained from parents or guardians were supplemented by accounts from other relatives and by information gleaned from hospital and clinic records, school teachers' reports and reports from therapists. Notes were obtained from maternity hospitals describing the births of 98 patients.

The methods and the order of examination varied greatly in different cases, but invariably included a detailed neurological examination, a search for associated malformations, and careful observation of behaviour, preferably on more than one occasion and in different surroundings.

Psychometric assessments were not obtained routinely for the purposes of the investigations because most patients of school age had already been assessed fully by psychologists. When psychometric assessments had not been made they were carried out on request by psychologists working for the Education Authority, the Scottish Council for the Care of Spastics or in local hospitals. Electroencephalograms had been obtained in many cases but not in all. Patients suspected of having seizures were subjected to electro-encephalography in the Department of Surgical Neurology by kind permission of Professor N. M. Dott. Similarly when it was felt important to know whether patients might have been rhesus incompatible with their mothers' blood grouping was undertaken by the South-Eastern Regional Blood Transfusion Service. Few special X-rays were requested, largely because many of the radiographic investigations which were thought to be indicated had already been performed.

Results of the survey. As a result of the survey it was possible to ascertain a representative group of children suffering from cerebral palsy in Edinburgh. An estimate of the prevalence of cerebral palsy in the child population was made. The clinical findings of patients and apparent causes of their cerebral

palsy were studied, with reference to the relevant early and contemporary literature. Their problems of adapting to life as handicapped people in the community were examined at the time of the survey and 10 years later in 1962-63, with the help of Miss Stella Jameson, Social Worker, appointed by the Scottish Council for the Care of Spastics.

Some research into the causes of cerebral palsy was undertaken, with the particular aim of defining the significance of perinatal injury as a cause of cerebral palsy. I have tried to show that the causes of cerebral palsy are at least as complex as the causes of stillbirth and neonatal death, and that the importance of " birth injury " as a cause of some types of cerebral palsy has been overestimated. The care of patients suffering from cerebral palsy is not discussed in this book.

In order to avoid having large appendices containing detailed descriptions of patients I have tried to summarise the relevant facts about them in tables placed in appropriate chapters. This policy has inevitably resulted in there being many tables, some of which contain information already tabulated in other chapters. I hope that readers will not find this repetition of information too annoying.

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Edinburgh, 1963.

ACKNOWLEDGEMENTS

THE initial studies of cerebral palsy in Edinburgh were made during the tenure of a George Guthrie Research Fellowship in the Department of Child Life and Health in the University of Edinburgh in 1952 and 1953. Professor Ellis suggested the investigation and gave me every encouragement during it. He has taken a most active interest in the preparation of this book and has kindly read many of the proofs. I owe him most grateful thanks.

I am grateful for all the help I received from the consultants of the Royal Infirmary, Edinburgh, the Western General Hospital, Royal Hospital for Sick Children, Bruntsfield Hospital and Princess Margaret Rose Orthopaedic Hospital. Fortunately they granted ready access to their patients and case notes and often went to the extent of arranging special clinic appointments and investigations for me. I am particularly indebted to Professor N. M. Dott for allowing me to have electroencephalograms performed in the Department of Surgical Neurology, and for his encouragement.

The investigation would have been impossible without the co-operation and encouragement of the Scottish Council for the Care of Spastics and particularly the late A. M. Fraser and Mrs Fraser, Mr G. A. Pollock and Dr James Naughton who gave me much good advice and much information. The Scottish Council for the Care of Spastics supported later research into the employment problems of patients and made possible the investigations reported in Chapter 25.

Dr Boog Watson and his assistants in the School Health Service and Dr H. P. Tait in the Maternity and Child Welfare Service went to a great deal of trouble to help me to ascertain patients and to obtain facilities for their examination. I am grateful to the Director of Education for his permission to visit schools and to Miss A. Paterson for much assistance.

Advice about statistics was freely given by Dr Peter McKinley and Dr C. M. Drillien at different stages in the investigation. I would have been lost without their help.

I should like to thank the many librarians who have been unfailingly helpful in obtaining books and articles, some with considerable difficulty. I have borrowed from the Central University Library, the Central Medical Library, Royal College of Physicians Library, Royal College of Surgeons Library and the National Library of Scotland in Edinburgh; The British Medical Association, National Hospital for Nervous Diseases, University College, Senate Library of the University, and Royal Society of Medicine in London. I am particularly grateful to the Librarians of the Royal College of Physicians in Edinburgh for finding books and summaries and translations of papers which others had regarded as unavailable. My father, Dr F. T Ingram, Mrs Hana Sambrook and Dr Ernst Levin have helped with translations, but I am especially grateful to my wife who has patiently translated many German books and papers, including the works on cerebral palsy of Sigmund Freud, and managed to make sense of them.

I am grateful to the Editors of *Archives of Disease in Childhood*, the *Journal of Mental Science*, *Developmental Medicine and Child Neurology*, *Brain*, and *Acta Paediatrica Scandinavica* for permission to reproduce material which first appeared in these journals.

I am grateful to Mr Clifford Shepley, medical artist, for Figures 1 to 5, 35 to 38, and to Miss C. Brydone for many photographs. The book would never have appeared without the untiring and cheerful secretarial help of Misses Cruickshank, Meiklejohn McKnight and Jenkins of the Department of Child Life and Health, University of Edinburgh. I am grateful to Messrs Charles Macmillan and James Parker of Messrs Livingstone for their patience and helpfulness throughout the preparation of this book.

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Edinburgh, 1963.

CONTENTS

CHAPTER 1

THE DEFINITION AND CLASSIFICATION OF CEREBRAL PALSY

DEFINITION. Cerebral palsy is used as an inclusive term to describe a group of chronic non-progressive disorders occurring in young children in which disease of the brain causes impairment of motor function. The impairment of motor function may be the result of paresis, involuntary movement or inco-ordination, but motor disorders which are transient or are the result of progressive disease of the brain or attributable to abnormalities of the spinal cord are excluded.

This definition is similar to the majority of definitions made by recent writers on cerebral palsy (Crothers, 1951; Wyllie, 1951; Benda, 1952; Andersen, 1954; Courville, 1954; Perlstein, 1955; Mitchell, 1961). Some authors have attempted to define a period of infancy after which cerebral palsy cannot be acquired. Kurland (1957), for example, limits the definition of cerebral palsy to disorders of motor function " recognised prior to the end of infancy (two years)." It seems preferable to avoid such arbitrary age limits to the definition as Polani does: " Infantile cerebral palsy can be defined as a syndrome of non-progressive brain damage caused by factors operating on an immature nervous system manifesting itself at birth or in early postnatal life, showing essentially a non-fitful disturbance of voluntary movements, and frequently revealing associated handicaps—intellectual, convulsive, sensory, emotional and specific educational " (Polani, 1956). A shorter definition than Polani's has obvious advantages, including ease of remembrance.

Cerebral palsy is an arbitrary definition. The disorders it comprises have in common motor dysfunction, but differ widely in aetiology, pathology and associated clinical findings (Freud, 1897). It is an artificially derived category of disease which has a certain administrative usefulness. But cerebral palsy is not a disease entity. It is unfortunate that attempts to treat it as if it were one have resulted in so much fruitless controversy about what it comprises and what it does not.

Since the definition of cerebral palsy is essentially clinical, variations in the use of the term between different writers and clinicians are unavoidable. For example, when mental deficiency or epilepsy are severe in a patient with mild cerebral palsy he may be diagnosed as mentally defective, or epileptic rather than as suffering from cerebral palsy. Such variations in classification are inevitable. More surprising is the inclusion of cases of amaurotic family idiocy, Friedreich's ataxia and progressive hereditary paraplegia under the heading of cerebral palsy in some recent series (Woods, 1957). Unfortunately it is still necessary to examine authors' definitions and usage of the term cerebral palsy very carefully before accepting their findings.

Classification.—Current classifications of cerebral palsy are clinical rather than aetiological or pathological. They have evolved gradually and are still undergoing changes as increasingly fine systematic distinctions between patients in each different category of cerebral palsy becomes possible. A review of the various classifications which have been employed in the past explains many of the present controversies about different classifications and terminologies.

OLDER PATHOLOGICAL AND AETIOLOGICAL CLASSIFICATIONS. Pathologists showed the first consistent interest in children with cerebral palsy and it was inevitable that, initially, clinical classification lagged behind classification on the basis of pathological changes.

Localised cerebral atrophy and cerebral hemiatrophy were noted to be associated with hemiplegia in childhood as early as 1827 by Cazauvielh, who attempted to distinguish between developmental abnormalities of the brain and brain injury. Subsequently other authors showed that it was possible to define a number of different forms of cerebral atrophy; for example, atrophy complicating old haemorrhagic lesions, atrophy accompanying loss of cerebral substance (porencephaly) and atrophy following inflammatory disease. At the same time it was shown that atrophy was not the universal finding at autopsy of children who had suffered from hemiplegia. Conversely atrophy might be associated with other neurological disorders than hemiplegia (Duges, 1826; von Lallemand, 1834; Henoch, 1842; Cruveilhier, 1862; Cotard, 1868).

In later years attempts were made to define clinical syndromes each of which was characteristic for specific changes. Thus Richardière (1885) described a clinical picture which he believed was pathognomonic for lobar sclerosis. Clinical findings on which a diagnosis of porencephaly could be based were described by Brissaud (1896). These and similar attempts to classify cerebral palsy on the basis of presumed pathology were subjected to devastating criticism by Freud (1897). He pointed out that late pathological findings were the result of a combination of the effects of the original damaging agent and of repair processes, and that in many cases the processes of repair effectively concealed the nature of the underlying disease. Thus it was often difficult even for pathologists to define the nature of the original disease which had caused brain damage. It was still more impossible for the clinician to make a reliable neuropathological diagnosis of children with cerebral palsy on the basis of symptoms and signs. After Freud's comments there was less enthusiasm for pathological differential diagnosis in cerebral palsy.

The possibility of classifying cerebral palsy syndromes by aetiological findings has been considered by a number of authors. The most comprehensive scheme was that presented by Sachs (1891) (Table 1). Unfortunately it was not possible to make categories which were mutually exclusive both in their aetiological and clinical findings. Thus hemiplegia could be of prenatal, natal or postnatal origin. From the practical standpoint, the fact that aetiological factors were so commonly multiple or unknown limited the usefulness of this form of classification severely, as Freud emphasised. Freud hoped that as more became known about cerebral palsy it might be possible

to define, within the major categories, subcategories consisting of disease entities with characteristic aetiological, pathological and clinical findings (Freud, 1897). To some extent his hopes are being realised; for example, by the definition of post-kernicteric dyskinesia due to rhesus incompatibility, the spastic paraplegia shown by the prematurely born infant and the clinical and pathological syndromes associated with toxoplasmosis. In none of the important current classifications, however, are aetiological findings used to define main categories, though subcategories of cerebral palsy are sometimes defined by cause (Phelps, 1950; Minear, 1956).

TABLE 1

The aetiological classification of cerebral palsy suggested by Sachs (1891)

Groups	*Clinical forms in order of frequency*
Paralysis of intra-uterine onset	Diplegia Paraplegia Hemiplegia
Birth palsies	Diplegia Paraplegia Hemiplegia Diataxia Cerebellar form
Acute (acquired) palsies . .	Hemiplegia Paraplegia Diplegia Choreic and athetoid disorders, unilateral and bilateral

EARLY CLINICAL CLASSIFICATIONS. It was only when distinctions were made between the symptoms and signs of paralysis of spinal origin and those due to cerebral damage that clinical classification of patients suffering from cerebral palsy became possible. Delpech appears to have been the first to consider the possibility that symmetrical paralysis of the lower limbs dating from early life might not be spinal in origin (Delpech, 1828). It was thirty years later, however, that this condition was confidently attributed to disease of the brain by von Heine in the course of a discussion on the differential diagnosis of spinal paralysis (von Heine, 1860).

The classical papers of Little appeared in 1843 and 1862. His contributions were almost entirely clinical. He realised the danger of abnormal birth to the child and set out to describe the disorders which could result, including three types of paralysis which would now be considered to be forms of cerebral palsy. These were " hemiplegic rigidity," " paraplegia or generalised rigidity " and a condition characterised by " disordered movement." It was only after some years had elapsed that " hemiplegic rigidity " was recognised as being the same as congenital hemiplegia. Charcot, for example, bewailed the fact that he had never encountered a case of hemiplegic rigidity in all the many children he had seen with paralysis affecting one side of

the body. Generalised and paraplegic rigidity became known as " Little's disease " and later " diplegia " (Freud, 1893). Little's category of patients with disordered movement was almost completely ignored for some years.

Though his descriptions form the first classification of cerebral palsy which can be considered adequate, Little himself was not particularly interested in classification. He was more concerned with demonstrating that the various clinical disorders he described were attributable to the same cause—birth injury. In fact, he did not consider paraplegic and generalised rigidity were due to cerebral damage.

Unfortunately it was only towards the end of the century that the work of Little became at all widely known in Germany and France. Even as late as 1897 Freud was unable to obtain a copy of Little's earlier paper. Thus the effect of Little's work on contemporary German and French work has been considerably exaggerated by later English-speaking authors, aware of the interest Little's papers aroused in Great Britain and America.

In France and Germany the tendency to group together non-progressive neurological disorders attributable to birth injury or disease in early childhood was less marked than in England and the United States. Descriptions of generalised rigidity, paraplegic rigidity, acute infantile hemiplegia occurring as a complication of infectious disease, and congenital hemiplegia following birth injury tend to be scattered throughout the neurological and paediatric literature (Henoch, 1842 ; Benedikt, 1868 ; Cotard, 1868 ; Erb, 1877 ; Jendrassik and Marie, 1885 ; Charcot, 1886 ; Marie, 1888 ; Brissaud, 1894 *a*, 1894 *b*). There was greater interest in correlating clinical and pathological findings than in classification. Thus one of the most strenuous controversies was whether paraplegic and generalised rigidity could both be attributed to disease of the brain. Some authors, notably Charcot himself and Déjerine, maintained that paraplegic rigidity was due to disease of the spinal cord, whereas generalised rigidity was due to cerebral abnormalities (Brissaud, 1894 *a* ; Déjerine, 1903). The majority of authors considered them to be one disease, differing only in severity and attributable to abnormalities of the brain (Freud, 1893 ; Ganghofner, 1894, 1895). Contemporary British writers were more concerned with accurate clinical descriptions of the neurological abnormalities shown by patients after birth injury. The classification in general use was essentially that of Little. The categories of " central birth palsies " recognised were hemiplegia, paraplegic rigidity, generalised rigidity and sometimes disorders in which involuntary movements were the major motor disability (Hadden, 1844 ; Ross, 1882 ; Gowers, 1888).

In the United States the term generalised rigidity was not much used. In 1885 Sarah McNutt attributed most cases of congenital cerebral palsy to subdural haemorrhage, the result of birth trauma. When the haemorrhage was unilateral it caused " hemiplegia." When it was bilateral it affected both sides of the body and resulted in " bilateral hemiplegia." The latter term was used by most American authors and included cases which would have been diagnosed as suffering from generalised rigidity in Europe (Lovett, 1888 ; Osler, 1889 ; Sachs and Peterson, 1890).

Hammond described " athetosis," the involuntary writhing movements of the fingers found in some patients with hemiplegia, in 1871. His account stimulated interest in children with involuntary movements complicating other forms of cerebral palsy or occurring as their main disability (Clay Shaw, 1873; Weir Mitchell, 1874; Raymond, 1876; Oulmont, 1878; Audry, 1892).

As a result of their work the definition of a further category of cerebral palsy characterised by involuntary movements became firmly established, though some authors were reluctant to make any precise distinction between this category and bilateral hemiplegia or generalised rigidity in which involuntary movements could also occur. As early as 1876, Gowers had described the various forms of involuntary movements which were encountered in different neurological conditions. He was at pains to distinguish between athetosis, as described by Hammond, and more rapid proximal choreoid and slow proximal dystonic movements in the limbs. He observed that most children who suffered from involuntary movements as their major disability showed movements of choreoid or dystonic type rather than athetosis. In spite of his work the term " athetosis " was applied to all forms of involuntary movements and became the name of the new category. Thus American classification was as follows :—

Hemiplegia.	Bilateral hemiplegia.
Spastic paraplegia.	Athetosis.

The work of Freud marked a significant advance in the classification of cerebral palsy, for he took great care to define his categories precisely. His classification was by neurological syndromes though he hoped that further study might differentiate disease entities which were also aetiologically and pathologically distinct within his clinical categories. Freud distinguished between unilateral and bilateral disorders of motor function. He called disorders in which there was bilateral impairment of motor function " the cerebral diplegias." His classification was as follows :—

Hemiplegia.
Cerebral diplegia, comprising—
- Generalised rigidity.
- Paraplegic rigidity.
- Bilateral hemiplegia.
- Generalised chorea.
- Double athetosis.

Freud emphasised the similarity in aetiological and clinical findings of patients with generalised and paraplegic rigidity and considered that they differed in severity of clinical manifestations rather than in kind. They were characterised by more or less symmetrical paresis of the limbs, more marked in the lower limbs than the upper. There was often associated strabismus, mental defect and epilepsy. A high proportion of patients were prematurely born and a history of birth injury was frequent.

Generalised and paraplegic rigidity or " Little's disease " could be distinguished from bilateral hemiplegia by the greater involvement of the

upper limbs than the lower in the latter, and by the fact that pseudobulbar palsy was more constant and severe in bilateral hemiplegia than in generalised rigidity. Severe mental defect and epilepsy were almost always associated with bilateral hemiplegia.

Freud recognised that additional categories of cerebral palsy might be required and, in particular, suggested that congenital non-progressive ataxia might eventually form one (Freud, 1897). Within 10 years a category of non-progressive ataxic cerebral palsy had been defined precisely by Batten (1903, 1905, 1907).

MODERN CLASSIFICATIONS. The increase of interest in therapy for patients with cerebral palsy since the early nineteen-thirties has meant that many clinicians and therapists with differing outlooks have become concerned with the diagnosis and treatment of the condition. Their differences in attitude are reflected in the different types of classification they have used.

The classifications most widely used in the last 20 years are based on suggestions made by Phelps (1940, 1941, 1943, 1950). Though these have varied in detail from year to year the basis of his classification has remained the same. Phelps abandoned classification by neurological syndromes and substituted for it a classification based primarily on the state of muscle tone and the presence or absence of involuntary movement. Further classification takes account of the number of limbs which are abnormal, aetiological factors, the presumed site of neuropathological changes and associated defects of the senses (Table 2).

The aim of the classification and of others like it is to give a brief description of the clinical manifestations which are likely to be helpful to therapists and others concerned with the practical management of patients. To some extent the classification succeeds in this. For example, the detailed sub-categories of " athetosis " allow almost any patients with involuntary movements to be classified in one or other of them. But in practice it is found that two clinicians seldom place the same patients in the same sub-categories. Moreover, the sub-categories are not mutually exclusive ; patients with involuntary rotatory movements of the limbs, for example, frequently show dystonic movements also. Similarly, the sub-categories in I—" Aspastic," " Spastic " and " Basilar "—are not mutually exclusive and the description " Aspastic Spastic " is hardly meaningful.

There are other more serious criticisms. Categories I and III and the sub-categories within them have changes of muscle tone as their distinguishing criteria. Yet the muscle tone of individual patients varies greatly as they mature, and may alter considerably from day to day and even from hour to hour according to emotional state, environmental temperature, state of alertness or fatigue, position and posture (Ingram, 1959 *a*). A patient with " Little's disease " may be hypotonic for some months after birth, pass through a stage when involuntary mass movements occur when his position is changed, and later show stages when antigravity hypertonus (rigidity) and then true flexor hypertonus (spasticity) are present. Using a classification in which categories are defined in terms of changes in muscle tone, repeated reclassification is required.

TABLE 2

Recent American classification of cases of cerebral palsy based on that of Phelps by Hellebrandt, 1950-51

I. SPASTIC—
- A. Aspastic.
- B. Spastic :
  - (1) Monoplegia.
  - (2) Hemiplegia.
  - (3) Paraplegia.
  - (4) Triplegia.
  - (5) Quadriplegia.
- C. Basilar.

II. ATHETOSIS—
- A. Tension.
- B. Non-tension.
- C. Dystonic.
- D. Flail.
- E. Arm neck.
- F. Deaf.
- G. Shudder.
- H. Hemi-athetoid.
- I. Cerebellar release.
- J. Rotary.
- K. Emotional release.
- L. Tremor.
- M. Unclassified :
  - (1) Paraplegia.
  - (2) Quadriplegia.
  - (3) Monoplegia.
  - (4) Recovered.

III. RIGIDITY—
- A. Intermittent.
- B. Continuous.
- C. Miscellaneous :
  - (1) Hemiplegia.
  - (2) Paraplegia.
  - (3) Triplegia.
  - (4) Quadriplegia.

IV. TREMOR—
- A. Intention.
- B. Constant.

V. ATAXIA—
- A. Cerebellar.
- B. Eighth nerve.

Unfortunately, a large number of classifications in use to-day are essentially modifications of that suggested by Phelps, for example those used by Asher and Schonell (1950), Andersen (1954), Collis *et al.* (1956), Henderson *et al.* (1961). In all of them muscle tone and the presence or absence of involuntary movement are the major initial criteria of classification.

Simpler classification based on categories defined in terms of changes in muscle tone, the presence or absence of involuntary movement and the topographical distribution of the motor defects have been suggested by a number of paediatricians (Evans, 1948 ; Wyllie, 1951 ; Illingworth, 1958 ; d'Avignon *et al.*, 1960). They all have the same defect as the classification proposed by Phelps, that categories are defined primarily in terms of changes in muscle tone. It is true that in the majority of patients adequate descriptive classification can be made using them, but it is impossible to classify a minority satisfactorily. For example, one cannot distinguish between tetraplegic patients with greatest neurological involvement in the lower limbs from those with bilateral hemiplegia in the Illingworth or Wyllie classifications.

Yet this distinction is important enough to lead Evans to make an exception for it in his system of classification (Evans, 1948).

TABLE 3

Classification of Wyllie, 1951

1. Congenital symmetrical diplegia.
2. Congenital paraplegia.
3. Quadriplegia or bilateral hemiplegia.
4. Triplegia.
5. Hemiplegia with the additional qualifications—
   (*a*) Spasticity.
   (*b*) Flaccidity.
   (*c*) Mixed types.
   (*d*) Athetosis.
   (*e*) Ataxy.

TABLE 4

Classification of Perlstein, 1952

By clinical symptoms	*Topographical involvement of extremities*	*By muscle tone*	*Severity*	*Etiology*
Spastic conditions	Paraplegia	Isotonic	Mild	A. PRENATAL : 1. Hereditary : (*a*) Static. (*b*) Progressive. 2. Acquired *in utero*: (*a*) Infection. (*b*) Anoxia. (*c*) Cerebral haemorrhage. (*d*) Rh. factor. (*e*) Metabolic disturbance. (*f*) Gonadal irradiation. B. NATAL FACTORS : 1. Anoxia. 2. Cerebral haemorrhage : (*a*) Trauma. (*b*) Pressure change, etc. C. POSTNATAL FACTORS: 1. Trauma. 2. Infections. 3. Toxic causes. 4. Vascular accident. 5. Anoxia. 6. Neoplasms and developmental defects.
Dyskinesias .	Diplegia	Hypertonic	Moderate	
Choreas .	Quadriplegia or tetraplegia	Hypotonic	Severe	
Athetoids .	Hemiplegia	—	—	
Dystonia	Triplegia	—	—	
Tremors .	Monoplegia	—	—	
Rigidity . .	Double hemiplegia	—	—	
Ataxia . .	Limited to both upper extremities	—	—	

Attempts to produce more comprehensive descriptive classifications have been made. The first of these was that of Perlstein (1952) (Table 4). Classification is by presumed site of pathology, clinical manifestations, topographical description, severity of motor involvement, muscle tone and