REFERENCES

1. VAN BOUWDIJK BASTIAANSE, M. A.: *Am. J. Obst. & Gynec.*, **68**: 151, 1954.
2. BROWN, W. E.: Haas Memorial Lecture, University of Michigan, April 1958.
3. BROWNE, F. J.: *Lancet*, **1**: 115, 1958.
4. COHEN, M.: *Arch. Int. Med.*, **81**: 879, 1948.
5. COPPEN, A. J.: *J. Psychosom. Res.*, **2**: 241, 1958.
6. DICKINS, A. M. *et al.*: *Brit. M. J.*, **1**: 776, 1956.
7. DIECKMANN, W. J.: The toxemias of pregnancy, 2nd ed., C. V. Mosby Company, St. Louis, 1952, p. 364.
8. Editorial Note [EASTMAN, N. J.]: *Obst. & Gynec. Surv.*, **14**: 680, 1959.
9. FEKETE, S.: *Lancet*, **1**: 642, 1958.
10. GORDON, E. S. *et al.*: *Obst. & Gynec.*, **4**: 39, 1954.
11. GOVAN, A. D. T. AND MUKHERJEE, C. L.: *J. Obst. & Gynec. Brit. Emp.*, **57**: 525, 1950.
12. GOVAN, A. D. T. *et al.*: *Ibid.*, **58**: 216, 1951.
13. HOFBAUER, J.: *Am. J. Surg.*, **84**: 394, 1952.
14. *Idem*: *Am. J. Obst. & Gynec.*, **71**: 255, 1956.
15. HOLLAND, E. AND BOURNE, A. W., ed.: British obstetrics and gynaecologic practice, Vol. I, F. A. Davis Company, Philadelphia, 1955, p. 236.
16. JEFFCOATE, T. N. AND SCOTT, J. S.: *Am. J. Obst. & Gynec.*, **77**: 475, 1959.
17. KAKU, M.: *J. Obst. & Gynec. Brit. Emp.*, **60**: 148, 1953.
18. LENNON, C. G. AND GARDINER, J.: *Ibid.*, **65**: 371, 1958.
19. LI, M. C., HERTZ, R. AND BERGENSTAL, D. M.: *New England J. Med.*, **259**: 66, 1958.
20. MAGARA, M.: *J. Internat. Coll. Surgeons*, **14**: 215, 1950.
21. MASTBOOM, J. L.: *Gynaecologia*, **134**: 217, 1952.
22. MOSES, A. M., LOBOTSKY, J. AND LLOYD, C. W.: *J. Clin. Endocrinol.*, **19**: 987, 1959.
23. PAGE, E. W.: The hypertensive disorders of pregnancy, Charles C Thomas, Springfield, Ill., 1953, p. 86.
24. *Idem*: *Ibid.*, p. 83.
25. PEARSON, M. G. AND PINKER, G. D.: *Brit. M. J.*, **1**: 777, 1956.
26. PIKE, L. A. AND DICKINS, A. M.: *Ibid.*, **2**: 321, 1954.
27. ROBINSON, M.: *Lancet*, **1**: 178, 1958.
28. ROSEN, H.: *Chicago M. Soc. Bull.*, **62**: 428, 1959.
29. ROSENBAUM, M. AND MALTBY, G. L.: *Arch. Neurol. & Psychiat.*, **49**: 204, 1943.
30. ROTTON, W. N., SACHTLEBEN, M. R. AND FRIEDMAN, E. A.: *Obst. & Gynec.*, **14**: 322, 1959.
31. SALERNO, L. J.: *Am. J. Obst. & Gynec.*, **76**: 1268, 1958.
32. SCHUURMANS, R.: *Lancet*, **1**: 835, 1957.
33. SCOTT, J. S.: *J. Obst. & Gynec. Brit. Emp.*, **65**: 689, 1958.
34. SOICHET, S.: *Am. J. Obst. & Gynec.*, **77**: 1065, 1959.
35. SOPHIAN, J.: *J. Obst. & Gynec. Brit. Emp.*, **62**: 37, 1955.
36. *Idem*: *Lancet*, **1**: 434, 1958.
37. TAUSSIG, F. J.: *Am. J. Obst. & Gynec.*, **34**: 731, 1937.
38. THEOBALD, G. W.: *Brit. M. J.*, **1**: 422, 1953.
39. VENNING, E. H.: *Clin. Obst. Gynec.*, **1**: 359, 1958.
40. WHITACRE, F. E., LOEB, W. M., JR. AND CHIN, H.: *J. A. M. A.*, **133**: 445, 1947.
41. ZINKE, E. G.: *Am. J. Obst.*, **53**: 226, 1906.
42. *Idem*: *Ibid.*, **63**: 217, 1911.

SPECIAL ARTICLE

AN INTRODUCTION TO THE DIAGNOSIS OF CEREBRAL PALSY AND THE USE OF A PUNCH CARD RECORD*

PRESTON ROBB, M.D.,† *Montreal*

STRICTLY SPEAKING, cerebral palsy can be defined as abnormalities of movement due to disorders of the motor systems of the brain. The causes of the abnormalities may be many and varied, and similarly the disability may vary as to type, degree and location. Although any injury to the brain at any time of life can cause cerebral palsy, here we shall consider only the condition as it occurs in infants or children. It is impossible for any centre with a broad program to limit its facilities to those with lesions of the brain alone. The child with a muscular disorder or spinal cord lesion also needs help which can be provided most practically by the cerebral palsy team. The diagnosis and treatment of cerebral palsy is a team project requiring many experts working in harmony. It is their object to help the child to develop scholastically and socially and to prepare himself for a useful life, a life that is not entirely dependent on the care of others. It is also their desire to help all children and their families who come seeking a "cure". In severe motor and mental retardation, it may mean months of work preparing the parents to accept custodial care of their child. In less severe conditions it may mean convincing the parents that the child does not need a lot of therapy, but should be allowed to develop on his own, naturally, and without overprotection.

The relationship of the child to his environment is evaluated by the social service worker. The intelligence is determined by the psychologist. The motor age is determined by the physiotherapist. His ability to carry out the activities of daily living is evaluated by the occupational therapist. The educational potential is determined by the members of the department of studies. Speech disorders are studied by the speech therapist. Emotional problems are considered by the social service worker, in co-operation with the psychiatrist. The orthopedist determines the need for special shoes, braces, or corrective surgical procedures.

The child with cerebral palsy is often born into a family ill-prepared either emotionally or financially to cope with the innumerable problems that arise. Instead of a normal healthy child, the parents must accept one that falls far short of their ambitions. In this sense the child is unwanted, feelings of rejection and guilt develop, and emotional problems in the child and family are common. It is the desire of the cerebral palsy team to help parents understand these problems, to help them take a relaxed attitude and provide an atmosphere of warmth and affection.

While the roles played by the various therapists will be stressed, it should be emphasized that the most important "therapists" are the parents. It is they who have the greatest influence on the child's development and who carry on treatment at home. At all times they should be considered as part of

*The punch card was prepared with the help and advice of the Medical Advisory Committee of the Cerebral Palsy section of the Canadian Council for Crippled Children and Adults under the chairmanship of Dr. W. Hawke. Punch cards may be obtained from the Canadian Council for Crippled Children and Adults, 31 Alexander St., Suite 115, Toronto.

†From the Cerebral Palsy Division of the Rehabilitation Services of The Montreal Children's Hospital, Montreal.

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the team, and should understand the aims of the group for treatment.

Having completed the evaluation, all members of the team gather, pool information and plan a practical course of therapy, compatible with the patient's potential and the environment in which he lives.

ANATOMICAL CONSIDERATIONS

The motor systems of the brain are complicated and not completely understood. However, a brief description may serve as an introduction to some of the syndromes that are broadly classified as cerebral palsy.

The giant pyramidal (motor) cells of the cortex lie anterior to the central fissure. Cells supplying the foot and leg lie in the upper part of this region near the midline. Below this are the cells supplying the trunk, the arm, the hand and face in that order. Nerve fibres (axones) from all these cells gather together at the internal capsule, pass down through the brain stem to the medulla, cross in a bundle to the opposite side, and pass down the spinal cord to the anterior horn cells. This is referred to as the pyramidal or corticospinal tract. Motor impulses are transmitted from the cortex to the anterior horn cells, and from there the impulse is transmitted along peripheral nerves to the muscles.

Lesions of the motor system above the anterior horn cells tend to cause spasticity, such as one sees in the ordinary hemiplegia or stroke. Damage to the anterior horn cell or the peripheral nerves causes a flaccid type of paralysis, such as one sees in poliomyelitis.

Deep in the brain are collections of neurons which exert a controlling influence on the impulses from the large pyramidal cells. The upper group are called the basal ganglia. Lesions here tend to cause chorea, athetosis, tremor and rigidity. Behind the brain stem, made up of the pons and medulla, is the cerebellum which is the great coordinating centre. Lesions here cause incoordination or ataxia.

It should be emphasized that the role which each area plays in the control of movement is not completely understood. Also, it is seldom that any one system is damaged alone, so that it is difficult and often impossible to say what region of the brain is involved in a particular child with cerebral palsy.

ETIOLOGY

The causes of cerebral palsy may be divided into three large groups: (1) antenatal (from conception to the onset of labour), (2) natal, and (3) postnatal (during infancy and childhood).

ANTENATAL CAUSES

(a) *Hereditary.*—Genetically transmitted defects in the nervous system may be present in the fetus before birth. An example of this is amyotonia congenita, a condition in which there is flaccid paralysis due to progressive degeneration of the anterior horn cells. Other hereditary disorders, although present at birth, may not be manifested until later in life. Different syndromes are produced depending on what part of the nervous system is affected. Thus hereditary spastic paraplegia is due to degeneration of the pyramidal cells and their axones. Friedreich's ataxia is due to degeneration of cerebellar systems and is manifested by a profound ataxia.

(b) *Anoxia.*—Most other prenatal defects in the nervous system are related to defective nutrition or oxygen supply. Anything which interferes with circulation of blood through the placenta or the umbilical cord will produce anoxia in the fetus. Improper implantation of the placenta and maternal anemia or bleeding may produce anoxia. This in turn may cause the death of neurons with subsequent motor defects.

(c) *Other causes.*—It is well known that German measles early in fetal life may produce abnormalities in the nervous system. Other virus infections have been suspected of causing damage but this has never been proved. Nor has x-ray radiation been shown to produce abnormalities in the human, although experimentally defects have been produced. Trauma to the mother does not affect the nervous system of the fetus unless it affects the implantation of the placenta. Nor do emotional disturbances in the mother affect the fetus. Toxemia in the mother may indirectly affect the fetus by causing infarcts or impaired circulation in the placenta.

NATAL FACTORS

(a) *Anoxia.*—Many things can go wrong during labour that can cause damage to the fetal brain. Maternal anoxia or hypotension may prevent sufficient oxygen being delivered to the placenta. Improper implantation of the placenta may cause its separation prematurely, with resultant hemorrhage and severe fetal anoxia. During birth the cord may be twisted or compressed, with resultant anoxia. Long and difficult deliveries, breech deliveries, the practice of holding the baby back during delivery, too heavy sedation, and other difficulties during birth may be the cause of anoxia. Cesarean section itself is not harmful, but the factors which have led to the operation may be the cause of brain damage. Placenta previa is a good example. Mechanical obstruction to breathing or collapse of a lung may also cause anoxia.

(b) *Birth trauma.*—Disproportion between the size of the head and the pelvis and other obstetrical difficulties with or without the use of forceps may cause cerebral contusion and hemorrhages. Sudden changes in pressure such as may occur in a precipitate or Cesarean delivery may cause cerebral hemorrhages.

(c) *Prematurity and other constitutional factors.*—The brain of a premature child is not sufficiently developed to withstand the trauma of birth, and although the head is small, damage may occur. The brain of a premature child may be abnormal before birth, and this abnormality may be in some way related to the premature birth.

Some children immediately after birth develop jaundice due to an Rh incompatibility. The excessive destruction of the baby's red cells causes severe jaundice; this in turn causes a staining of the nuclei of the brain (kernicterus) and their destruction. Usually this can be prevented by immediate and, if necessary, repeated exchange transfusions.

Difficulty in initiating breathing or cyanosis is a common sign of birth injury in a newborn. It is often hard to determine whether the difficulty in breathing is the result of brain damage or the cause of it.

POSTNATAL FACTORS

(a) *Infection.*—Infections, acute or chronic, virus, bacterial or protozoan, may attack the brain, causing motor, sensory, visual and intellectual deficits. Meningitis may cause occlusion of blood vessels, which in turn produces a motor disability. Brain abscess secondary to congenital heart disease, mastoiditis, pneumonia or other infections may be the cause of hemiplegia. Often hemiplegia is associated with an infection and is heralded by a seizure. One cannot be sure whether the paresis is due to an actual inflammatory condition of the brain or to an associated vascular occlusion, such as a cortical venous thrombosis. Probably infection or associated lesions are the chief causes of postnatal hemiplegia.

(b) *Trauma.*—In this day of high-speed travel, trauma plays an increasingly important role in the etiology of postnatal cerebral palsy. At times a relatively minor head injury may be the cause of a serious brain injury. More frequently a motor accident results in skull fractures, cerebral contusion, laceration and hemorrhages. As well as the hemiplegia from major hemispheral lesions, we often see multiple brain stem hemorrhages which cause a most distressing form of bulbar palsy.

(c) *Vascular.*—Spontaneous occlusion of blood vessels in children is surprisingly frequent. It may be due to congenital defects in the vessel walls or congenital vascular malformations. Arterial thrombosis is seen in polycythemia associated with congenital heart disease. Embolism is sometimes the cause of a vascular occlusion. Cerebral hemorrhage is encountered with certain blood disorders of the newborn. Finally, cortical venous thrombosis may be associated with infection elsewhere in the head or body.

(d) *Tumours.*—Tumours in children are frequently malignant and their complete removal or destruction with x-ray is impossible. However, there are tumours that can be removed or arrested successfully, and the child may be left with a motor disability. In these instances, rehabilitation plays an important role in the adjustment of the child.

(e) *Anoxia.*—Cerebral anoxia can occur in children. Such accidents as those involving carbon monoxide, cardiac arrest during operations or near drowning may cause profound brain damage.

CLASSIFICATION

Children with cerebral palsy may be classified according to the type of motor disability, the site or region of the body involved, and the severity of the disorder. There are many different forms, and these may be of mixed types, so that it may be difficult to classify a patient properly. As the child grows older, and the nervous system develops, the type may change.

In the Montreal Children's Hospital clinic a simple classification is used, and if the condition shows mixed characteristics this is indicated. Thus, a child may be "spastic with athetoid movements", or have "choreoathetosis", etc. The types of motor disability are: (1) spasticity, (2) athetosis, (3) chorea, (4) rigidity, (5) dystonia, (6) tremor, (7) ataxia, (8) hypotonia and (9) unclassified.

Spasticity.—Spasticity is characterized by the presence of exaggerated stretch reflexes, increased deep tendon reflexes, a Babinski's sign and a tendency towards contractures. There is a stiffness which prevents voluntary movement, but there are no uncontrolled abnormal movements. This type of disability is usually associated with lesions in the pyramidal tracts. Children born prematurely, who have suffered from birth trauma, or have developed a hemiplegia, most commonly show evidence of spasticity.

Athetosis.—The athetoid patient is characterized by incoordinated, uncontrolled movements and abnormal posture. The movements are relatively slow as compared with those of the patient with chorea. There may be varying degrees of muscular tension. Fixed deformities or contractures are uncommon. It is commonly due to anoxia at or around the time of birth, or to neonatal jaundice, such as hemolytic disease of the newborn. The chief pathological lesions are in the basal ganglia.

Chorea.—The child with chorea has sudden, spontaneous, rapid, uncontrolled movement, occurring during rest or when attempting voluntary activity. It is uncommon as a manifestation of cerebral palsy, but it is seen in Sydenham's chorea as part of the rheumatic fever complex. The lesions are in the basal ganglia.

Rigidity.—Rigidity is characterized by a stiffness of joints owing to an inability of antagonistic muscles to relax. It is sometimes referred to as "lead pipe rigidity". It is due to lesions in the basal ganglia. It may or may not be associated with tremor. It is most common in parkinsonism, but is also found in certain post-anoxic syndromes.

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Dystonia.—Dystonia is a condition in which the extremities, neck and trunk assume distorted, grotesque positions which may be held for varying periods of time. The muscles involved are in marked spasm.

Tremor.—Tremor is a rhythmic, alternating movement which may be present at rest or during movement. It sometimes disappears during voluntary movement, only to reappear when the movement is completed. The lesion is in the basal ganglia, and is seen in parkinsonism and some of the hereditary degenerative disorders.

Ataxia.—Ataxia is a form of incoordination brought about by voluntary or involuntary movement. These patients have a disturbance in their sense of balance and equilibrium. It is usually due to lesions of the cerebellum or cerebellar systems, but it may be due to certain disorders of sensation (e.g. loss of sense of position or labyrinthine disease). Apart from tumours, the most common cause in children is Friedreich's ataxia, a hereditary degenerative disorder of the cerebellar system.

Hypotonia.—In some very young children with brain injury, the most outstanding feature is the lack of muscular tone or hypotonia. They have been variously described as the "limp child" or the "flaccid child". As the child grows older, the tone may become normal, or there may be spasticity or some other motor disorder. Other children, with a reduced number or an absence of anterior horn cells, have a motor disorder varying from diminished tone to complete flaccid paralysis with absent deep reflexes. This is the so-called "lower motor neurone disease", and is seen in amyotonia congenita, poliomyelitis and the Guillain-Barré syndrome.

Unclassified.—In the very young child, when the nervous system is immature it is often difficult to classify these disorders. It is frequently better to leave them "unclassified" until the brain and spinal cord are more completely developed and the clinical picture is clearer.

SITE OR REGION OF INVOLVEMENT

Paraplegia.—This involves the legs only. It is usually spastic, although if it is due to destruction of the lower end of the spinal cord, there will be a flaccid paralysis. To avoid confusion, we have stopped using the word "diplegia". This term implied chiefly involvement of the legs, but there could be minimal involvement of the arms as well.

Quadriplegia.—This involves both arms and legs, and frequently there is involvement of the face and throat. The degree of involvement may differ in the face, arms and legs. These children may be spastic, athetoid or ataxic.

Hemiplegia.—This indicates a lesion in one hemisphere of the brain and a paralysis or paresis of the extremities on the opposite side of the body. The extremities are usually spastic.

Monoplegia.—This indicates the involvement of one limb only. It is seldom seen associated with cerebral lesions but is frequently encountered after birth injuries to the brachial plexus.

Double hemiplegia.—This is a useful term for a disorder in which both sides are involved, but in which one side is affected to a more marked degree than the other.

No motor disorder.—Not infrequently, a child presents without any motor disorder, but is suffering from aphasia or hemianopsia as a result of a brain injury.

DEGREE OF INVOLVEMENT

It is often difficult to define the degree of involvement, as the legs may be severely affected, the arms moderately so, and the facial muscles only minimally involved if at all. However, it is useful to attempt to classify the severity of involvement, if possible.

Mild.—The patient has some form of cerebral palsy but there is no limitation of normal activity.

Moderate.—The involvement is severe enough to handicap the individual in walking, self-help and communication, but not enough to disable him entirely.

Severe.—The involvement is severe, resulting in total incapacity with almost no useful function of the legs or arms and very limited speech capacity. For example, in the case of hemiplegia, "mild" implies that the hand carries out useful activity; "moderate", that the hand serves as a helping hand; and "severe", that the hand serves no useful purpose.

ASSOCIATED DEFECTS

In the diagnosis and evaluation of a child with cerebral palsy one should attempt to analyze the total problem. After determining the etiology, the nature, the site and severity of the motor disability, the associated defects, such as visual and auditory defects, and disorders of the teeth, heart or other congenital defects, are considered.

Visual.—The most common defect in the eyes is an internal strabismus (esotropia). It is important to recognize this and to seek the aid of an ophthalmologist to protect the vision in both eyes, until such time as operation can be carried out. Diminished visual acuity is often first suspected when the teacher finds the child holding objects close to the eyes. Glasses may be a great help in overcoming this difficulty. A child with a hemiplegia should always have the visual fields tested to make sure that there is not a hemianopsia, or loss of vision on the side of the paralysis.

Auditory.—Hearing defects are common, particularly in children who have had neonatal jaundice due to hemolytic disease of the newborn. Hearing may be impaired in a child who has stopped speaking after an illness such as meningitis. To determine the auditory acuity requires the help of an

expert otologist and an audiogram. It is often difficult to determine whether a child is deaf, whether he can hear but pays no attention, or whether he is unable to comprehend what sound he hears. Children with cerebral palsy are prone to sinusitis, mastoiditis, adenoiditis and tonsillitis. This is often attributed to their ability to clear the nose or throat properly. Here again the otolaryngologist can be of help.

Dental.—Defective decidual teeth with pitting are almost always present in children who have had severe neonatal jaundice. The alignment of the teeth may be defective owing to excessive thumb-sucking, prolonged use of the bottle or unknown reasons. Ordinary cavities in the cerebral-palsied child may present a special problem. Frequently it is necessary to employ a general anesthetic to overcome the spasticity or abnormal movements before the dentist can do his work.

Congenital heart disease.—Children with congenital heart disease may be born mentally deficient, and have one or other type of cerebral palsy. It is not uncommon for them to develop a hemiplegia due to cerebral thrombosis, embolism or brain abcess. A treatment plan must be worked out in consultation with the cardiologist.

Genitourinary problems.—Bladder training of the child with cerebral palsy is often difficult. It may be dependent upon the intelligence of the child, the presence of a spinal lesion and the emotional environment in which he lives. The child with a spina bifida or meningocele presents a particularly difficult problem. It is fair to say that there is no satisfactory solution to the problem of incontinence. Careful watch must be maintained for evidence of pyelonephritis, cystitis, and renal or bladder stones. Here the urologists are of great help. They have devised operations such as the ileal bladder for the incontinent state, but a great deal of attention is, nevertheless, required in order to prevent complications.

Epilepsy.—Epileptic seizures are the result of brain damage. They may take the form of minor attacks or generalized convulsions. A "grand mal" seizure infers a generalized convulsion with loss of consciousness and tonic or clonic movements of the arms and legs, frequently with tongue biting and incontinence. A "petit mal" attack indicates a transient loss of consciousness or "absence", without a generalized convulsion. There may be minor twitchings of the extremities and rolling of the eyes. A focal epileptic attack generally starts with abnormal movements in one extremity and progresses to involve all the extremities. Loss of consciousness is not a necessary feature. A focal attack may start in any area of the brain and remain localized in that area or spread to other areas. Typical would be the attack starting in the visual area, with flashing lights; the sensory attack with focal paresthesias; attacks characterized by loss of speech originating in the speech areas; and the psychomotor seizure originating in the temporal lobe, consisting of attacks of automatism or strange behaviour. Epileptic attacks may or may not be associated with behaviour disorders or mental retardation. An electroencephalogram helps to locate the site of abnormal electrical potentials. If the seizures are few and far between, it is sometimes better to withhold medication. If the seizures are frequent, they can usually be controlled by the judicious use of anticonvulsant medication, such as phenobarbital, diphenylhydantoin (Dilantin), trimethadione (Tridione), primidone (Mysoline), methylphenylethylhydantoin (Mesantoin) methsuximide (Celontin), mephobarbital (Mebaral), phensuximide (Milontin). Parents should be warned that it is necessary to adjust the medication until a combination is found that is not toxic to the child and at the same time affords good control of the seizures. Occasionally, in a hemiplegic child, the seizures cannot be controlled by any form of medication. In these cases serious consideration should be given to surgical excision of the epileptogenic areas of the brain.

SUMMARY

The purpose of this article has been twofold: Firstly, to provide a simple introduction to "cerebral palsy". It is emphasized that the term "cerebral palsy" is not a diagnosis, but simply a term to cover a multitude of conditions in children that result from abnormalities in the motor systems of the brain. Possible etiological factors are outlined. A classification, including type, site of involvement and severity, is given. Some of the associated defects are presented.

Secondly, a punch card is introduced as a manner of indexing and to aid in the follow-up of patients.

APPENDIX

INSTRUCTION FOR USE OF PUNCH CARDS

The card illustrated in Fig. 1 has been prepared to provide a way of indexing the various types, causes and abnormalities commonly found in patients with cerebral palsy and to establish a uniform indexing system across Canada. It is not intended as a form on which the whole history can be recorded. It has been developed in order to maintain records of the various types of cerebral palsy under the care of any particular clinic. For those interested, cards may be obtained from the Canadian Council for Crippled Children and Adults. The suggestions below may serve as a guide for those using the cards.

Anatomical Diagnosis

In most patients it is impossible to define clearly an anatomical site for the lesion. There are some patients, however, with definitely localized lesions, and it is important that they should be classified.

The term "brain generally" should be used in cases in which the lesions appear diffuse and difficult to localize, where there appears to be a combination of several syndromes, or where there is gross intellectual retardation in addition to the motor disability.

"Pyramidal system" is used in patients with spasticity alone. "Basal ganglia" applies to patients with athetosis

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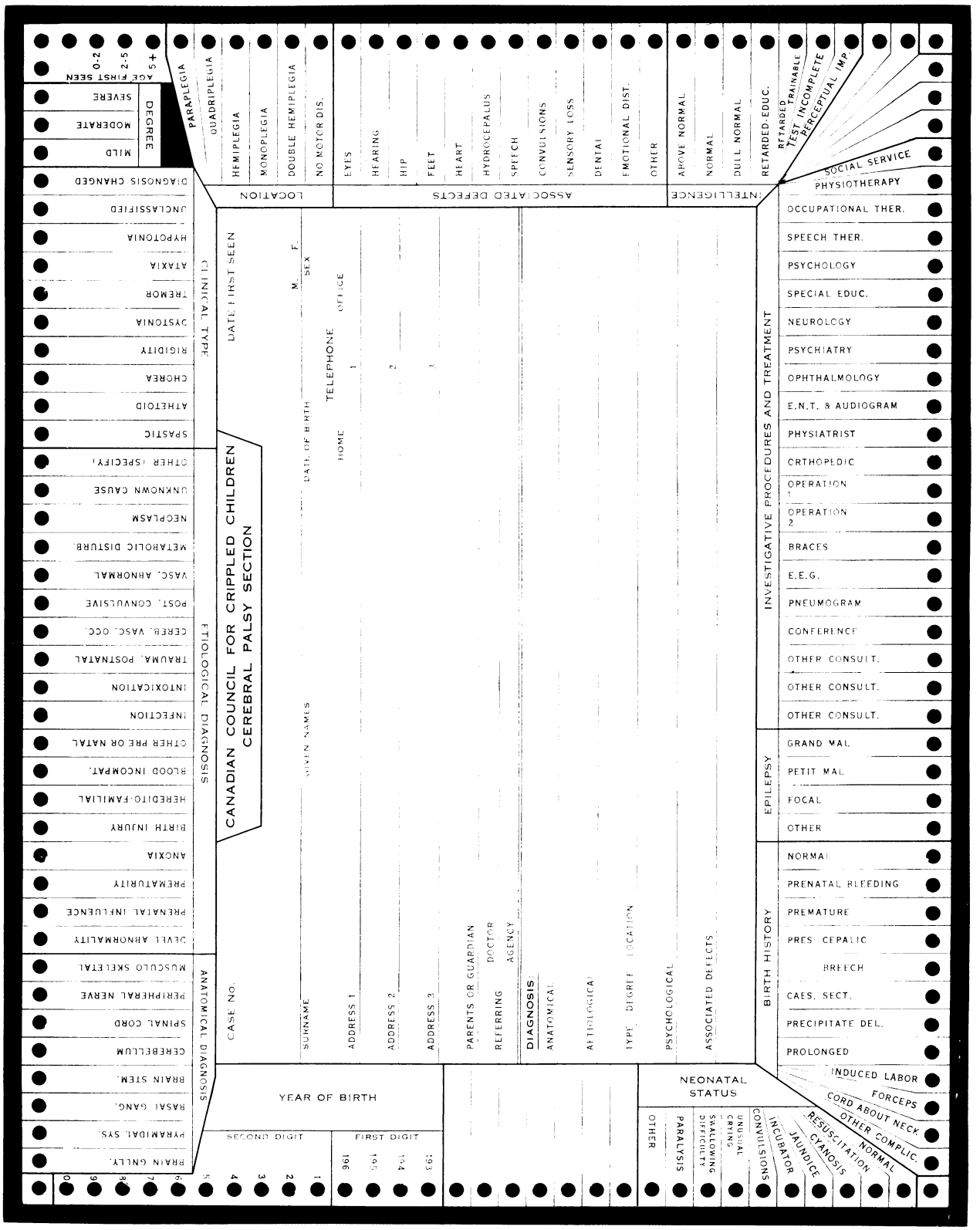

Fig. 1

and chorea. In patients with ataxia, the cerebellum is indicated. Although lesions of the spinal cord or peripheral nerves do not cause cerebral palsy, patients with meningoceles, Guillain-Barré syndrome, etc., also require rehabilitation and may be treated in a cerebral palsy clinic.

Etiological Diagnosis

The list provided does not cover all diagnostic possibilities, but the major groups are indicated. It is intended that a complete etiological diagnosis should be written out in the space provided. If two causes are suspected, both holes should be punched. "Develop-

ment Abnormalities" should be used where the condition is believed to have been present at birth and when it cannot be definitely classified as due to prenatal influence or be of heredito-familial origin.

"Prenatal Influence" is to be recorded when it is felt that the cerebral palsy is due to some intrauterine environmental defect, for example, abnormalities of the placenta, intrauterine infection, etc. If there is intrauterine infection, it should be coded under both "Infection" and "Prenatal Influence".

"Heredito-familial" should be used where there is a positive family history of a similar condition or the disorder is unquestionably an heredito-familial condition.

Clinical Type

The difficulty of defining the type of motor disability in a very young child is recognized. In cases in which it is difficult to assess the clinical type, it may be necessary to punch two classifications. As the child grows older and the type appears to change, this can be indicated on the card.

The definitions of the various clinical types are described above.

Location

This has been kept as simple as possible.

The term "paraplegia" is used to include cases with symmetrical involvement of the lower extremities, so frequently noted in premature infants.

"Double hemiplegia" is used when both sides are involved but one side is much more affected than the other.

A more accurate and descriptive diagnosis can be recorded on the face of the card. The back of the card is available for any other details or unusual features concerning a particular patient. Since the card has been printed, experience has shown that it is useful to record deaths, discharges or placements on one of the blank spaces on the left-hand side.

CASE REPORT

SARCOMA BOTRYOIDES
REPORT OF TWO CASES

E. W. GAUK, M.D., *Edmonton, Alta.*

SARCOMA BOTRYOIDES is a descriptive term applied to a rare malignancy occurring primarily in children. In a comprehensive review of this disease McFarland[1, 2] points out that there are about as many terms applied to this tumour as there are cases reported. Presently there exists no uniformity of thought regarding tissue origin, nomenclature or classification of this growth. It is for this reason that reviews of the literature contain a marked discrepancy in the number of cases reported, and that cases included in one study would be considered unsuitable for another. Furthermore, individual case reports have been duplicated in separate series, adding to the confusion of subsequent reviews of the literature. There are, however, two concepts regarding this disease that are generally agreed upon. Firstly, the term sarcoma botryoides is perhaps a good one, since the clinical recognition of the grape-like nature of the tumour usually makes the diagnosis. Secondly, the tumour does arise from embryonic mesenchymal tissue and, therefore, does have the propensity to differentiate into varied histological elements.

Sarcoma botryoides most frequently involves the urogenital system of young children. About 60% of these mesodermal tumours occur under the age of two years,[3] and several cases have been observed at birth.[4] Novak[5] has illustrated and described the commonest mode of presentation. "This sarcoma presents as a grape-like mass of pinkish, edematous polyps involving the cervix and usually also the upper vagina." With comparable frequency, sarcoma botryoides is known to occur in the bladder.[6] Cases involving the prostate,[7] nasopharynx[8] and common bile duct[9, 10] are also reported.

Although sarcoma botryoides is definitely rare, it constitutes the majority of urogenital malignancies in children.[11] Urogenital carcinoma in children is exceedingly rare.[12] The rarity of this malignancy is further emphasized when one realizes that the records of several major medical centres[13, 14] contain but a handful of cases.

It is hoped that the two cases submitted here will illustrate the clinical features of sarcoma botryoides as it occurs in the bladder and in the vagina.

CASE 1.—This white girl was brought to hospital at 11 months of age with a severe urinary infection of two weeks' duration. After antibiotic therapy she was well enough to be sent home within a week. She again became extremely ill a week later. Her temperature rose to 106° F. (41° C.) and she appeared markedly dehydrated. Severe diarrhea preceded admission, and it was noted that she would pass urine only with straining and with much pain. Culture of the cloudy urine grew *Staphylococcus aureus*. Proteinuria was also present. The white blood cell count was 11,000 per c.mm. and the hemoglobin value was 11 g. %. An intravenous

*From the Department of Pediatrics, University of Alberta. The author is presently a resident in pediatrics at the Children's Memorial Hospital, Chicago.