JOINT MEETING No. 3

Section of Neurology with Section of Orthopædics and Section of Pædiatrics

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DISCUSSION ON INFANTILE CEREBRAL PALSIES

Dr. R. M. Norman (Stoke Park Colony):

Pathology and Ætiology

During the past sixteen years I have examined post-mortem 45 brains of individuals who had suffered from cerebral palsy and I hope to further the purposes of this discussion by analysing briefly the results of this work and by describing some of the commoner pathological findings. This material is derived mainly from certified mental defectives at Stoke Park Colony, only 5 cases having come from general hospitals. As regards the method of clinical selection, all the patients in this group showed severe disorders of motor function and I have not included any examples of the mild generalized spasticity so frequently found in low-grade mental defectives and for which, so far as I am aware, there is no recognized pathological basis. As will be seen in Table I, the younger age groups predominate—a reflection of the severity of many of the cerebral lesions presented by the series as a whole. On clinical grounds this group may seem to have little in common with those more fortunate patients who are capable of benefiting from a programme of treatment in a cerebral palsy unit. Yet I believe it not unlikely that lesions of the same type may well be present in both classes of patient, though doubtless the extent and localization of the cerebral damage differ.

TABLE I.—DEATHS FROM CEREBRAL PALSY

Age at death (years)	*No. of cases*
0–9	19
10–19	14
20–29	6
30–54	6
	45

TABLE II.—MALFORMATIONS ASSOCIATED WITH CEREBRAL PALSY

Polymicrogyria	6
Porencephaly	2
Cerebellar atrophy (granular layer type)	2
Tuberose sclerosis	1
Sturge-Weber syndrome	1
Malformation of caudate nuclei	1
	13

The malformations comprised 13 cases or roughly 30% of the group (Table II). Polymicrogyria was found in 6 brains and was also present to a less extent in the 2 cases of porencephaly. It seems advisable to denote by "polymicrogyria" the special prenatal cortical anomaly referred to here since the term "microgyria" has been so loosely used in the past. Crome (1952*a*) has recently given an excellent account of the condition but perhaps a brief description of its main features may not be out of place in view of the fact that it is still sometimes confused with atrophic lobar sclerosis. The coarse aspect of polymicrogyria is readily identified by the finely wrinkled or bossed surface of the rather wide convolutions often found in these brains (Fig. 1), an appearance aptly likened to a chestnut kernel by Greenfield and Wolfsohn (1935). On section the reason for the name becomes apparent—one sees a wealth of small plications formed by bands of nerve cells lying deep to the surface of the brain. In one well-recognized variant the primitive cortex is composed of two such layers of nerve cells separated by a conspicuous fibre lamina (Fig. 2). The deeper of the two layers of nerve cells was regarded as a heterotopic formation by Bielschowsky (1915, 1923)—an indication, therefore, that a slowing up in the migration of neuroblasts from matrix to the periphery had taken place during the early formative phase of cortical development. In the 6 cases under review polymicrogyria was associated with symmetrical spastic paresis in three instances, with triplegia once and with hemiplegia twice. In the hemiplegias the malformation was strictly unilateral and the pyramidal tract of the affected hemisphere absent or greatly reduced in size. From the clinical standpoint small-headedness is by no means the rule—indeed 2 of the bilateral cases were complicated by a mild internal hydrocephalus. Congenital dislocation of the hips was recorded in one of these

cases and in another the presence of a pre-natal malformation had been suggested during life by bilateral coloboma of the iris. The parents of this child were first cousins but I can find no suggestion of genetic causation in the literature of the condition.

The remaining 32 cases in which no malformation of prenatal origin was found have been classified according to the main site and character of the lesions (Table III). Destructive processes

TABLE III.—PREDOMINANT SITE AND TYPE OF LESIONS IN PARANATAL CEREBRAL PALSY

Cerebral cortex (i) Cystic degeneration	13
(ii) Sclerotic atrophy	12
Lesions of corpus striatum and/or thalamus ..	5
Cerebellar atrophy (Purkinje cell type)	1
Thrombosis of sagittal sinus (recent)	1
	32

affecting the cerebral cortex and subjacent white matter predominate. In the absence of clear-cut signs of malformation pathological criteria alone are usually insufficient to allow one to determine with confidence whether the damage to the brain has occurred during the later stages of pregnancy, during birth or in early infancy. Every case has to be interpreted in the light of a good obstetrical or neonatal history—hence the advantage of examining brains of younger patients. The histories of 13 cases were sufficiently detailed to make the diagnosis of birth injury highly probable. In 8 others the pathological findings were similarly suggestive of anoxic or vascular damage but the birth histories were either not known or not suggestive of trauma. It must be realized, however, that serious birth injury may take place in apparently normal labour (Brouwer, 1949). In 3 additional cases, all examples of extensive cystic degeneration of the brain, the obstetrical histories suggested that anoxia during late pregnancy had played a contributory or even a decisive part in the pathogenesis. In one case the mother had been grossly anæmic and had suffered from transfusion rigors after which the fœtal movements ceased. In another there had been a retroplacental bleeding of considerable size while in the third case the survivor was one of twins, the other having been dead for several days at the time of birth. Such possibilities of intra-uterine anoxia should certainly be borne in mind in cases of presumed birth injury and made the subject of special enquiry. From the point of view of ætiology it is interesting to note that of the twelve patients in this paranatal group whose birth weights were known no fewer than six were premature in the sense of having weighed 5½ lb. or less at birth, while 15 out of 22 were first-born. These findings indicate that cerebral palsies are, in a substantial proportion of instances, birth palsies, to use Gower's expression, and that even in a severely mentally defective group of patients such as this malformation plays a less important role.

Little's (1862) theory that neonatal asphyxia is an important cause of cerebral palsy has recently been greatly expanded by Courville (1950) who sees in cerebral anoxia a probable pathogenetic factor common to a wide range of neonatal nervous disorders. Courville would also restrict the term "birth injury" to gross lacerations of the brain and its coverings or blood vessels. I believe it less pedantic to use the term birth injury in a wider sense so as to include all forms of cerebral damage sustained during birth. These are now known to vary from a patchy or laminar loss of nerve cells to massive softening of the greater part of both hemispheres. The strains and stresses of parturition obviously entail risks other than those of neonatal asphyxia. Apart from direct injury to blood vessels causing hæmorrhage or thrombosis there are the dangers of venous stasis as emphasized by Schwartz (1924, 1927) and before him by Holland (1920, 1922). There is the allied and highly important factor of œdema of the brain set up by serous transudates from vessels damaged by anoxia (Hallervorden, 1939). There is also the possibility that the nerves supplying the walls of blood vessels may themselves be injured at birth so that a later and perhaps progressive degeneration of tissues may result from functional circulatory disturbances (Wohlwill, 1936). Finally, there is Rydberg's (1932) theory that cerebral birth lesions commonly follow a drop in arterial blood pressure below a critical point, this failure to maintain an efficient circulation being due to extreme pressure upon the fœtal skull or to sudden and violent fluctuations in intracranial tension. The following example of cystic degeneration of the brain may perhaps be best explained along the lines of Rydberg's hypothesis:

A first-born, male infant, was delivered in a Liverpool Maternity Hospital after a labour lasting 18½ hours with a second stage of 2 hours 10 minutes. The mother had some contraction of the pelvic brim and trial labour rather than Cæsarean section had been decided upon since the baby was judged not to be large. Delivery was normal and the birth-weight 6 lb. 6 oz. The infant was limp at birth but responded rapidly to having the air passages cleared and after the administration of oxygen and carbon dioxide. Six hours later twitching of the feet was noted. The baby was feeble and reluctant to feed and five days after birth became semi-comatose and was found to be in a state of flexor spasm. The pædiatrician who saw him at this time considered that severe intracranial trauma had occurred. When admitted to the Colony at the age of 4½ years he was an idiot with spastic quadriplegia and epilepsy and death occurred two years later. The brain weighed only 507 grammes and showed extensive bilateral softening of the cortex and subcortical white matter mainly within the distribution of the middle cerebral arteries (Fig. 3).

There is, however, an important group of cases in which sclerotic atrophy of convolutions is found bilaterally in the neighbourhood of the sagittal sinus and in these it is plausible to seek the origin of the lesions in some obstruction to the venous return—as in Schwartz's theory of venous stasis. In 5 of my cases which were characterized clinically by cerebral blindness both calcarine areas and the adjoining superior parietal lobules were affected by ulegyria. Such shrunken and sclerotic convolutions almost invariably exhibit greater damage in the parts hidden below the surface of the brain (Fig. 4) and this is also true of the cerebellar ulegyria sometimes caused by birth injury.

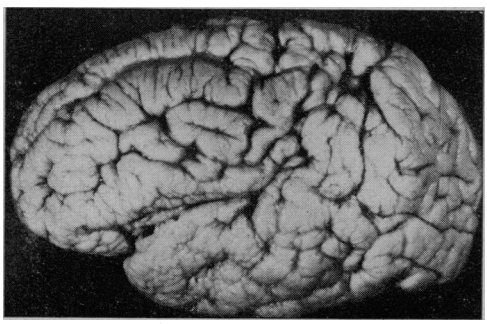

FIG. 1.—Polymicrogyria. Typical "chestnut kernel" brain.

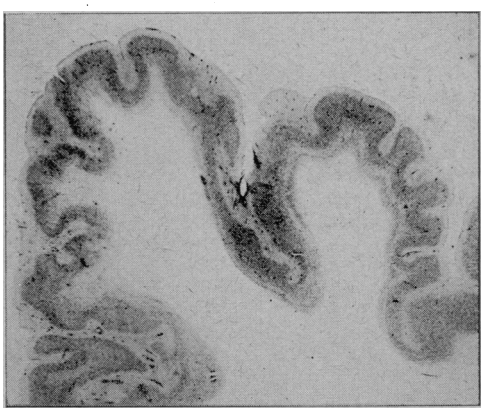

FIG. 2. — Microscopical appearance of polymicrogyria. Note the multiplicity of small convolutions and the great reduction in the number of sulci; also the two layers of cortical nerve cells on the right. (Cresyl violet × 2·5.)

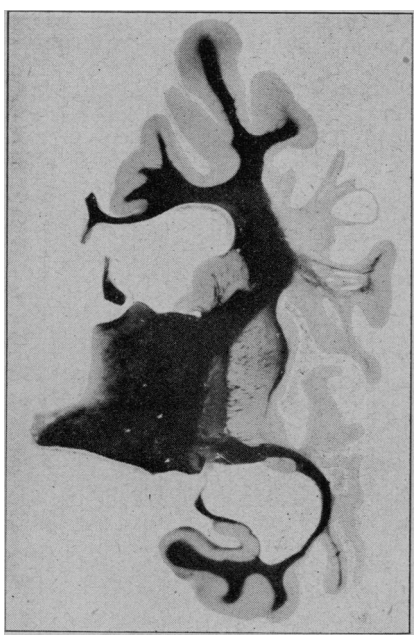

FIG. 3. — Cystic degeneration of the cerebral cortex and subcortical white matter. (Kultschitsky-Pal stain. Natural size.)

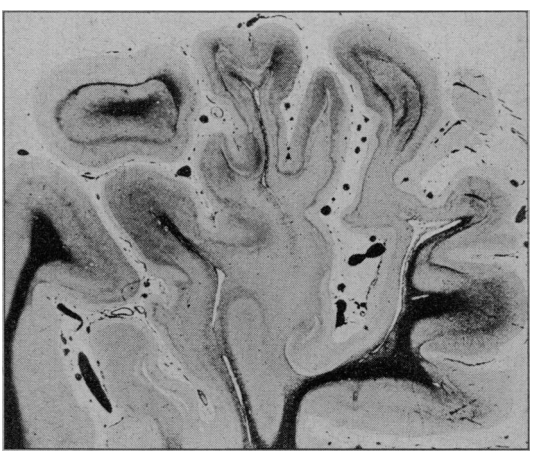

FIG. 4.—Typical ulegyria. Note the greater destruction of the deeper parts of the gyri. (Heidenhain's stain for myelin × 3·7.)

Hallervorden (1939) has attributed this special vulnerability of the gyral wall to the mechanical effect of compression of the cortex against the skull. Blood will tend to be driven into the vessels of the sulci and the ill-effects of vascular congestion and stasis upon the nerve cells may be aggravated by transudation of œdema fluid which will interfere still more with oxygenation. Such a waterlogging of the tissues is also thought to be especially likely to involve the relatively loosely constituted third layer of the cortex and to be an important factor in the production of laminar atrophy.

Another lesion characteristic of birth injury—and one peculiarly damaging to the growing brain—is the paraventricular softening of the central white matter which was so well described by Schwartz

(1924) and related by him to bleedings from or stasis in the radicles of the internal cerebral veins (Fig. 5). Spongy softenings or actual cavities may be found along almost the whole length of the centrum semiovale, from frontal to occipital poles. The cortex is thus undercut and the pyramidal tracts destroyed or inhibited in their post-natal growth. In certain cases, this type of lesion expresses itself in a thinning of the white matter with wide dilatation of the lateral ventricle without there being much appreciable atrophy of the cortex itself to account for this marked degree of hydrocephalus *ex vacuo*.

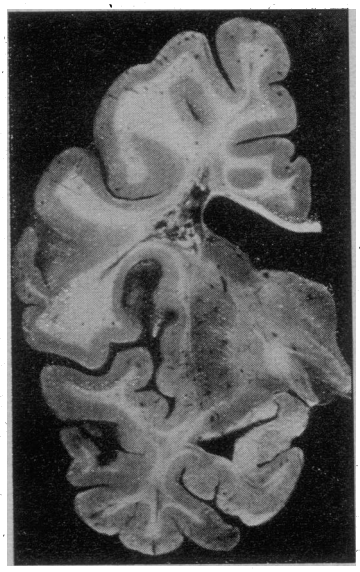

FIG. 5. — Coronal section of a hemisphere showing long-standing paraventricular softening.

Any discussion of the subcortical lesions of birth injury would be incomplete without some reference to *état marbré* of the corpus striatum—the commonest pathological finding in athetosis. The consensus of opinion, including the Vogts' (1951), now favours the view that this condition is not a prenatal malformation but the consequence of vascular or anoxic lesions occurring at birth or less often in early post-natal life. The abnormal networks of myelinated fibres are usually seen in regions showing conspicuous nerve cell loss and gliosis (Fig. 6) and similar evidence of a destructive process is often to be seen elsewhere in the basal ganglia or cerebral cortex of the same brain. In all probability the athetoid postures often assumed by the hand in young hemiplegics are the consequence of coincidental involvement of the corpus striatum of the damaged side of the brain.

In contrast to the predominantly neostriatal distribution of lesions in *état marbré* the late neuro-pathological sequelæ of kernicterus are seen typically in the subthalamic nucleus and globus pallidus (Fig. 7), structures seldom severely affected by birth trauma. In the only survivor of hæmolytic disease due to rhesus incompatibility recorded in this series—a child of 17 months with severe athetosis and head retraction—I found the globus pallidus, subthalamic nucleus and Ammon's horn of both sides selectively and symmetrically damaged.

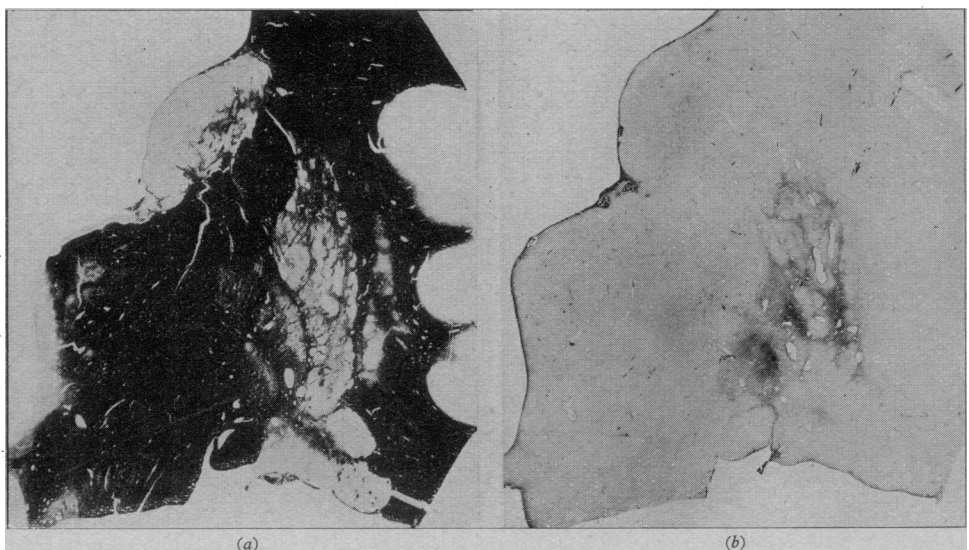

FIG. 6.—*État marbré*. Note in (*a*) the abnormal myelinated fibres in putamen and caudate nucleus. The lateral part of the globus pallidus shows slight myelin loss. (Kultschitsky-Pal × 1·9.) In (*b*) the corresponding neuroglial overgrowth is seen. Note the extensive involvement of the globus pallidus. (Holzer stain × 1·9.)

In my small series of 9 cerebral palsies of post-natal origin 8 were hemiplegics, 6 being examples of acute hemiplegia ushered in by a succession of fits in a hitherto apparently healthy infant. The pathological features of these brains included false porencephaly, atrophic lobar sclerosis and hemiatrophy of the brain with intact pyramidal tracts. In all these well-known conditions vascular lesions were probably responsible for the initial damage, as in the following case:

A 9-year-old idiot previously without signs of spasticity or epilepsy suffered from mild fever and diarrhœa for two days and then had a succession of major fits involving the right side of the body. Although the convulsions were arrested by treatment she remained semi-comatose and died twenty hours later. Six hours before death she exhibited signs of right-sided hemiparesis with increased tone and exaggerated tendon reflexes in upper and lower limbs, a positive Babinski response and tonic innervation of the right hand. At post-mortem the brain showed thrombosis of the middle part of the sagittal sinus and also of both rolandic veins, the left one being the more extensively affected. There was a widespread hæmorrhagic infarction of the left hemisphere with a minimal bleeding on the right side.

This case is of topical interest, in view of Mitchell's (1952) paper on hemiplegia following venous thrombosis and also the observations on the association of gastro-enteritis and cerebral palsy made by Crome (1952*b*) and by Schlesinger and Welch (1952).

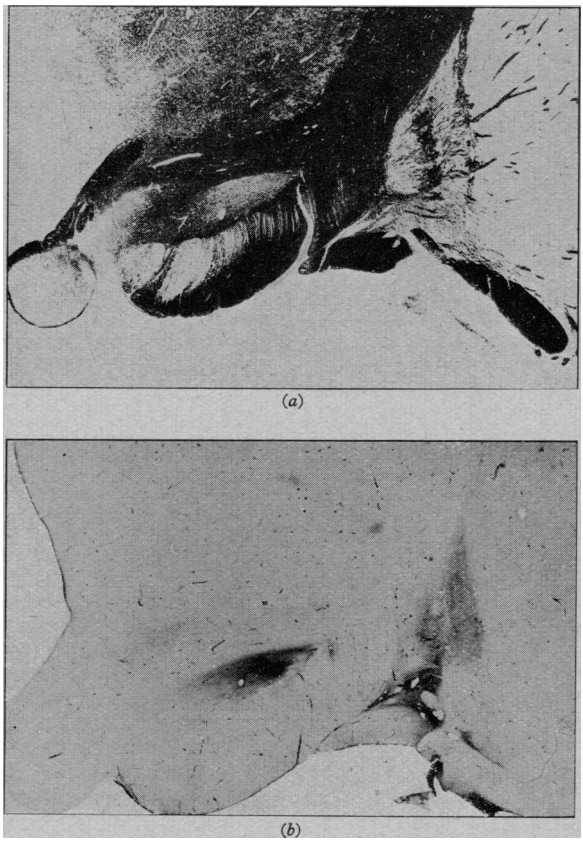

FIG. 7.—Late sequelæ of kernicterus. Note in (*a*) the loss of myelinated fibres in the subthalamic nucleus and globus pallidus (Kultschitsky-Pal × 3) and in (*b*) the corresponding increase in neuroglial fibres in the affected nuclei (Holzer × 3).

REFERENCES

BIELSCHOWSKY, M. (1915) *J. Psychol. Neurol., Lpz.*, **22**, 1.
—— (1923) *J. Psychol. Neurol., Lpz.*, **30**, 29.
BROUWER, B. (1949) *Proc. R. Soc. Med.*, **42**, 603.
COURVILLE, C. B. (1950) *Bull. Los. Angeles neurol. Soc.*, **15**, 155.
CROME, L. (1952*a*) *J. Path. Bact.*, **64**, 479.
—— (1952*b*) *Arch. Dis. Childh.*, **27**, 468.
GREENFIELD, J. G., and WOLFSOHN, J. M. (1935) *Arch. Neurol. Psychiat., Chicago*, **33**, 1296.
HALLERVORDEN, J. (1939) *Z. ges. Neurol. Psychiat.*, **167**, 527.
HOLLAND, E. (1920) *Trans. Edinb. obstet. Soc.*, **40**, 112.
—— (1922) *J. Obstet. Gynaec., Brit. Emp.*, **29**, 549.
LITTLE, W. J. (1862) *Trans. obstet. Soc. Lond.*, **3**, 293.
MITCHELL, R. G. (1952) *Arch. Dis. Childh.*, **27**, 95.
OSLER, W. (1889) The Cerebral Palsies of Children. London.
RYDBERG, E. (1932) *Acta path. microbiol. scand., Suppl.* 10.
SCHLESINGER, B., and WELCH, R. G. (1952) *Gr. Ormond St. J.*, No. 3, 14.
SCHWARTZ, P. (1924) *Z. ges. Neurol. Psychiat.*, **90**, 263.
—— (1927) *Ergebn. inn. Med. Kinderkeilk.*, **31**, 165.
VOGT, C., and VOGT, O. (1951) *Neurology (Minneapolis)*, **1**, 218.
WOHLWILL, F. (1936) Handbuch der Neurologie. Berlin, **16**, 35. Ed. Bumke, O., and Foerster, O.

Dr. P. H. Sandifer:

Incidence of Infantile Cerebral Palsy

Although it was in this country, as long ago as 1843, that spastic and athetoid infantile palsies were first described by W. J. Little (*see* Little, 1853 and 1862), yet it was in the U.S.A., in recent times, that there began a renaissance of interest in the treatment of these disorders, largely due to the work of Phelps (1940) and Carlson (1941).

In the United States Phelps' figures are generally quoted (1946). He estimates that in every 100,000 of population there will be produced, each year, 7 cases of I.C.P. whose destiny is as follows:

TABLE I

1 case	14%	Death before aged 6 (usually in infancy)
2 cases	28%	Feeble-minded Permanent custodial care needed
1 case	14%	Severe physical handicap Custodial care and education needed
2 cases	28%	Moderate physical handicap Education and treatment needed
1 case	14%	Mild physical handicap No special problem

The chief problem, according to Phelps, is the 28% moderately handicapped cases who number, as 20-year-olds or under, 400 per million of population.

In 1948 the British Council for the Welfare of Spastics (B.C.W.S.) conducted a survey in which Local Authorities in England and Wales were questioned, two-thirds replying.

The over-all average incidence rate was 0·92 per thousand child population under 16. The average incidence rates derived from each individual Authority gave 1·12 per thousand.

A further series of surveys in selected areas, conducted in this country by the Ministry of Education in 1947–48, showed wide variation in the incidence of cerebral palsy in children of school age. The highest incidence was 2·5 and the lowest 0·3 per thousand in the county boroughs providing the statistics. (In contrast, American figures show a remarkable uniformity of incidence.) From the figures provided by their survey the following tentative conclusions can be drawn:

(i) On the average the incidence of I.C.P. is a little over 1·0 per thousand children of school age.

(ii) The total number of children under 16 with I.C.P. in England and Wales is approximately 6,000.

It would be interesting to know what proportion of these 6,000 children fall into the several syndromes of infantile cerebral palsy. But figures published vary enormously due, it would seem, to varying criteria of diagnosis, particularly in distinguishing spastic from athetoid forms. Those who believe (Phelps, 1941*a*, *b*; Evans, E. S., 1946; Evans, P. R., 1948) that the athetoid are about as common as the spastic forms, stress that "tension athetosis" is frequently mistaken for spasticity of pyramidal type.

It is also important to know what proportion of these 6,000 or so children are capable of some kind of suitable training. In October 1952 a memorandum on the training of teachers of cerebral palsy children was submitted to the Ministry of Education's National Advisory Council (on the training and supply of teachers by the B.C.W.S.). This memorandum made the following estimate:

TABLE II

Capable of attending ordinary schools	25% approx.
Probably capable of benefiting from special schools	60% approx.
Incapable of benefiting from any schools because of serious mental defect (at best only suitable for training in Occupational Centres)	15% approx.

The "New Treatment" of I.C.P.

These figures give a rough idea of the size of the problem and they prompt the question whether it is a good investment to deal with it by spending large amounts of money and man-hours of skilled labour in organizing centres for the treatment of these cases.

Before venturing an answer it would seem wise to examine this "New Treatment" of infantile cerebral palsy. To me it seems to be no more than the application of the principles which underlie skill-learning in normal subjects, but with particular patience and understanding. For this reason it would perhaps be worth while to consider the process of acquiring skills.

James Collier, in his Savill Memorial Oration of 1929, speaking of localization of function in the nervous system, had this to say: ". . . we must keep carefully in mind that the function of movement and the function of sensibility are not two separate functions, but that they are so intricately blended as to be inseparable. We have no consciousness of movement in ourselves except from the sensations which result in its accomplishment. We learn every skilled act by gradually laying down, by repetition,

the pattern of the sensations which are produced by its correct performance. We reproduce them from the memory of these sensory patterns. We become clumsy with our skilled acts when out of practice because the memory of the finer details of the sensory pattern tends to fade and we reinforce them again by further repetition."

These remarks of Collier seem to me apposite to the clinical problem presented by I.C.P. For here there is more than just a motor deficit. Thus the inability to perform intended motor actions prevents the accumulation of a store of these memories of sensory patterns of movements and postures upon which voluntary movement depends. Lack of practice means lack of skill—and the untreated I.C.P. is grossly handicapped by lack of opportunity for practice.

Collier continued with these words: "There is in the function of movement a running analysis from the proprioceptive system and sometimes, in addition, from the visual and auditory systems, which is absolutely essential to this function."

The importance of this "running analysis" on motor function he illustrated by the experiments of Osborne and Kilvington on the dog. These workers connected the proximal end of the middle cord of the brachial plexus on one side to the distal end of the middle cord of the brachial plexus on the other side. The dog recovered well and could walk, run and hold down a bone with either fore-paw. Examination after recovery showed that the extensor afferents and efferents of one fore-limb had been thrown into the middle cord of the opposite brachial plexus and therefore into the hemisphere of the same side. Electrical stimulation showed that the cortical points for extension of the right fore-limb had been shifted from the left hemisphere to the right.

Experiments of this kind, and also clinical observation, as Walshe (1948) has argued, are not compatible with the view that movements are represented in fixed points in the cortex. Further knowledge of cerebral localization has followed the study of hemispherectomized subjects. This operation, first performed by Dandy and more recently by Krynauw (1950) is now almost a commonplace operation amongst neurosurgeons who seek to relieve the fits and behaviour disorders of one type of I.C.P.—infantile hemiplegia. We have learned how surprisingly well these hemispherectomized subjects can use the contralateral limbs—sometimes with less spasticity than before operation. Goody and McKissock (1951) have sought to explain this amazing fact and have suggested that, at birth, the infant has no absolute localization of function in its cerebral cortex. As they themselves put it, "the brain stands ready for training". If one hemisphere is damaged, other parts of the brain to some extent take over the function of the parts that cannot develop. Whether these functions are taken over by lower centres of the same side, cortical or subcortical centres of the opposite side or by a combination of both, it is not known. But it would seem that, in young children, this "plasticity" of the brain in re-allocating functional sites may enable the infantile diplegic and quadriplegic to do what the infantile hemiplegic does—namely to achieve some degree of motor skill by enlarging the scope of surviving brain tissue—provided there are favourable circumstances. The essence of the "New Treatment" is to provide these favourable circumstances. As Collier pointed out regarding the dog experiment of Osborne and Kilvington, functional recovery of the afferents is of enormous importance and must precede the regaining of efficient motor function which depends upon proprioceptive guidance. So, too, with learning skills by the I.C.P. patient (as in normal subjects) it is what goes in via the afferents which largely determines what comes out via the efferents. The favourable circumstance which treatment should aim at is the provision of an abundance of co-ordinated afferent stimuli, particularly from the paralysed parts, so that the child can acquire a repertoire of movement and posture memories and, through repeated practice, skill in translating them from thought into action. By way of example, in teaching the child to feed itself or to write, the hand is held, grasping the spoon or pencil, and the arm moved passively so that food is transferred from the plate to the mouth or symbols made to appear on the paper. These passive feeding and writing movements are made under guidance and control and, by repetition, a store of memories of these movement patterns is built up—memories of movement which must precede the performance of voluntary movement. It is likely, too, that the size and functional efficiency of the nerve cells concerned in the motor acts imperfectly performed in infantile cerebral palsy are favourably influenced by repeated activity.

In the treatment of infantile cerebral palsy, as in the treatment of poliomyelitis, there is the primary benefit which is the gaining of motor skills: there is also the secondary benefit of avoiding those evil complications of the disease in the form of deformity and contracture. C. D. Agassiz (personal communication, 1952) maintains that deformity and contracture are preventable in children of normal mentality taken early enough. If this challenging contention proves true, then the tenotome, the instrument which Little introduced into this country, is unnecessary. It would follow that, if more effort were expended in preventing these complications, there would be less need to try and cure them by corrective operations, with all the waste of ward accommodation and skilled man-hours which avoidable operations involve.

But such benefits, when physical disability is severe, are probably best achieved in special units; and about these we have asked the question, "are they a good investment?" I think the answer depends on the selective machinery of such units. If only those patients are admitted in whom a successful response to treatment in a reasonable period is predicted with fair accuracy, then they would seem

to be worth while. Cases which should not be (and are not) admitted are those amenable to indirect treatment—the 25% who will reach ordinary schools and who can be adequately treated in out-patient clinics by instructing the mother in what she should do. Likewise those should be rejected who are too defective mentally to benefit.

These Special Centres have shown us how surprisingly well some of their cases do. But nobody has compared, as far as I know, a treated with an untreated group. It would seem important, however, to avoid those dangers which followed the introduction of the "New Treatment" of poliomyelitis—extravagant claims for particular techniques and quaint physiological dogmas.

Adult Cases of I.C.P.

But the good results of good treatment are largely wasted if ways and means are lacking in finding these people work in later life. There are no figures available concerning the employment of I.C.P.s. The M.R.C. Memorandum No. 28 on "The Employment Problem of Disabled Youth in Glasgow" (H.M.S.O., 1952) states "The severely paralysed, no matter how intelligent or capable they may be, do not stand much chance of finding a job in Glasgow. As they themselves say: 'People take one look at you and that's the end of any chance of a job.'" The experience of B.C.W.S. suggests that this is true of the whole country.

It would be useful to know what proportion of I.C.P.s surviving infancy survive into adult life where they raise this problem of employment, also the problem of custodial care. No figures are available. The B.C.W.S. has information concerning a little over 2,000 cases. The vast majority of these are young children. The Midland Spastic Association, which is trying to register all I.C.P.s in their area, likewise find that much the greater proportion are children.

This lack of information about adult cerebral palsies has led to speculations concerning the early death of these subjects.

On going through cases of I.C.P. who came to the Royal National Orthopædic Hospital over a two-year period, 182 were identified. Of these about 79% were under 30 and 21% over 30 years old. One would expect a higher proportion of children than adults to attend a hospital for treatment and the age distribution does not show any surprising preponderance in favour of the children. The figures were as follows:

TABLE III

Ages ..	1–9	10–19	20–29	30–39	40–49	50 and over
Cases	49	62	32	23	9	7
% ..	26·9	34·1	17·6	12·6	4·9	3·8

	Cases	%
Over 30	39	21 (approx.)
Under 30	143	79 (approx.)

Some Clinical Aspects of I.C.P.

My experience of this condition in infants and very young children is very limited, but at the other end of the scale I have seen the diagnosis of infantile cerebral palsy delayed till the autumn of life. These cases present as mild spastic paraparesis of late onset with pyramidal but no sensory signs. Careful enquiry reveals a life-long minor disability which has been accepted for so long that it is scarcely recognized (as may be the case with victims of mild peroneal muscular atrophy). The past history may contain information which reveals that disability is nothing new: for example, that "irons" needed to be worn in childhood; that there was delay in learning to walk; that although football was played at school, running was so clumsy that he always played in goal. With advancing years we become slower and less nimble on the legs and tire more readily. If there is any underlying motor handicap, this process will be accentuated and disability previously accepted because familiar, will now be recognized so that medical advice is sought.

In children too, one sees, from time to time, the complications of spastic diplegia—deformity—bring them before the doctor, the underlying cerebral lesion being missed. Thus a child, reported to have played games at school, may start to walk on the toes and to show an ungainly gait. Examination reveals extensor plantar responses and contracture of the Achilles tendons, in the absence of sensory or sphincter disturbance and with brisk abdominal reflexes. Again careful enquiry may provide the clue, with hints that minor motor disability has long preceded the onset of deformity.

Sometimes, in young people with spastic diplegia, the diagnosis comes up for review, because the disability is progressive. Thus a child may gain fair but delayed skill in walking, only to lose this skill later. The deterioration, in such cases, is not due to any progressive disorder in the nervous system but to a complication of that stationary disorder in the form of contracture. As the tendon shortens so walking deteriorates and disability increases.

It would seem important to use the Special Units for Infantile Cerebral Palsy for collecting information. I think long-term comparisons should be made between comparable groups treated in different ways—for example, those treated in Special Centres with those receiving no special treatment.

It is also important to test the contention of Agassiz—that deformities are preventable if children of normal intelligence start treatment early enough. If the future proves him right, we shall have gained a valuable fragment of knowledge for use in treating infantile cerebral palsy.

Prevention.—The infant brain may, we know, be damaged in various ways in different sites in the pre-, para- and post-natal periods, producing cerebral palsy of symmetrical or asymmetrical type by lesions most frequently involving the cortico-spinal tracts, the corpus striatum or both. We need to make further progress before we can do much more than think in terms of preventing cerebral palsy.

The incidence of premature births, difficult labour and neonatal asphyxia is higher for cerebral palsied than for normal infants. There is a relationship, though not always a direct one, between abnormal birth and abnormal brain. Nevertheless mechanical trauma and asphyxia sometimes do cause infantile cerebral palsy and to some extent these causes are preventable.

That there is a relationship between maternal ill-health and fœtal deformity is suggested by experimental findings in pregnant animals subjected to such insults as vitamin lack (Warkany and Schraffenberger, 1944; Warkany, 1947; Wilson and Warkany, 1950), dye injections (Gillman *et al.*, 1948), radiation (Kaven, 1938) and anoxia (Ingalls, 1950). Even with anencephalus, in which evidence has accumulated indicating a genetic origin (Schade, 1939; Böök and Rayner, 1950), it has been shown by McKeown and Record (1951) that there is a curious seasonal incidence of births with this abnormality. Why it should be that there is a higher incidence of anencephalic malformations amongst embryos conceived in March or thereabouts, is not known. But it does suggest the operation of an environmental factor in ætiology and thus provides hope that further knowledge may give some measure of control over this and other types of congenital cerebral abnormality.

REFERENCES

Böök, J. A., and Rayner, S. (1950) *Amer. J. hum. Genet.*, **2**, 61.

Carlson, E. R. (1941) Born that Way. New York.

Collier, J. S. (1930) *Brit. med. J.*, i, 55.

Evans, E. S. (1946) *Proc. R. Soc. Med.*, **39**, 317.

Evans, P. R. (1948) *Proc. R. Soc. Med.*, **41**, 402.

Gillman, J., Gilbert, C., Gillman, T., and Spence, I. (1948) *S. Afr. J. med. Sci.*, **13**, 47.

Goody, W., and McKissock, W. (1951) *Lancet*, i, 481.

Ingalls, T. H. (1950) *New Engl. J. Med.*, **243**, 67.

Kaven, A. (1938) *Z. KonstLehre*, **22**, 247.

Krynauw, R. A. (1950) *J. Neurol. Psychiat.*, **13**, 243.

Little, W. J. (1853) On the Nature and Treatment of Disorders of the Human Frame. London.

—— (1862) *Trans. obstet. Soc. Lond.*, **3**, 293.

McKeown, T., and Record, R. G. (1951) *Lancet*, i, 192.

Phelps, W. M. (1940) *J. Bone Jt. Surg.*, **22**, 1004.

—— (1941*a*) *J. Amer. med. Ass.*, **117**, 1621.

—— (1941*b*) *N.Y. St. J. Med.*, **41**, 1827.

—— (1946) *Sth. med. J., Nashville*, **39**, 132.

Schade, H. (1939) *Erbarzt*, **7**, 116.

Walshe, F. M. R. (1948) Critical Studies in Neurology. Edinburgh.

Warkany, J. (1947) Advances in Pediatrics. Editor: A. G. de Sanctis. New York; **2**, 1.

——, and Schraffenberger, E. (1944) *J. Nutr.*, **27**, 477.

Wilson, J. G., and Warkany, J. (1950) *Pediatrics, Springfield*, **5**, 708.

Mr. E. Stanley Evans: There are now in this country a number of cerebral palsy centres, including the pioneer centre at Carshalton, and others in Croydon, Birmingham, Edinburgh and Ivybridge. These are all special centres dealing in varying ways with the diagnosis and assessment of those suffering from cerebral palsy, and with their treatment, training and education.

The care and treatment of the spastic child is not the prerogative of the neurologist, pædiatrician, psychiatrist, the expert in physical medicine, or the orthopædic surgeon, but demands team work and participation by all these and others such as the aurist (to exclude pitch deafness), so that the child can be treated comprehensively by the physiotherapist, occupational and speech therapists, and by the expert in vocational guidance and training. In no condition is it more necessary for each specialist and therapist to play his part, not by an individual or isolated approach, but as a member of a well co-ordinated team.

The first essential is the accurate diagnosis and assessment of the physical condition of the child, as to the nature and extent of the condition, whether spastic, athetoid or ataxic, and whether mono-, hemi- or quadriplegic. In particular, the differentiation between the child suffering from spastic quadriplegia, whose muscles exhibit the spastic stretch reflex, and athetoid quadriplegia with tension, may not be easy until after a period of observation. Secondly a mental assessment must be made, so that the child who is intelligent and educable may be selected for comprehensive treatment. This may be extremely difficult even for the expert educational psychologist, and is concerned with potential mental ability and not with the ability to carry out certain performance tests demanding neuromuscular co-ordination, and the ability to communicate with others, for example by means

of speech. The degree of mental ability does not necessarily bear any relationship to the extent of the physical handicap. There are those with gross physical disability associated with widespread involvement of the brain and mental impairment for whom practically nothing can be done, others with slight physical disability and marked mental impairment, and yet others with gross quadriplegia and normal intelligence. With our present limited resources it is essential to select for treatment those who are educable and not too severely handicapped physically.

The aims of treatment are:

(1) To train the child in the elementary skills of daily living, including dressing, feeding, personal hygiene and walking, so that he may become socially sufficient and presentable.

(2) To establish and improve the various means of communication by speech, writing and typing.

(3) To educate the child and train the individual vocationally to the highest level of achievement.

These aims are more difficult to attain in the older child, but when carefully selected even these may respond in a remarkable manner if stress is laid on education, training and welfare.

Treatment is essentially conservative in character, for even when operative procedures are necessary they are but incidents in a comprehensive and prolonged programme of treatment. Physiotherapy has an important role to play and is concerned with function rather than anatomical improvement, with the conservation and development of muscle power rather than with weakening of apparently over-active muscles, and it must be physiological and dynamic rather than corrective and static. Relaxation and the elimination of purposeless movements must govern the treatment of the athetoid, whilst muscle training and the establishment of increased joint mobility is necessary for the spastic child. Exercises in the prone position, crawling and the development of reciprocal movements, are practised in some centres. Relaxation is first secured in the fully recumbent position, assisted by deep breathing exercises, and then with the aid of Guthrie Smith slings, under-water exercises, and a relaxation chair. Treatment and the training of skills proceed by easy progressive stages. The skill of feeding is acquired by passive feeding in the relaxed recumbent position, and then progressively by using a special feeding table and utensils until ultimately the child can eat and drink more or less normally. The skill of walking is acquired by achieving sitting and standing balance, the use of special skis and surgical appliances, until the child is able to stand and walk alone. Improvement in the function of the hands is secured by developing the sense of touch, by handling objects in sand, by handling large wooden bricks, and then smaller ones, by open finger and brush painting, and then by writing or the use of the typewriter in order to provide an additional means of communication.

Control of the trunk and shoulder and pelvic girdle muscles must be obtained in the spastic child, and the muscle power of the weaker antagonists of spastic muscles must be developed. This is not easy to secure because contraction of the antagonist is inhibited by the stretch reflex of spastic muscle. This difficulty is found when an attempt is made to develop the gluteus medius in the presence of spastic adductors of the hip, and provides one of the indications for operation.

Operations are of value in releasing contracted structures, thereby correcting deformities; in paralysing temporarily overactive spastic muscle groups, and in enhancing function by stabilization of certain joints. Surgery must be as conservative as possible provided the desired correction is obtained, in order that the sum total of muscle power in the limb is not seriously diminished.

If the affected foot remains in a position of fixed plantar flexion when the knee is flexed to 90 degrees, the structural contracture is probably in the soleus and posterior capsule of the ankle-joint. Posterior capsulotomy and elongation of the tendo achillis should be performed, the amount of elongation being determined beforehand. When the foot can be dorsiflexed to the mid-position only when the knee is flexed to 90 degrees, and there is a fixed flexion deformity of the knee of 30 degrees or more, posterior capsulotomy of the knee combined with distal stripping of the gastrocnemius origin is necessary. If this is insufficient, tenotomy of the semitendinosus, of the tendinous portion of semimembranosus, and of the posterior fibres of the ilio-tibial band will usually suffice. The knee should be moderately corrected in plaster, or on a Thomas's knee splint, and gradually straightened during the ten or fourteen days following operation.

Soutter's muscle slide operation and subcutaneous adductor tenotomy are indicated when marked flexion adduction contracture of the hip is present. Stoffel's orthodox operation of selective nerve section with a view to producing permanent paralysis of spastic muscles is generally contra-indicated, because the total muscle power in a limb is thereby reduced considerably. On the other hand temporary paralysis produced by crushing the nerve supply to spastic muscles may be invaluable, as it allows the antagonists to be trained and developed without evoking the stretch reflex. The abductors of the hip, for example, may be strengthened after the nerve supply to the bulk of the spastic adductors has been crushed, so that when the adductors recover the gluteus medius may be well developed, and muscle balance achieved without permanent diminution of muscle power. The function of the hemiplegic hand is often improved following an arthrodesis of the wrist, provided a suitable position is selected. A temporary plaster splint may serve as a guide to the optimum position which must combine the ability to relax the fingers and grip in the flexed position, with the increased gripping power in full dorsiflexion. Generally the mid-position should be selected with the second metacarpal in line with the axis of the radius.

The important factors in the management of those suffering from the infantile cerebral palsies are:

(1) Accurate physical and mental assessment, and the selection of patients likely to respond to treatment.

(2) Comprehensive conservative treatment at the earliest age possible, and ancillary operative procedures when essential.

(3) Formal education for the child of school age, as treatment and education are complementary, and improvement is often reciprocal.

(4) Provision of more special centres for cerebral palsy, and the active co-operation of well-informed parents. These centres are expensive to establish and to maintain, and it is reasonable to consider whether the cost is justified in view of the results obtained. Provided reasonable improvement is acceptable, the value of these centres cannot be assessed on financial grounds only.

Dr. J. P. M. Tizard:

The Future of Infantile Hemiplegics

A survey of the long-term effects of cerebral palsy was begun at the Children's Medical Centre, Boston, in 1951, and I joined Dr. Bronson Crothers, previously Head of the Neurological Department in the Children's Hospital, and Dr. Edith Meyer, the psychologist, in an attempt to discover the fate of patients with infantile hemiplegia, who had received investigation as in-patients within the previous twenty years. Here, in outline, are some of the findings of this survey.

There were records of 450 cases of infantile hemiplegia and from these we selected cases of children who had been normal at birth and who had developed normally until overcome by some disaster which was unmistakably associated with the development of hemiplegia. About 150 conformed to this requirement, that is about a third of the total, the remaining two-thirds having histories suggesting that the hemiplegia was determined at birth or possibly, in some cases, before birth.

We traced 95 cases and of these examined 51. 43 were male and 52 female. 57 had a right and 38 a left hemiplegia, but cases in which the onset was sudden were equally divided between right and left.

TABLE I.—AGES OF THE 51 PATIENTS WHEN LAST SEEN

Age	Under 4 years	4–7	8–11	12–15	16–19	20–23	24–27	28+
No. of patients	4	12	12	7	5	6	2	3

There are difficulties in selecting cases in which hemiplegia has developed after birth. Hemiplegia present at birth does not usually become obvious until the infant is several months old. As can be seen from Table II we were not wholly successful in excluding the birth palsies.

TABLE II.—MODE OF ONSET OF HEMIPLEGIA

A.	Sudden onset with convulsion	39	
	(1) Ill before convulsion	28*	(Measles 4, roseola infantum 3, triple toxoid immunization 2, "colds" 4, pertussis 1, epidemic encephalitis 1, fever ? cause 6, others 7)
	(2) Well until convulsion	11†	(1 facial nævus, probably Sturge-Weber syndrome)
B.	Sudden onset without known convulsion ..	8	
	(1) Ill before hemiplegia	7	(Pertussis 1, scarlatina 1, chickenpox 1, influenza 1, fever ? cause 3)
	(2) Well until hemiplegia	1	
C.	Meningitis	7	(Pneumococcal 2, meningococcal 1, hæmophilus influenzal 1, streptococcal 1, organism unknown 2)
D.	Head injury	7	
E.	Probably dating from birth	8	
F.	Miscellaneous	26	(Mostly of gradual onset)

*2 found to have subdural hæmatomata.

†2 found to have subarachnoid hæmorrhage.

The first group consists of children who suddenly developed convulsions, and, on recovering consciousness, were found to be paralysed on one side. The majority of these were suffering from some febrile illness before the convulsion, but 11 were well and the fit came out of a clear sky. One of these had a probable Sturge-Weber syndrome, but the remaining 10 were normal children. The mode of onset of hemiplegia was so sudden in these cases as to suggest a vascular accident. There is a good deal of indirect evidence to suggest that in many of these cases there has been an occlusion of the middle cerebral artery. However, I have heard some direct evidence from three American neurosurgeons who performed carotid arteriograms in 4 cases of infantile hemiplegia shortly after

the event, showing in each case blocking of the main stem or of branches of the middle cerebral artery in the affected hemisphere.

Some of our cases had been labelled "encephalitis" but in the great majority there was little evidence to support this diagnosis. Measles encephalitis is very rarely complicated by permanent neurological sequelæ, but I suppose that the two conditions might occur coincidentally. When the C.S.F. had been examined within the first few days following the hemiplegia it was usually normal, although in a few instances it was found to contain up to 20 or 30 white cells per c.mm. However, after an interval of a week or more pleocytosis was a more common finding, and when lumbar puncture had first been carried out after such an interval, the finding of cells had sometimes led to the probably false diagnosis of encephalitis.

In the last group (F) hemiplegia had become manifest at a considerable period following that at which birth palsy is usually detected, but we could not be certain in many that there had not been some previous brain damage, possibly dating from birth. For instance, some of these children had had recurrent fits prior to the onset of the hemiplegia.

These cases were not intentionally selected in any other way. But there must probably always be some prior and unwanted selection to prevent one's claiming a series such as this as really representative. The mildest cases of hemiplegia might not find their way to the Neurological Department, nor perhaps to a hospital at all. This point is illustrated by a film of a 3-year-old boy who has a left hemiplegia dating from birth. Although the hemiplegia is quite obvious on clinical examination, his gait is not remarkable, except that the left arm swings a little less than the right when he runs. If one listens to him running one can detect the uneven sound of his footsteps.

The importance of detecting these not very obvious cases of hemiplegia is not because the motor defect is likely to prove a very serious matter, but because of its significance in relation to selective mental defect and fits.

Fig. 1 shows the age of onset in all 95 cases excepting the 8 with known birth palsy.

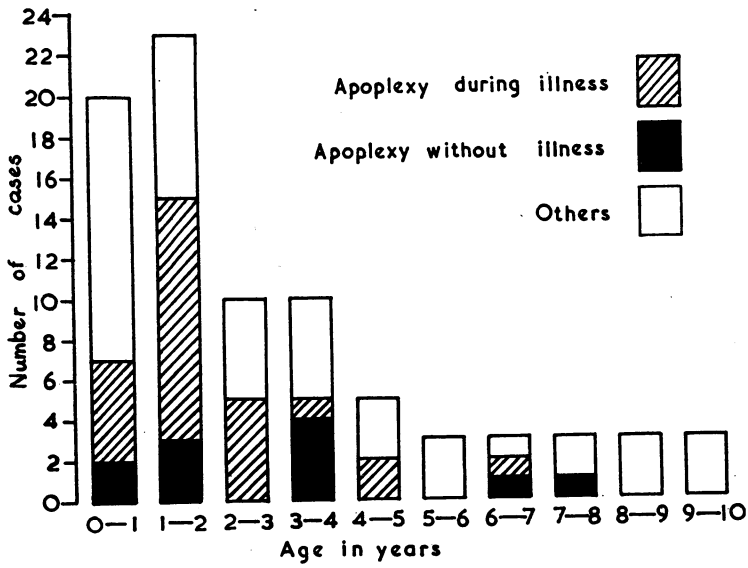

FIG. 1.—Age of onset of hemiplegia.

51 patients were seen in the Neurological Department when an interval history was obtained, particular attention being paid to motor and speech development before and after the hemiplegia; the extent of the handicap imposed by the motor defect; the effect of physiotherapy and especially of so-called "muscle training"; the time of onset and frequency and the nature of fits; behaviour and emotional problems in relation to the family situation and finally the school record and adult occupation. The patients were given physical and psychological examinations and in most cases an EEG was performed.

In few instances did the motor disorder of the affected leg impose serious handicaps. All could walk, most could run. In the great majority of cases, however, the hand on the affected side was useless for performing any fine action and, in general, these patients went through life as if they only had one hand.

Short accounts of the sensory findings have been given by Tizard and Crothers in 1952, and by Tizard in 1953.

Speech was never seriously affected except in those with gross mental defect. Even in children developing right hemiplegia after the age of 5 years aphasia lasted but a few weeks and normal speech had returned in each case by three months.

Table III gives data relating to epilepsy.

TABLE III.—EPILEPSY

Fits at onset, 32		No fits at onset, 19	
Recurrent fits from onset of hemiplegia	18	No fits subsequently	9
Fits after an interval	7	Fits after an interval	10
No fits except at onset	7		

Duration of fit-free period:

Under 1 year	1<3	3<5	5<7	7<10
3	6	4	0	4

Time interval following hemiplegia	1 year	3 years	5 years	10 years	15 years
Proportion of patients with recurrent fits	35%	49%	50%	66%	54%

In 32 of the 51 patients the onset of hemiplegia was accompanied by fits. Very often the first fit was generalized, and a few hours later a second fit with clearly lateralizing features occurred and, on recovery, the child was found to be paralysed on that side. In most cases fits continued; in some there was a latent period of freedom followed by fits, while in others no further fits had occurred up to the time of examination, nineteen years later in 1 case. (Some of this latter group may yet develop epilepsy; in 6 of the 16 less than four years had elapsed between the onset of hemiplegia and the time of examination.) Those who had no known fit at the onset of the hemiplegia showed the same liability to develop epilepsy later. 3 patients, who developed epilepsy after such periods of freedom, had frequent attacks for six, eight and eight years respectively, when the fits ceased completely without any change in medication. The fits were, incidentally, very resistant to medication.

In the majority of cases seizures were characterized by clonic movements of face, arm and leg on the affected side, with or without spread to the normal side and loss of consciousness. But every variety of epilepsy was encountered from momentary akinetic seizures to generalized *grand mal* attacks; the former being commoner in the younger and the latter in the older patients. The type of epilepsy not infrequently changed over a period of years. For instance, one patient had 4 generalized convulsions during measles complicated by bronchopneumonia at the age of 15 months and was left with a left hemiplegia. Nine years later she began to have attacks characterized by a feeling of numbness on the left side and jerking of the left arm and leg. These attacks occurred two or three times a week for nine years. She was then completely free from fits for a further nine years, but in the past five years has had occasional attacks of tingling of the lips and dysphagia.

That anti-social behaviour, fits of temper and violence are common in patients with infantile hemiplegia is well known and was emphasized by Strümpell (1884) in his original paper. There was a relative infrequency of such behaviour in the histories of the patients we saw. There seemed to be a rather close correlation between behaviour disorders and the frequent occurrence of fits. Not only were temper tantrums more common in children suffering from epilepsy than those free from fits, but also parents of individual children reported that meanness and tempers occurred much more often during a period of frequent seizures, while, when the child was free from attacks, his nature might appear to undergo a complete change. In thinking of these emotional storms as being connected in some way with cerebral dysrhythmia we should not overlook the great importance of environment, especially the handling of the children by parents and teachers, in the causation of behaviour disorders. Over the years Dr. Crothers and his associates acquired a profound understanding of the problems of bringing up children with mental or physical handicaps and of the emotional difficulties engendered by mismanagement. I think the wise advice given to parents was in no small measure responsible for the rather infrequent histories of behaviour disorders. To give an example, psychological tests showed again and again the characteristic intellectual pattern of brain-damaged children; good verbal abilities combined with defects in abstract reasoning and in those visuo-motor functions which apparently underlie the ability to understand symbols on which, of course, all school learning depends. Unless parents and teachers were aware of this selective mental defect, a child's superficial brightness combined with poor performance might lead to the unjust charge of idleness or inattention and therefore to rebellion on the part of a child. Another case of behaviour disorder may be found in the history of a pair of uniovular twins, one of whom had developed hemiplegia at the age of 2½ years. At 7 years of age they were quite amiable little girls but as they grew up both became disagreeable and subject to attacks of temper. The hemiplegic twin resented the fact that her sister could work in a factory and earn twice as much money as she was given for helping in the house. The normal twin, on the other hand, resented the extra care and attention given by the parents to her sister. The effect on the normal brothers and sisters of a mentally or physically handicapped child in the home would be an interesting subject for psychological study.

Dr. Edith Meyer and I tried to classify all the cases we had seen or heard of into three groups.

In Table IV the term "competitive" indicates that the patient, if a child, is able to compete socially and academically on more or less equal terms with children of his own age or, if an adult, is able to support and care for himself, albeit not necessarily at the level that might be expected from the nature of his social environment.

TABLE IV

	Competitive	Competitive with concessions	Non-competitive
Cases seen	11 (2)	11 (4)	29 (25)
Cases heard from ..	4 (3)		23 (15<22)
Dead 11			

(Figures in brackets indicate those still having seizures.)

The second group contains those patients who, as children, have had to be granted a reasonable amount of concessions by parents and teachers and as adults are able to contribute to their own needs, but who need some form of economic and social protection.

Many of the patients in the third, "non-competitive" group are already in institutions. The remainder, if children, are unable to take part in the social and academic activities of their age group, or, if adults, are entirely supported and cared for by relatives.

It will be seen from Table IV firstly, that competitiveness bears a close relation over the whole group to the incidence of epilepsy and secondly, that the prognosis for infantile hemiplegia as a whole is poor.

Sensory defects.—Sensory testing can be carried out successfully in quite young children. Mapping of the visual fields by confrontation methods and the examination of various cortical modalities of sensation, such as two-point discrimination, position sense, stereognosis, and so on, may be carried out with ease in an intelligent and co-operative child of 5 years.

We feel that our findings confirmed the observations of Penfield and Robertson (1943) on the connexion between retardation of growth and damage to the post-central cortex. All those with loss of discriminative sensation showed under-growth of the affected limbs, that is shortening as well as loss of muscle bulk. Patients with even severe motor defects who showed no shortening had no such sensory loss. I mention these sensory defects for a special purpose—to emphasize the futility of so-called "muscle training" to an arm which has no meaning to its owner. For instance, with loss of cortical sensory modalities, the patient cannot know the position of the arm in space and is presumably unable to acquire a memory of previous movements.

An intelligent young hemiplegic woman with under-growth and unilateral cortical sensory loss discussed frankly with us the medical management she had received in childhood and adolescence. With reference to that muscular training which was designed to make her in various respects independent, her most serious criticism of physiotherapy was the emphasis on training the affected arm which she had early appreciated was a waste of time. She complained of the lack of assistance she had received in trying to make the good arm doubly useful. In fact, the most useful muscle training she ever received was from a girl friend who taught her to do her hair with her one good hand.

In conclusion, I should say that I can find no clear evidence that the ultimate result is influenced by the nature of the original illness, the occurrence of fits at the onset or the side of the hemiplegia.

REFERENCES

PENFIELD, W., and ROBERTSON, J. S. M. (1943) *Arch. Neurol. Psychiat.*, **50**, 405.
STRÜMPELL, A. (1884) *Jb. Kinderheilk.*, **22**, 173.
TIZARD, J. P. M. (1953) *Arch. Dis. Childh.*, **28**, 243.
——, and CROTHERS, B. (1952) *Trans. Amer. neurol. Ass.*, **77**, 227.

Dr. N. S. Alcock: From my experience with the Dame Hannah Rogers School for Spastics at Ivybridge, I am struck with the number of unsolved problems in the clinical field: for instance, the effect of posture in controlling the involuntary movements of athetosis.

I am sure that these centres, although expensive, are justified provided they are regarded as research projects.

Dr. William Dunham: I should like to stress the fact that treatment must include constant supervision throughout the day; a succession of formal treatments alone cannot be expected to have more than transient effects on neuromuscular function. While the planning of such supervision demands expert knowledge of the difficulties in movement experienced by the individual child, its execution demands infinite patience springing from concern for his welfare. Usually, the most practical and economical solution is for the parents to work under supervision. For the intelligent child at school the co-operation of the teachers is also required. Residential accommodation providing the same essentials is necessary to deal with special difficulties of diagnosis or management.