THE SECONDARY FORMS OF MENTAL DEFICIENCY.

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Mental defect may properly be regarded as a disorder of the mind, and in approaching its study we find the making of a satisfactory classification just as difficult as in the wider domain of psychiatry. In the early part of last century mental defectives were classified largely on a physical basis, their anatomical defects or stigmata being used for the invention of racial types—the Kalmuck, Aztec, Papuan, and so on. At a somewhat later date a new classification was attempted on the basis of pathological anatomy, and this in its turn has been superseded by a scheme evolved from the standpoint of causation, which divides amentia into two classes: the primary forms, determined by neuropathic inheritance; and the secondary forms, determined by acquired factors operating before, at, or after birth. The former are said to account for 90 per cent. and the latter 10 per cent. of all cases, but in my experience too much emphasis has been placed on a history of mental defect in the parents or near ancestors, and I believe that future research will show that more than a quarter of all cases of mental defect are due to acquired factors.

In actual practice it is not always easy, or even possible, to determine whether mental defect is innate or acquired, and doubtless in the causation of some cases both groups...
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...forms of mental deficiency sustained at this period.

Thirdly, there is the post-natal period, in the course of which the brain of the growing child may be damaged by the toxins of acute infections, by the rupture of veins during convulsions, by direct trauma, and so on.

Returning to the pre-natal period, when the nourishment of the foetus is effected through the placental circulation, it is logical to assume that metabolic disturbances or disease in the mother may also injuriously affect the brain of the offspring. Certain

phase of rapid development, and the period of foetal life, which is principally concerned with growth. Doubtless, at any period of intra-uterine life the brain is liable to accidents which may impede or arrest its formative processes, but it is especially during the second period, when the foetus becomes nourished by maternal blood, that the common types of gross lesion occur. At birth there is further possibility of damage to the brain. Difficult and prolonged labour, instrumental delivery, and especially asphyxia neonatorum, are considered to be important agents in the causation of certain paralytic fevers, and notably small-pox, are known to occur in utero, and may possibly be the cause of arrest of brain development, but on this point we have as yet little exact knowledge, and for an undisputed and classical example of intra-uterine infection it is necessary to fall back on syphilis. Most observers are agreed that this disease has little influence as an ante-conceptional cause of mental deficiency, but an infection in early uterine life may unquestionably be responsible for arrested development of the brain and the production of feeble-mindedness, if it does not kill the foetus outright.
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To what extent must syphilis be held responsible for mental deficiency is still a matter of considerable controversy. Regarded by some as merely a concomitant of no greater significance than, say, an infection with the tubercle bacillus, by others the Spirocheta pallida is assigned a most important part in the genesis of amentia.

Sir Frederick Mott, in his evidence before the Royal Commission on the care and control of the feeble-minded, expressed the opinion that, "Syphilis is an active agent in the production of congenital weakness and the degeneracy that accompanies it." Other workers have found no reliable evidence to support such a view, and Goddard goes so far as to say, "One cannot help having the feeling that if syphilis is a cause of feebleness, it does so only under most favourable conditions." The evidence derived from the application of the Wassermann reaction is equally conflicting, and can, I think, only be explained by a difference in the quality of the material investigated. In my experience age is a factor of some importance, as a positive reaction may become negative when the child reaches adolescence, and consequently, in comparing statistics from different sources, misleading conclusions may be arrived at unless the ages of the patients investigated are known. The results may also be expected to vary according to the social status of the group examined, for in an institution filled with the children of slum dwellers, the incidence of congenital syphilis is likely to be higher than in a private home for defective children of the upper classes. Probably also the type of case investigated is of some importance, the incidence of syphilis being lower in a series of unselected cases than in a similar number of selected, on account of special physical disabilities—infantile palsies, and so on. A few years ago I investigated 800 patients in the Leavesden Mental Hospital, and obtained a positive Wassermann reaction in 19.75 per cent. The majority of the patients in this hospital are in the lower grades of mental deficiency, many of them being afflicted by physical infirmity, particularly nervous disease. It cannot, therefore, be claimed that the cases in this study...
were unselected, but the results approximate very closely to those of my colleague, Dr. Gordon, who in a recent investigation of 575 mentally defective found a positive Wassermann in 15.65 per cent.

There is, I think, no doubt that congenital syphilis has undergone considerable modification in type since the beginning of this century, possibly because the infection is of a milder type. The more usual clinical signs such as rhabades, Hutchinsonian teeth, and eye changes, form a clinical picture which is only seldom met with, and statistics based on the presence or absence of the classical signs will lead to a very erroneous idea of the incidence of the syphilis taint. In a series of 173 cases showing a positive Wassermann, I was only successful in finding two who presented the classical triad of bilateral eighth-nerve deafness, keratitis and notched incisors. Stigmata of congenital syphilis may indeed be entirely absent. On the other hand, in late congenital syphilis the Wassermann reaction may be negative, although the patient presents obtrusive signs of the disease. In looking for evidence of syphilitic infection, much information of diagnostic value can often be obtained from an examination of the larger joints. Thickening and broadening of the bony structures in their neighbourhood is very suggestive of congenital syphilis, particularly in the case of the elbow-joint, where there is often a limited range of movement, or an abnormality of the carrying angle to which Stoll has given the term "knock-knee elbow."

**The Pathological Anatomy of Syphilitic Amentia.**

The cerebral lesions of syphilitic amentia are not well known, and the following brief exposition may therefore be of some interest. A certain number of developmental anomalies, such as anencephaly, porencephaly and microcephaly, have been recorded, and in a few cases uncomplicated by paralysis I have been able to find imperfect development and numerical deficiency of the cortical nerve cells. Usually, however, when syphilis affects the nervous system of the foetus, it does so in an unmistakable manner, the pathological changes being of a gross character easily recognizable by the naked eye.

The lesion most often found is of the meningo-vascular type, and though the brain may show marked damage in one particular area, multiple diffuse affections are the rule. Pathologically, these resemble fairly closely the changes found in adult syphilis, but the alterations in the meninges and vessels have seldom the cellular character which distinguishes a progressive syphilitic process, and vascular occlusion leads to a sclerosis rather than a softening of cerebral convolutions. Depending on the stage at which it develops, the syphilitic lesion may be accompanied by secondary atrophy in those portions of the nervous system with which the affected areas are functionally related. The characteristic diffuseness of the syphilitic process is well illustrated by the following two cases.

Fig. 1 shows the appearance of the cerebral hemispheres of a syphilitic idiot,
hemiplegic from birth, who died in her twenty-first year. The left cerebral hemisphere is smaller than the right, and the pia-arachnoid covering both much thickened. Certain of the gyri above the left Sylvian fissure are shrunken and sclerotic (fig. 2), and on coronal section the left lateral ventricle is found to be dilated (fig. 3). Both the internal capsule and lenticular nucleus have been largely destroyed by an old softening in their neighbourhood. At the base there is a marked degree of leptomeningitis, the arteries are thickened and the cerebellum shows a crossed atrophy of its right hemisphere.

The second case (fig. 4) is that of a syphilitic idiot with right hemiplegia. The brain is small and shows a chronic leptomeningitis affecting its entire surface. All the convolutions behind the right precentral gyrus show the appearance known as microgyria (fig. 5), and on the left side the lower two-thirds of the motor cortex are atrophied and much firmer than other portions of the cortex (fig. 6). For the latter condition it is convenient to employ the term secondary microgyria. In both these patients the blood Wassermann was strongly positive, but the spinal fluids showed practically no departure from the normal. This seems to be quite the usual serological finding in syphilitic aments who survive to adolescence or adult life, and may, I think, be interpreted as indicating a non-progressive form of lesion. In contrast to the parenchymatous forms, the damage to the brain is sustained at an early period, the spirochaete dies or becomes latent, and the patient is left with permanent damage to the nervous system. Certain writers have expressed the view that the amentia of syphilis terminates in the development of juvenile general paralysis, but this is certainly not my experience, and I have seen only one patient in whom this possibility is at all likely. The patient is an epileptic idiot, aged 25, who, in addition to a positive Wassermann reaction in his blood and spinal fluid, gives a typical paretic curve with the colloidal gold test. It is now nearly
five years since the tests were performed, and as yet there are no other signs suggestive of general paralysis.

The mental enfeeblement which accompanies congenital syphilis may be of any degree, though as a rule those who exhibit paralysis are low-grade imbeciles or idiots. Owing to the multiplicity and diffuseness of the lesions the paralysis, when present, may assume many different forms. An investigation of 173 syphilitic aments at Leavesden revealed 2 with tetraplegia, 12 with diplegia, 17 with paraplegia, 18 with hemiplegia, and 4 with hydrocephalus. Syphilitic aments are frequently epileptic, and in the series quoted above 48 per cent. were subject to fits.

Treatment.—I have no experience in the treatment of very young syphilitic aments, but in the adolescent stage the results are disappointing, and I have never seen any mental improvement follow treatment by the various arsenical preparations commonly in use. Nevertheless, the recognition of this type of amentia is not without value, for it does occasionally permit one to arrest at an early stage lesions which, if untreated, lead to gross disfigurement. Unless the possibility of syphilis is borne in mind, syphilitic bone disease may be mistaken for that of tuberculosis, and indolent ulcers on the extremities may be wrongly ascribed to the influence of circulatory defects. Syphilitic aments, and especially those with paralysis, benefit very little from the exhibition of bromides or luminal, but in some, treatment with the arsenicals diminishes the number and severity of the fits in a gratifying manner.

Juvenile General Paralysis.

Although juvenile general paralysis may not develop until puberty, or even later, the invasion of the nervous system by the organism of syphilis probably takes place at an early age, and the disease may therefore be regarded as a form of secondary amentia. It is by no means so rare as was once thought, and in the last few years I have seen eleven cases. From the adult type it differs in that the duration is usually longer and remissions are practically unknown.

The usual history is of a gradual mental and physical deterioration, occurring at school age and in some cases initiated by convulsions. The child becomes increasingly stupid, indifferent to its surroundings, dirty in its habits, and eventually bed-ridden with contractured limbs, marked emaciation, and in fact all the classical features which distinguish the third stage of the adult type. Neurological signs characteristic of general paralysis are in-

![Fig. 7.](http://pmj.bmj.com/)
bral cortex; otherwise the morbid histology resembles that of the adult form.

The prognosis in juvenile general paralysis is bad. Treatment by induced malaria has little or no effect on the mental state of the patient, though it does appear to prolong life. One patient whom I treated five years ago is still alive, bereft of all intelligence and helpless in every way.

CRETINISM.

Cretinism or congenital myxoedema is caused by the loss of the secretion of the thyroid gland, and the mental deficiency which constitutes one of its chief symptoms is the direct consequence of that loss. In spite of the absence of the thyroid, typical symptoms are not usually present at birth, probably because the autocoids from the mother's thyroid are conveyed through the placenta, and after birth through her milk. Being improvable, cretins often come first under notice in general hospitals, and the number admitted to institutions for the mentally defective is very small. The physical signs present a certain uniformity of type and are not easily confused with those of any other disease. The body is stunted, podgy and heavy-looking. The head is large in proportion to the size of the body and is dolicocephalic. The subcutaneous tissue is infiltrated with solid oedema which imparts to the face its heavy expression. The cheeks are fat and baggy, the complexion earthy, the nose pug-shaped, the eyes set far apart, the tongue large and protruding, and the teeth carious. Elastic swellings may be present in the supraclavicular regions and axillae. The large pendant abdomen frequently shows an umbilical hernia. The hair is abundant, but coarse and dry. The limbs are short, broad, and often rickety. Many cretins are deaf and dumb, and in some the lower limbs are paralysed. These appearances are well seen in the accompanying illustration (fig. 8) of a male sporadic cretin, aged 25. The patient, of Polish origin, was found abandoned in London, and on admission measured 3 ft. 2 in. in height. He had a dental age of 12 years and weighed 4 st. 3 lb. He was a profound idiot, unable to articulate or to walk. He had a positive Wassermann in the blood and a number of discharging sinuses were present in the neck and right leg. After a year's treatment with thyroid extract his weight fell to 3 st. and he began to take some interest in his surroundings. His clinical history was brought to an abrupt termination by the sudden develop-
adid cretin varies from profound idiocy to slight mental enfeeblement. As a rule he is apathetic, placid and good-tempered, though if provoked he may show outbursts of violence. The amount of mental improvement depends very largely on the age at which treatment is commenced. Infant cretins may attain a normal mental level, but in those of a maturer age the mental development is never commensurate with the physical, some degree of feeble-mindedness being always the end result. Occasionally the alteration in the mental state is not altogether in the direction one would wish, for I have seen a sluggish, inoffensive cretin transformed by thyroid treatment into a vicious lout, who used his new-found strength for tyrannizing weaker patients and demolishing the ward furniture.

It is, of course, essential to keep up the treatment throughout the patient's life, and it is interesting to note that treated cretins seem to age very slowly, retaining the physiognomy of youth well into adult life. The patient whose photograph I show you (fig. 10) looks like a child of 10, though he is actually in his twenty-fifth year.
MACROCEPHALY.

The first variety to be considered in the group of large-headed or macrocephalic cases is the condition known as hydrocephalus.

The essential feature of internal hydrocephalus is an excess of fluid in the cerebral ventricles which become distended, sometimes to an enormous degree. The cause varies in different cases, but the great majority are a sequel of meningitis in the neighbourhood of the fourth ventricle, which blocks the foramina of Magendie and Lusckia so that cerebro-spinal fluid cannot escape to the points of absorption, the microscopic villi in relation to the large venous sinuses. Fig. 12 illustrates the appearance of the floor of the fourth ventricle in a case of obstructive hydrocephalus. Owing to the marked thickening of the ependyma, no details of structure can be made out on the floor which is traversed by two abnormally-placed vessels. The patient was an epileptic imbecile, who died of fits in his thirty-third year. His head measured 70 cm. in circumference, and in addition to somatic signs of congenital syphilis he had a positive Wassermann reaction in both blood and cerebro-spinal fluid.

More rarely the ventricular foramina are freely open, but the absorbing power of the arachnoid space is diminished, or the cerebro-spinal fluid is prevented from reaching the absorbing surface by adhesions at the base of the brain.

In a typical case the ventricles are widely dilated, the corpus callosum is reduced to a thin membranous sheet, and the cerebral hemispheres are converted into two large vesicles which collapse when the ventricles are punctured. The sulci are diminished in depth, the convolutions are broad and flattened, and the atrophy is sometimes so pronounced that the cortex and underlying white matter have a thickness of only a few millimetres. Clinically the appearance of the hydrocephalic is very striking. The cranium is much enlarged, bradycephalic and globular in form with the greatest circumference at the temples. The height of the forehead is exaggerated, the eyes widely set with sclerotics showing above the iris. Percussion of the skull is accompanied by an increased feeling of resistance, and the scalp is thin and tightly stretched. When the degree of hydrocephalus is marked, there is often interference with sight and hearing, and spastic paraplegia is not uncommon.

Hydrocephalic patients are usually placid, affectionate, and the least troublesome of all aments. In spite of the fact that the cerebral hemispheres may be extremely atrophied, the mental defect rarely exceeds that of imbecility, and many hydrocephalics respond well to training and can be taught to occupy themselves with simple handicrafts.

One patient under my care, whose head measures 27⅜ in. in circumference, has an exceptionally good memory for the ordinary events of hospital life and an extraordinary capacity for calculating dates.

If, for example, he is given any date, after a few moments' pause, in which he rocks his body to and fro, he is able to state the day of the week correctly. I have tested this capacity for calculating dates as far back as December 25, 1812, and have very rarely
found him make a mistake. His phenomenal aptitude in this direction is all the more remarkable, as he is totally unable to perform simple problems in arithmetic. He cannot, for example, multiply three by eight nor add together the number of days in two months.

**Diffuse Sclerosis.**

A second type of macrocephaly is that in which there is enlargement of the entire brain without distension of the ventricles. Diffuse sclerosis, as the condition is termed, is decidedly rare and not very easy to distinguish from hydrocephalus. The diagnosis rests on the observation that in hypertrophy the enlargement of the skull is most pronounced above the superciliary ridges, and the head is square rather than round. The pathological basis appears to be a diffuse neuroglial sclerosis which renders the consistence of the brain much firmer and heavier than normal. Patients with cerebral hypertrophy are usually of low mentality and are subject to epileptic fits, muscular weakness, and tremor.

**Tuberous Sclerosis.**

Although this disease is not associated with enlargement of the head, it belongs to the forms of sclerotic amentia and may conveniently be considered here. Clinically, tuberous sclerosis is characterized by the occurrence of epilepsy in conjunction with mental defect.

According to Freedman, the disease begins between the fourth and seventh months of foetal life and is neither familial nor hereditary.

In a typical case the brain is studded with sclerotic nodules, which on the cortex are slightly raised, umbilicated and yellowish-white in colour. Sub-ependymal tumours, rather like candle gutterings, are found in the ventricles, and multiple tumours are common in the heart, kidneys, skin and other organs.

The disease usually begins in early child-

hood with the development of epilepsy, and when once initiated the fits become gradually more and more severe and are often the direct cause of death.

Mental enfeeblement may be present from the first, or the patient may be normal for several years and capable of attending school for a few years, but sooner or later signs of deterioration are noticed.

The child ceases to take interest in its toys, loses the power of attention, and becomes increasingly dull and eventually faulty in habits. To this picture there may be added focal signs such as spastic paralysis, muscular spasms and speech defect. The clue to the recognition of this comparatively rare disease is often afforded in early adolescence by the appearance of adenoma sebaceum on the face. The eruption takes the form of small, firm, whitish or reddish nodules distributed over the cheeks and nose in a butterfly pattern. Between the nodules it is usual to find small teleangietases which give the complexion a mottled red. Fig. 13 is of a feeble-minded girl in whom the facial eruption is particularly well-marked. In other cases there is no eruption on the face, but rather larger nodules may be found on the trunk. When the skin is entirely unaffected diagnosis must be largely a matter of conjecture.
No treatment has any effect on the disease, which usually proves fatal in childhood or early adult life.

PARALYTIC FORMS.

Some degree of paralysis is one of the commonest symptoms of the more severe grades of amentia, and may be seen in practically any part of the body. While it is customary to classify infantile cerebral palsies into those having their onset in intra-uterine life, those due to accidents at birth, and those acquired subsequent to birth, in many cases the history is too meagre to decide to which special group a case shall be assigned, and it is perhaps more useful to divide paralytic amentia into two broad classes: the progressive and the non-progressive. To the former group belong juvenile general paralysis, amaurotic family idiocy, and certain rarer forms of cerebral degeneration. In the latter group one recognizes all forms of cerebral and spinal paralysis which do not of themselves lead to the death of the patient.

Among the progressive forms juvenile general paralysis is by far the commonest, and has already been referred to.

Of the other, amaurotic family idiocy is the best known, and the propriety of including it in the secondary forms of amentia may perhaps be questioned, for the disease is associated with a definite hereditary taint and occurs almost exclusively in members of the Hebrew race. But, on the other hand, the distinctive pathological changes and the clinical course of the disease afford good grounds for believing that it is of toxic origin and not due solely to inherited causes. As regards its clinical features, mental impairment beginning during the earlier months of life and ending in complete idiocy is one of the most striking symptoms, and this is accompanied by a progressive paralysis which leads to a fatal termination at about the age of 2 years. Diminution of vision terminating in absolute blindness is a constant feature, and is associated with changes in the macula lutea and subsequent optic nerve atrophy. The morbid changes in the nervous system consist of a widespread degeneration of the ganglion cells of the retina and central nervous system. Amaurotic idiocy is not often encountered in institutions for the mentally defective, but occasionally one meets with examples of the rarer juvenile form. In this type, which was first described by Spielmeyer and Vogt, there is no predilection for the Jewish race, and as the age of onset is later, and the fundus changes not pathognomonic, the disease is liable to be confused with other conditions, and particularly the juvenile form of general paralysis, which not infrequently begins at about the same age. To illustrate the clinical course, the following case may be cited.

The patient, the second child in a family of seven, seemed normal in every way until 5 years of age, when she contracted scarlet fever. Thereafter she was noticed to have become dull and, though able to go to school, made no progress and was still in the first standard when 7 years old. A little later her eyesight began to fail, and by
her thirteenth year she was almost blind. Soon afterwards she commenced to have fits, and when admitted to hospital in 1920 she showed nystagmus, inactive pupils, marked speech defect, generalized muscular tremor, and loss of sphincter control. Her mental development was equal to that of a child of 8 years. In 1924, when she came under my care, she was bed-ridden, contracted, and completely devoid of intelligence. The Wassermann reaction was negative. She died three years later from pulmonary tuberculosis. At the autopsy the brain was found to be small with atrophy of the cerebral convolutions and folia of the cerebellum. Microscopic examination showed widespread degeneration in the ganglion cells (fig. 14), atrophy of all cerebellar layers, and rarefaction of medullary nerve fibres.

**Schilder's Encephalitis.**

Brief allusion may also be made to the even rarer form of progressive paralysis to which Schilder has given the name encephalitis periaxialis diffusa. This disease, which may attack adults as well as children, is characterized by mental reduction, progressive blindness and bilateral paralysis, and a fatal termination in a few years. The principal pathological changes consist of demyelination of the white matter of the brain, spreading symmetrically from the occipital poles, and sparing the subcortical band of white matter. The patient in the accompanying illustration (fig. 15) is an imbecile girl in whom the disease has progressed very slowly during the past five years. Two other members of the family have been under the care of a neurologist, and in one the diagnosis has been confirmed by post-mortem examination. The general appearance of the patient corresponds very closely to the "attitude of adoration" described by Freud in his study of the diplegias of childhood. The head is retracted with the eyes staring upwards, the elbows are pressed into the sides, and the hands are held pronated below the face. Mentally, the patient is childish, timid and resistive. She can recognize different individuals by the sound of their voices, but has little knowledge of time, and a mental age of 8 years.

**Paralytic Amentia — Non-progressive.**

This group is a large one, and includes all cases in which there is old-standing destruction of the motor neurones. We may consequently meet with monoplegia, hemiplegia, diplegia, paraplegia, and certain forms associated with involuntary movements.

The paralyses of intra-uterine origin may be the expression of gross cerebral defects, polio-encephalitis, and agensis of the cortical neurones. Birth palsies may have as their pathological basis meningeal hæmorrhage, lobar sclerosis, cysts, and partial atrophies, while the post-natal forms may be caused by vascular lesions, meningitis and encephalitis.

It is often stated that while diplegias are for the most part congenitally determined, the hemiplegias and monoplegias are of post-natal origin, but to this rule there are many exceptions. The pathological lesions display much variety in their mode of origin, and to illustrate how difficult it is to arrive at any decision as to the pathological nature or extent of a lesion causing an infantile palsy, I may refer briefly to the following cases:—
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The first was a helpless idiot, aged 24, whose condition was stated to be caused by a head injury at birth. The patient had a divergent strabismus of both eyes and was paralysed on the right side, the right arm and leg being markedly wasted, but no shorter than those on the opposite side. Slight flattening of the cranium was present over the left parietal bone. At the autopsy the brain was found to be unusually small, and the left cerebral hemisphere sclerosed and shrunken to almost half the size of the other hemisphere.

The second was that of an epileptic imbecile, aged 18, with partial paralysis of the left arm and leg, dating from birth. He was able to wash, dress, and feed himself, and had a fair knowledge of time and place. When occupied with ward work he employed principally the right arm and hand, although he could hold objects with his left hand, and was able to bring it into use when sweeping or in cleaning utensils. The left foot was inclined to drag a little, but it did not hinder his progress to any extent, for he could climb stairs without much difficulty. At the post-mortem examination the cranium was noted to be somewhat flattened in the neighbourhood of the right parietal bone, and on exposing the brain a large cavity was found in the substance of the right cerebral hemisphere and covered by a thin sheet of arachnoid membrane. When this was removed it was seen that the cavity merged into the body and posterior horn of the lateral ventricle. The convolutions surrounding showed a somewhat radial distribution, and those absorbed in the cavity included the greater part of the pre- and post-central gyri and portions of the middle temporal and superior and middle frontal convolutions. The defect was therefore a typical example of true porencephaly, and was probably determined by an obliteration of certain branches of the right middle cerebral artery.

To describe the numerous types of paralysis which may be encountered would occupy more time than is at my disposal, and I may therefore pass on to a consideration of the mental features.

To a considerable extent the situation of the cerebral lesion is a factor in determining the degree of intellectual defect. Gross brain disease limited to one hemisphere is compatible with the milder forms of amentia, but when both hemispheres are involved the effect on the mind is always serious, for obviously disease of both halves of the brain must have a more disturbing influence upon the mental development of the child than a unilateral lesion. Moreover, gross cerebral lesions are sometimes merely complicating or reinforcing factors in an amentia originally determined by neuropathic inheritance, and in such cases, where both innate and acquired factors have been in operation, the mental defect is always marked, and often accompanied by gross stigmata of degeneration which give to the primary ament his ugly features. Diplegic patients are exceptional in that, though both halves of the brain are involved, their intelligence may be only slightly impaired, and they do not as a rule suffer from epilepsy. On the other hand, patients with paralysis limited to one half of the body often develop epilepsy in the early years of life and continue to have fits throughout their existence. Minor epilepsy is relatively uncommon, and in my experience has no influence on the defective's behaviour, but severe and uncontrollable epilepsy leads to a progressive deterioration in bodily and mental health, and many of its victims display the explosive epileptic temperament so often seen in the insane.

Another complicating feature in paralysed aments may be the presence of involuntary movements, particularly athetosis. When bilateral, the condition usually affects the face as well as the limbs, and with grimacing there may be pronounced articulatory defects.

Marie has stated that in double athetosis there is no disturbance of intelligence, but
in the few cases I have seen it has been possible to detect some degree of feeble-mindedness. Patients of this type are, however, usually cheerful, and are interested in all that goes on.

**General Management and Treatment.**

In conclusion, I would like to say a few words about the management of secondary aments.

Mental defect is a permanent and ineradicable condition, and consequently its victims require life-long care and control. Much may be done in some cases by careful home training, but unless the parents, and especially the mother, are able to devote a considerable part of their time and energy to this task, the patient will be better off in a special institution where he is able to associate on equal terms with companions, and thus be stimulated by the spirit of emulation.

The class of ament with whom I have dealt does not make very promising material for education in the "three R's," and for the majority our efforts must aim at making the best use of such capacity as they possess by means of industrial or technical training. It would take too long to detail the different ways in which this can be accomplished, but, briefly stated, the preliminary training consists in exercising the sense organs, in arousing spontaneity and attention, and in developing the reasoning powers as far as is possible. In idiots, even the power of instinctive attention is very feeble, and the most one can expect of them is the establishment of habits of cleanliness. With low-grade imbeciles rather more can be done, and in their training it is essential to make free use of play and organized games, care being taken that the periods of exertion are short. For the higher-grade secondary aments the provision of suitable indoor occupation such as sewing, knitting, mat-making, raffia work, chair-caning, and so on, is essential, and it is here that the services of a trained instructor are so useful.

In paralysed aments loss of movement in the affected limbs is seldom absolute, and much improvement can sometimes be effected. At the outset movement and co-ordination of the larger muscle groups are encouraged by teaching the child to grasp and move to their appropriate place large wooden insets, bricks and rings. Rhythmic exercises which involve synchronous movements are also useful, and at the end of six months, if these measures are carried out daily, the patient may have gained sufficient mastery of his limbs to be able to walk and to feed himself.

Dribbling at the mouth is a common and unpleasant symptom in paralytic amentia, and since it has seldom an organic basis, the habit may be eradicated by exercises directed towards strengthening the muscles of the mouth and pharynx. For this purpose one employs whistles, trumpets and pipes for blowing bubbles.

As regards general hygienic measures, mental defectives require to be warmly clad, and their paralysed limbs must be massaged and kept warm if chilblains are to be avoided. In the winter the number of articles soiled at night is always greater than at other seasons, for nothing so conduces to the habit of bed-wetting as cold, and the night nurse must be instructed to use extra blankets and to rouse his faulty patients at regular intervals.

The scope of medical treatment is restricted and, apart from the treatment of epilepsy, cretinism and syphilitic manifestations, medicinal measures are largely confined to combating sleeplessness and regulating the excretory organs.
The Secondary Forms of Mental Deficiency

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