lar need, and also attempts at desensitization by specific therapy, whether by means of protein extracts or bacterial vaccines. In many cases considerable benefit has been obtained by the method of general desensitization by various forms of protein shock, the method commonly used comprising injections, intramuscular or intravenous, of such substances as peptone, milk, anti-typhoid vaccine, and recently the use of tuberculin. Intramuscular injection of the patient's own blood in quantities varying from 1 to 5 c.c. at weekly intervals, is a measure which I have found of considerable benefit to many cases in the out-patient department, when for various reasons admission of the patient for prolonged investigation was impracticable. This has the advantage of an extremely easy technique, and I have never seen any unpleasant reactions or other untoward effects. Of a fairly large number of patients so treated a sufficient proportion have undoubtedly improved to an extent which has encouraged me to persist in the treatment. I believe that good results have also been obtained by intravenous injection of sodium iodide in normal saline. Apart from these special points, treatment on the general lines indicated in most textbooks is of importance. Attention to diet, regulation of the bowels and the regular administration by the mouth of iodide of potassium with tincture of stramonium are among the routine measures which do undoubtedly afford considerable relief. For the actual distress of the severe attack injections of adrenalin are probably the most effective and best known remedy, but it should be remembered that very unpleasant and even alarming symptoms of giddiness, faintness and even syncope have occasionally followed the use of this drug. The combination of atropin with adrenalin is said to lessen the risk of occurrence of such symptoms. As an alternative to adrenalin the new synthetic preparation ephedrin has been found of considerable use, and has the advantage that it may be

given by the mouth in doses of half a grain.

Attention has been called by Scott [5], to the value of X-rays in the treatment of asthma, beneficial results having been obtained from applications once or twice a week with a small dosage over a large radiation field. According to his experience this seems to offer considerable possibilities for future research.

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AMENTIA IN RELATION TO CEREBRAL DISEASE AND ABNORMALITY.

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I MUST begin by admitting freely to you that I am not speaking as a specialist or expert in the scientific aspect of mental deficiency. I am attempting to speak on the subject from a different point of view; to discuss the aspect of mental arrest or impairment which is met with by the neurologist in the cases which are brought to a children's hospital or a nervous hospital, or seen in consultation. I think that this point of view is worthy of some consideration, because a very large number of these children are brought to the neurologist, and because the element of amentia which is present is one of the most important factors in the treatment of these patients, whatever

the actual symptoms may be which have attracted the attention of the parents. My object will merely be to describe some types of cerebral disease or abnormality which are likely to involve some element of amentia, and to discuss the bearing of the latter on prognosis and treatment.

I should like to make a few remarks in explanation and defence of the rather unscientific attitude which I shall take up with regard to classification. You are all aware of the fundamental distinction, in theory at any rate, between *amentia* and *dementia*, dependent on the question of whether the defect was due to a failure of development or to a deterioration of faculties already developed. In adolescents and adults I think that this is a distinction of great importance, and that every effort should be made to keep it clearly in mind. For this reason I view with misgiving the recent legislation which has widened the scope of the Mental Deficiency Act so as to include sufferers from epidemic encephalitis. Nevertheless, in children one has to admit that it is often absolutely impossible to establish any hard and fast distinction between these two types of defect, because one has no means of determining how much arrest or retardation there has been, or how much is to be accounted for by subsequent deterioration. Take as an instance an epileptic boy of 9 with an intelligence defect; how is one to estimate, in the absence of a reliable medical history, the extent to which his mental faculties may have developed in the first instance? From the fact that "the age is early" one may be inclined to class him as an ament; from the fact that he is now deteriorating one is given equally good grounds for regarding him as a dement.

Still more emphatically must one point out the limitations of the attempted distinction between primary and secondary amentia, when these terms are applied to children who suffer from some form of cerebral disease. I personally do not hesitate

to say that in the present state of our knowledge this distinction cannot be made, and that to make it involves the begging of some unsolved questions. The reason for this is that the whole problem of the ætiology of infantile cerebral disease is in the melting-pot at the present moment; there are many conflicting opinions and but few established facts. There are a number of morbid conditions which a few years ago were confidently classified as "primary"—attributed to agenesis or true developmental failure—but on the ætiology of which no such unanimity prevails to-day. Perhaps the best instance of them is porencephaly. Porencephaly is a condition in which cavities, sometimes of considerable size, are found in the cerebral hemispheres. It is found in post-mortem examinations, sometimes of cases showing no definite abnormality during life, but more often of cases which show signs of lesion of the cortico-spinal tracts, or of mental defect, or of both. Until fairly recently it was assumed on all sides that this was a prenatal defect; it was described by some authorities as being due to a primary agenesis, and by others as the result of thrombosis or embolism during intra-uterine life. In many books you will find reference to primary porencephalic amentia. At the present day, largely as the result of the researches of P. Schwartz, of Frankfort, the view that porencephaly is a primary defect is by no means universally held, though it has its adherents. Schwartz has brought forward a considerable amount of evidence in support of his contention that porencephaly is an entirely secondary condition, is in fact the end-result of venous hæmorrhages from tributaries of the vein of Galen, which takes place during delivery or shortly after. From this it will follow that anyone who, like myself, regards the hypothesis of Schwartz as at any rate more convincing than any other, will look on an amentia associated with porencephaly, or a diplegia associated with porencephaly, as being *par excellence* a secondary condition;

one who does not share this conviction will continue to speak of a primary porencephalic ament. I merely mention this as a good example; similar doubts may be raised about almost any other morbid cerebral condition in early life. The point which I want to stress is simply that at the present state of our knowledge the ætiology is a matter of opinion and not of fact, and that it is unscientific to embark confidently on the diagnosis of primary amentia in individual cases. That the majority of them are primary is likely, because the preponderance of the hereditary factor points to a germinal defect, but clinical data are insufficient for the differentiation of all but a few of them, and even the morbid histology of some of them is so obscure that microscopical examination of the brain does not fully establish the nature of the condition. I think that it is within the experience of most of us to have found some definite indication of cerebral disease in the post-mortem examination of a case which we had previously regarded as a "simple primary ament." In the cases which I shall discuss I shall treat mental deficiency entirely from the symptomatic and practical point of view, without much reference to its exact origin.

The type of child which concerns us most is that which is roughly classed as *infantile spastic paralysis*. Patients belonging to this group (it is merely a group and not a disease-entity) present a problem of far greater importance than do the other types to which I shall refer, because in them the question is not merely one of diagnosis, classification, prognosis, but of the possibility of active, effective treatment. Furthermore, the estimation of the degree of mental defect present in these children is of the greatest practical importance to the neurologist, because it is the factor above all others which stands in the way of the treatment which is necessary for recovery of the motor disability.

Infantile spastic paralysis is the result of a lesion of the cortico-spinal tract which

constitutes the upper motor neurone. The lesion is typically in the cortex, or the white matter of the subcortical region. The paralysis occurs as either a hemiplegia or a diplegia; the latter group is commonly referred to as Little's disease.

The ætiology is a much-disputed subject which does not closely concern us at the moment. I will just mention the three principal views which are held on the *usual* mode of their origin; I think that all will agree that each of these causes is responsible for *some* of them, but opinions differ as to which is the cause of the majority. They are regarded as: (1) Primary degeneration of the cerebral neurones (Collier); (2) residual effects of meningeal hæmorrhage, and particularly of the superior longitudinal sinus (Adie); and (3) residual effects of intracerebral hæmorrhage, particularly of tributaries of the vein of Galen (Schwartz).

The symptoms presented by infants who are affected by lesions of these kinds will of course vary according to the exact site of the disease or damage. The symptoms referable to the pyramidal tracts will be very slight at birth and in the first few months, because the infant's pyramidal tract is incompletely developed, and its movements are dominated by lower centres. Later on, a child with a pyramidal lesion will develop spasm in the affected limb, and will tend not to develop new voluntary movements in it. Within wide limits, those in whom the cortex is involved tend to suffer from fits and mental impairment, while those whose lesions are subcortical suffer from a greater degree of spastic weakness.

Now the *prognosis* in these children depends upon the extent to which they will react to treatment, and the only effective treatment is the initiation of active movements. To get this done, various measures are usually necessary which are perfectly simple but require a good deal of perseverance. For instance, in a hemiplegia the sound arm is tied up, so that if the child wants to handle anything it must do it with

the stiff one. The normal infant does want to handle things, and its instinctive desire to do so is made use of in this way, and in a great proportion of them a very good ultimate result may be obtained. But the infant who has suffered extensive damage in the cortex, particularly of the frontal lobes, may suffer from some degree of amentia in addition to the motor disability. In this case the normal instinctive activities of the infant—almost ceaseless during the waking state—are replaced by a state of apathy which is an absolute bar to the initiation of active movement; you may tie up the sound arm, but the child will merely lie with both arms immobile. For all these children, aments or otherwise, the process of learning is going to be more difficult than for normal ones, and in later years all new functions must be taught them with a good deal of patience and perseverance. But this is of no avail unless there is a will to learn, and if the child's attitude is one of apathy or negativism the improvement cannot be achieved. For this reason the prognosis in a case of infantile hemiplegia or diplegia depends upon the presence or absence of associated amentia, and not nearly so much on the condition of the affected limb. The prognosis in a very large number of these cases, when there is not an appreciable degree of mental impairment, is really quite good, so that the determination of the mental state is very important; it constitutes a question about which you will always be pressed by the parents when you see these children in the course of practice, and the problem which you will then be confronted with is precisely that which I want to discuss at some length now.

The first point to remember is this; in giving a prognosis of this kind you are not estimating the child's intelligence at the moment, but trying to arrive at some idea of what it may be in the future. In the simple primary aments the degree of intelligence remains *roughly* constant; a child which has the characteristics of an imbecile at the age

of 4 is likely to have them to a fairly similar degree at 8. As a result of this we have got into the habit of talking of the condition as a permanent one, and of basing the prognosis simply on the results of mental tests at a given moment. In general, I think that prognoses made in this way, by experienced observers, turn out to be right in a remarkably high percentage of cases, as far as simple primary aments are concerned. But I am firmly convinced that when we are dealing with children who are suffering from a cerebral lesion—particularly a residual one—this is very far from being the case, and the more experience I have of these children the more am I confirmed in this opinion. Except in cases of gross idiocy, I do not believe that when you see a diplegic or hemiplegic child in the first three or four years of life, you can do any more than say that the mental development is *retarded at the moment*, a fact which is probably obvious to all unbiased observers, though not always to the parents. You know that the child has leeway to make up, and will need a good deal of help in doing so, but I would caution you against prophesying as to how much it *will* do so, until you have observed for a considerable period the way in which it responds to your efforts to further its development. If you proceed as if you were dealing with a simple primary ament, and say that the child will or will not attain the mental development of a more or less normal individual, I think that you will be embarking on a venture which may lead to a good deal of trouble for yourself, and perhaps some risk of interfering with the child's chances of obtaining suitable treatment. I am ready to admit that I am unable to diagnose permanent mental defect in these children, with any certainty, in infancy and early childhood. I am very much inclined to doubt whether anyone else can do so, as it is so often my experience to be consulted about a child of 8 or 10 who still has some degree of paresis, and to hear that a diagnosis of mental deficiency has been made by a

competent observer in earlier years, though no evidence of it has remained. These children, I may remind you, will be treated by the general practitioner rather than the mental expert, and they are at least as important to you as the more typical forms of amentia; this is my excuse for going into some detail about them.

If one considers the clinical characteristics of these children, some of the reasons why one can foretell so little about their mental possibilities become obvious. They have a motor disability, by which movement is rendered difficult or impossible in some part of them. It is not only the gross movements which are hampered; complicated voluntary movements such as those involved in speech are affected. Moreover, one of the most important functions of the corticospinal tracts is inhibition of superfluous movements which in the infant have been performed by lower centres. Consequently a fairly common symptom is hyperkinesia in some form or other, particularly grimacing. There may be some stiffness of the pharyngeal muscles, so that the child dribbles. So that you may be confronted with a child of about 3 who does not talk, grimaces, and dribbles. If you meet with this combination in a child whose motor system is normal, further investigation will usually show evidence of mental defect. But if you find that there is a pyramidal lesion, whether hemiplegia or diplegia, then you must discount all these purely motor disturbances as evidences of mental impairment, because you cannot tell whether the failure to speak is due to an intelligence defect, a failure to register impressions, or whether it is merely that the child has not yet overcome its mechanical difficulties in articulation.

Now when you come to estimate the intelligence of a small child, almost all the tests which you use involve some motor function in their performance; you are left with almost nothing else. But you generally can form some idea of the amount of interest

that the child takes in his surroundings, by observing the response to visual, auditory, and tactile stimuli as shown by movements of eyes or some part which is not spastic. A state of *apathy*, in which there is no desire to watch or handle objects, is the principal if not the only symptom which you may regard as really ominous. But even this must be interpreted with a certain amount of caution; do not attach too much significance to apathy until you have found out whether the child is apathetic under favourable conditions. The majority of these children are late in reaching the so-called milestones of infancy and childhood; they are late in sitting up, crawling, &c. A very important part of the treatment both of the mental and the physical disability consists in correcting or compensating this delay in adopting new positions. For instance, a child of 15 months who has never done anything but lie on its back may present the appearance of extreme apathy, but as soon as you do what should have been done long before, but very often is not done, and get it into a sitting posture by the use of pillows, a chair with a bar, or some other means, it may rapidly acquire an interest in its surroundings and begin to do a lot of things which it could not do while lying down. If there is no response after a number of months to your efforts to stimulate the sensori-motor reactions while keeping the child in a position which will give him a fair chance, then you are justified in a strong suspicion that permanent mental defect is present.

What I have said so far applies mainly to infants and children of the pre-school age. To summarize: When there is a spastic lesion one should be very chary of diagnosing mental defect in the sense of a *permanent* condition (i.e., of a kind which will come under the M.D. Act); this can only be done with certainty by observing over a long period the extent to which the child's interest can be aroused when suitable conditions are provided. It is of the greatest importance that this should be done

early. Too often one finds that it has been postponed and that the child has lain inert for months or years. I am used to hearing two phrases, sometimes attributed to the doctor; that the child "needed rest," and that "they thought that it would grow out of it." They do not need rest, they need activity and interest. They do not often grow out of it spontaneously, but are apt to deteriorate if left to their own devices.

The next point to consider is the mental state of these children at a later age, say 5 to 10. It is at this stage that the question often comes to a head, because the child has now reached the school age, and the question of *educability* arises. At this period one can attempt to form some more definite ideas about the prognosis, but still there are a number of pitfalls. In the first place do not judge by appearances. The silly, inane look, and the indistinct "mouthing" articulation, which give an appearance of mental vacuity, are often really the effects of dysarthria, and the child has good powers of understanding and talks quite good sense. In the second place, if you are going to apply mental tests, put your watch in your pocket. The standardized time-limits, which are I think justified in the result when applied to ordinary aments, have no place here. The child is running its race in splints, and is not going to achieve anything like normal reaction times. Such tests as ability to interpret pictures, tell the time by the clock, tell you something about the street he lives in, are the most valuable, for they are the tests that give you evidence as to whether he is registering and retaining impressions.

Now as to this question of "educability," which is very likely to be the point on which you will be consulted.

Let me remind you that the child will come under the scope of the M.D. Act if it "appears to be permanently incapable of receiving proper benefit from the instruction in ordinary schools." This classification, intended for and admirably suited to the

primary aments, raises considerable difficulties when applied to cerebral diplegics. The majority of them are unable, I think, to obtain proper benefit in them, and are moreover liable to develop unsatisfactorily if thrown among normal children. The reason of this is that they are under these circumstances so liable to suffer from the very thing that it is most important to protect them from; a sense of inferiority. If the child does this, half the battle is lost; it becomes discouraged and diffident, avoids effort from fear of failure, and tends to recede into itself and live in phantasy. There is also a tendency to compensate in other ways, and the fact that some of these children develop unpleasant traits, such as spitefulness and cruelty, is due far more, I believe, to their very natural sense of inferiority than to the actual cerebral damage itself. For these reasons one is anxious to get them out of the ordinary schools. But I personally am very reluctant to admit that in the majority of cases the "inability to profit" is permanent, and I think that doubtful cases should not be certified. And if they are sent to special schools for mental defect the disadvantages are nearly as great. They are among children, the great majority of whom are never going to make much progress, who are, to put it unscientifically, "duds"; a fair proportion of them are also mischievous and generally unpleasant. This does not constitute an environment which will help the child to overcome the handicap with which he started. When it can be done, I think that far the best thing is to get the child into a special school for cripples, and overlook as far as possible the mental disability. In private practice, the ideal to aim at is to get these children educated by a tutor or governess in groups of three or four. Mind and body require to be developed simultaneously, and special attention should be paid to handicrafts and such activities. I think if you follow such cases in your practices up to the years of adoles-

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cence, you will often have occasion to be glad that you saved one of these children in earlier years from certification as feeble-minded.

There is one important point which I purposely did not mention until now, the significance of *fits*. This is a very difficult question, on which opinions seem to differ a good deal, and I can only give you my experience. Of the cases which I have seen, the great majority, both of those who have done well later and those who have done badly, have had fits in infancy. And of the minority who have not to my knowledge had fits, not all have made good progress. So I am not able to draw any prognostic significance from fits occurring during the first eighteen months. As you know, quite apart from the presence of cerebral lesions, fits in tiny children mean rather little, and fits in older ones or in adults mean rather a lot. But of the cases I have seen who have made good mental progress up to and beyond puberty, very few have had more than an occasional fit after the fourth or fifth year. The onset of measles, or a fall, or an unduly exciting occasion, may perhaps be a legitimate stimulus to account for a fit, but if convulsions or lapses of consciousness occur during the child's normal activities, on more than a few occasions after the age of 4, I personally do not regard the prognosis as at all favourable. Fits also influence the progress in another way, and I believe it is an important one. In order to control them one must give certain drugs. These drugs are usually "sedative," intended to diminish mental activities. The ones usually given certainly have a deleterious effect on the child's mental progress. I think that a great deal of the retardation of many of these children is due to the prolonged use of bromide, given in large doses over a long period, sometimes necessarily, but I think usually unnecessarily. On this point I know that opinions differ, but I have entirely given up the use of massive doses of bromide, and I

make it my object to find the smallest dose of whatever drug I am using which will keep the child more or less free of fits; I would rather that the child had a fit or two in the year than that it spent the year drenched with bromide. And in the majority of cases that are going to do at all well, I generally find that small doses will do this. I always try first to see what can be done with a drug which I find perfectly harmless, borax. Of course it is not nearly so effective as the others, but in a mild case it may be enough for a long time, and it is a great advantage to the child to escape the more potent drugs. If this does not control the fits, I add bromide, in small doses such as $\frac{1}{2}$ gr. to $\frac{1}{4}$ gr. t.d.s. I seldom go higher than this; if it fails I go on to luminal for a few weeks and then back to the borax. I am fully convinced in my own mind that children make better mental progress on this treatment than if they are kept perpetually under the influence of bromide.

Before leaving this subject I should like to say a word about the question of giving a prognosis, which is so very important to you in general practice, because I have found so often that the parents have been resentful with or have lost confidence in their doctor because of what has been foretold, or more often not foretold, about these children. Do not tell them that the child will "grow out of it," or words to that effect. Tell them that the brain is damaged, but that the mind is capable of being developed to a considerable extent; that there is a very good chance of the child becoming a normal member of society, but one who is not going to be suitable for hard brain work. If in the course of time you find that the child is not progressing, and is clearly an ament, tell them so, gradually perhaps, but at any rate let them know it from you rather than discover it in spite of you. Otherwise there is always a tendency for them to think that if you had known it earlier something more might have been done.

Epilepsy.—Of all the mentally retarded children which are commonly seen by the neurologist, epileptics are probably the commonest. I do not want to say much about them, but to point out that, as we see them in early life, they do not make up an entity, but are merely a mixed group of a whole number of conditions exhibiting a common symptom. Some will turn out normal in the end, others will be cases of chronic idiopathic epilepsy, others no doubt have cerebral birth lesions of which there are no physical signs. But in early childhood one cannot distinguish these types from one another. The main characteristic of the mental impairment is usually an extreme lack of attention of a particular sort. It is not real apathy, the child is always taking notice of something, but it is impossible to get him to keep his attention on any one thing for any length of time. Consequently many of them seem fairly normal in the pre-school age, but as soon as lessons begin they fall behind their fellows and are always getting into trouble. In my experience the greater number of epileptics showing slight amentia (one is but little concerned with the grosser cases of imbecility) are those who have few definite fits but fairly frequent "absences," momentary lapses of attention without any muscular relaxation or loss of consciousness. A child of this kind often has a true fit every now and again, perhaps when he is convalescing from a fever or gets unduly excited. This is a type of patient which will often be brought to you, not so much for treatment as for advice about the sort of life he should lead. Though the prognosis is not very good, taking these patients as a whole, yet it is worth doing what you can as long as the amentia is mild, as some of them do improve. The first thing is to get the parents' attention directed away from the fits, and on to the mental development, which is an aspect of the case that has often escaped them. Make them realize that he will want more teaching, not less, than a normal child, but that it will have to be done quietly and patiently. Get them to keep the

child occupied, particularly in such ways as learning to make things, or nature study. Secondly, try to control the fits with the smallest, not the largest dose of drugs that will do it. Finally, see that the child is not prevented unnecessarily from doing things. A few pursuits, such as riding a bicycle, or swimming alone, must obviously be forbidden, but they can do nearly all the things that another child does, and are much the better for it. Watch the result of your treatment for a year or so with an open mind. You will have a fair number of cases whose mental retardation does not turn out to be permanent; on the other hand, when you do find that it is progressive, you will do well to recognize that the prognosis is unfavourable, and admit

(*To be continued*).

THE CANARY ISLANDS AS A HEALTH RESORT.

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It has always been a source of wonder to me that the Canary Islands have not become more popular as a health resort, more especially of late, since I have become more intimately acquainted with them personally in search of health.

I can only think that the boggy of the Bay of Biscay exercises a terrorizing effect upon the travelling public, but, as I will show later, this drawback may be obviated.

The Canary Islands are the nearest resort to the British Isles where one is *certain* of warmth, sunshine and dryness during the winter months.

The group consists of seven larger islands and several smaller ones. They are distant roughly 1,700 miles from England. They are situated between the latitudes $27^{\circ} 4'$ and $29^{\circ} 3'$ north and longitudes $13^{\circ} 2'$ and