

AMAUROSIS IN INFANTS.¹

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At birth the infant possesses certain faculties, while others develop during the period of growth. The faculty of vision is among the latter, and certain components do not appear for many months. The normal infant is born into the world with a retina sensitive to light, with the beginnings of a specialised central area—macula—in the retina, as evidenced by the power of momentarily fixing a light, and with good coördinated control of eye movements in the vertical plane. After the first few weeks, usually at about the fifth week, the power of prolonged fixation has appeared, and attempts at binocular fixation can be observed. The horizontal movements of the eyes are not completely coördinated and under control until about the sixth month of life; and at this age, definite desire for binocular fixation can be demonstrated. At the end of the first year binocular vision should be well established. Binocular vision, or, in other words, the desire for fusion of the images of the two eyes, continues to develop up to the age of six. This is significant when dealing with cases of squint, in which absence of this faculty of fusion is the underlying cause, as, after the age of six, attempts to improve this faculty will fail. It has been stated that the child's real "field of vision" is largely dependent on its powers of locomotion. During the first few months the child recognises, and clutches, objects only when they are brought within its grasp. Good judgment of distance is not fully developed until the third year.

It is necessary to keep the above-mentioned features in the vision development of the infant clearly in one's mind when dealing with cases of supposed defective, or absent, sight in young children, and further to remember the usual date of appearance of other powers—holding up of the head, sitting up, &c.—which gradually make their appearance in the growing infant. It is a fairly common experience among the ophthalmic out-patients of this hospital, to have infants brought up because their parents have doubts as to whether they can see, and it is first necessary to confirm the parents' diagnosis. The infant should be taken into the dark room, and the bright light from an electric torch flashed into the eyes, from a distance of not more than 2 ft. The light should be flashed from each side and from in front of the infant. If there is sight present, momentary fixation of the light should occur. This test will eliminate the totally blind infant, but routine examination of the little patient should, of course, be carried out in all cases, and the fundus examined after dilatation with a mydriatic, for which examination, in nearly all cases, an anaesthetic will be required. In making the routine examination particular attention should be directed in the first place to the reaction of the pupils, the movements of the eyes, the presence or absence of squint or of nystagmus, the colour of the irides, and the state of refraction of the eye. It should be noted whether head-nodding or head-shaking has been observed, and inquiry should be made to ascertain whether the infant rubs its eyes. This symptom often denotes a healthy retina, but with cloudy or opaque media. The child tries, as it were, to rub away the obstruction which prevents the light from reaching the retina. The parents should be questioned as to possible consanguinity, and the family history investigated. The position of the infant in the family should be found out, and particulars as to its birth ascertained. Any serious illness, or the history of a fit, or fits, should be inquired into.

Impairment of Vision with no Gross Ocular Lesion.

Now, it is obvious that a variety of causes may be acting to produce an impairment, or absence, of sight in an infant. I do not propose in this lecture to deal with those causes in which an obvious ocular lesion is found, such as cataract, malformation of the eye, or gross retinal disease. Such cases explain themselves. Rather do I intend to deal with those cases in which no gross ocular lesion is discoverable.

It has long been recognised that apparent absence of sight in a young infant may be the first sign of future mental deficiency. It is easy to lose sight of this fact when the case is viewed from an ophthalmic aspect, but it should always be borne in mind. A considerable number of infants who are first brought up on account of their sight fall into this category. It is not, of course, possible to make the diagnosis at the first visit. The child should be watched at regular intervals, and the appearance of other powers should be looked for. For instance, the infant should be able to hold its head up by about the fourth month, and should be able to sit erect by the ninth month. If these powers are late in appearing the diagnosis will be strengthened. Delayed eruption of the teeth can be taken as further evidence of future mental deficiency. Mentally defective children, for the most part, eventually obtain some vision, and are usually able to get about by themselves. The lesion is probably cortical, as in every case which I have observed the pupils reacted well to light.

Partial Albinism.

The next group of cases to which I wish to draw your attention are those cases in which there is a deficiency of pigmentation in the tunics of the eyes. Albinism is well known to be one of the commonest causes of nystagmus. Everyone is familiar with the full grade albino, but in many cases the pigment deficiency may be local, and may only occur in the retina and choroid. The iris of the new-born infant is usually blue in colour, even in those cases which eventually develop brown irides. The brown-ing of the iris is due to the deposition of pigment, occurring in the first few months of life. The proper pigmentation of the eye, therefore, is not fully accomplished at birth; but many cases have the iris sufficiently pigmented to prevent the typical red pupil of the albino, yet there is not sufficient pigmentation of the choroid and retina to subserve good vision. It is this type of case to which I am referring. The appearance is reasonably characteristic in its ophthalmological picture. The general colour tone of the fundus is lighter and brighter than normal, and the larger choroidal vessels can be seen shown up upon the white sclerotic as a background. But it is a case for a comparison with normal—the normal infant's fundus shows a relative pigment deficiency as compared with the adult—and it is rather a matter for the expert with the ophthalmoscope to decide upon the due preponderance of this feature in any given case of amaurosis. It can be definitely stated, however, that deficient pigmentation is a cause of delayed development of fixation, and that the eventual prognosis as to sight in such cases is good.

Temporary Amaurosis Associated with Basal Meningitis.

Thirdly, I would mention those cases of temporary or fleeting amaurosis which are associated, in all probability, with a basal meningitis. They are a clearly-cut group. The children affected are usually a few months old. There is the history of an illness, often quite slight, though at times more severe, which may be associated with fits, head retractions, and vomiting. Following upon this the infant's sight, which had previously been quite good, fails. This failure of sight may be only for a few days, after which it returns unimpaired, or it may be for longer. The loss of vision is in proportion to the severity of the illness. These cases of fleeting amaurosis recover

¹ Substance of a lecture delivered at the Hospital for Sick Children, Great Ormond-street.

completely, and there are no signs of disease in the eyes—such as optic atrophy—to be seen subsequently.

In all probability, however, these cases are identical in origin, though differing in severity, with those cases in which blindness ensues after a somewhat similar but more severe initial illness, and in which pallor of the discs is subsequently found. It has been suggested, as an explanation of the pathology of these cases, that a basal meningitis seals temporarily the foramen of Majendie, and so causes distension of the third ventricle, which by pressing upon the optic chiasma interferes with the visual pathways.

The following cases will illustrate the conditions associated with amaurosis in infants, which I have attempted to describe in this lecture.

Mental Deficiency.

CASE 1.—Female infant. Was first brought up to hospital in 1906 because the parents thought that she had defective vision. She was prematurely born at the seventh month, and suffered from fits for the first two years of life. Fixation was very doubtful, and though the discs were noted as being suggestively pale, yet it was considered that there was not sufficient in the fundi to account for the defective vision.

She was seen again in February, 1914, when 9 years old. "Is an obvious idiot, does not seem to see, though on occasions appears to notice things. Can talk well but without sense. Lower limbs completely spastic, cannot sit up alone, no control of sphincters. Is quite a baby in her habits, sucks a teat, gets into severe tempers." The pupils reacted readily to light. The eyes were markedly divergent. The discs showed temporal pallor, but the fundi were normal in other respects.

She was last seen in January, 1921. "General condition much as before, idiot, spastic flexed legs, cannot walk, rolls head frequently from side to side. Eyes divergent, pupils active to light, sees light and looks towards it sometimes."

CASE 2.—Female infant, when first seen 7 months of age. Was brought up because she "took no notice." She was one of twins at the mother's sixth confinement. The other twin died from "bronchitis"; rest of family alive and well. She has had "fits of spasms and cryings" since a fortnight old. General condition seems good, but she does not yet hold her head up. Eyes parallel, no nystagmus, pupils react to light. It is difficult to obtain fixation, except occasionally with a very bright light. Media of the eyes clear, and fundi normal.

She was next seen two months later: "Did not fix the light; the fits have now ceased; can only just hold her head up; movements of the limbs appear good; is sleeping better; no teeth yet." Two months later again, aged 11 months: "Fixation is obtained with a strong stimulus. It appears to take a stronger stimulus to evoke fixation than with a normal infant. Has now cut two teeth."

CASE 3.—Female infant, aged 8 months when first seen. "Mother thinks that she does not notice things. First child, no previous miscarriages, fits for first six months; these have now ceased, good general condition, moves limbs well, eyes straight, no nystagmus, pupils react, fixation difficult to obtain, if at all. Media clear, fundi show a slight pigment deficiency." Seen two months later: "Fixation obtained more easily, occasional convergence." Seen two months later: "Mother says that the child does not seem to see as well as other children of the same age, and is backward. Cannot sit up by herself yet, no teeth, does not crawl. Will fix and follow the light, but seems slow in making up her mind to do so. It would appear that a more powerful stimulus is needed to arouse her attention."

The first case has been followed to its conclusion. Of the second and third cases this can hardly be said, but I would point out that no satisfactory lesion in the eye can be found for the defective vision, and that the initial fits, the failure to hold the head up, to sit up and to crawl, and the late appearance of the teeth, are suggestive features. Further, the manner of the fixation of the light by the eyes, when obtained, is very characteristic. These infants have a curiously languid way of performing this act.

Pigment Deficiency.

CASE 4.—Male infant, aged 4 months when first seen. Was brought to hospital "because he took no notice and rubbed his eyes." "Seventh child; other children alive and well and have good eyes; all are very fair, as also is the father; normal, healthy infant in other respects; eyes parallel; slow horizontal nystagmus; pupils react to light; fixation difficult to evoke, if at all; media clear, fundi show marked pigment deficiency, large choroidal vessel shown up on white sclera as background."

Seen five months later; now definitely follows objects. Mother says that he does not like bright light. Nystagmus had ceased until a few days ago when he got a bad cold, since when it has returned.

Temporary Amaurosis.

CASE 5.—Female child, aged 18 months. Quite well until one month ago; then "screaming convulsions," after which the child was apparently blind. Felt for its bottle and did not see it, crawled round its cot and hit its head. When seen both pupils were large and reacted very fully to light; fundi showed no abnormality. Seen again a week later: Child now sees well, pupils normal size and react to light readily.

These cases are taken from the report of an investigation, carried out at the Royal London Ophthalmic Hospital by the Lang research scholars, which appeared in the *British Journal of Ophthalmology* for August.

BACTERIAL FOOD POISONING FROM MUTTON.

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To the medical officer of health, the pathologist, and, to a lesser degree, the general practitioner the sudden outbreaks of serious and sometimes fatal disease still commonly, but erroneously, referred to as "ptomaine poisoning" are always of great interest. Although more than one hundred such epidemics have been reported in this country, and although about 80 per cent. of these were due to flesh foods of various kinds,¹ no instance is recorded where mutton was proved to have been the food primarily responsible for the transmission of the disease. In May of this year, however, there occurred in Manchester a limited outbreak of food poisoning in which, in all probability, this relationship was present.

Summary of Chief Features of Outbreak.

Six separate households were affected, the number of individuals attacked being 25. Abdominal pain, vomiting, diarrhoea, and muscular cramps were present in most cases. There was often profuse sweating, and in a few cases fever and delirium were reported. One patient, D. G., aged 40, died on the third day of illness; in the other cases recovery took place after an illness of from about 24 hours to 7 or 8 days. The average period of incubation was found to have been from 17 to 20 hours.

The clinical diagnosis of bacillary food poisoning was rapidly confirmed by the isolation from the tissues and secretions of D. G.² of a bacillus of the food poisoning (Gaertner) group, further particulars of which are given later. Also, the blood of most of the individuals attacked agglutinated in relatively high dilution (1:250 in one case) both a known bacillus of the food poisoning group (*B. suispestifer*, type Mutton) and the bacillus isolated from D. G. Other organisms of the food poisoning group were agglutinated in lower dilution or not at all. Bacteriological examination of the faeces and urine from a few cases was negative.

All the individuals attacked had recently eaten some kind of meat food purchased from the same retailer. Four persons in one house became ill after eating "veal chops," but in every other case the illness followed the consumption of some form of mutton (leg, shoulder, breast of mutton; mutton chops). Six patients ate, in addition, some beef roasted in the fat in which some of the mutton was cooked. A notable negative instance was provided by the case of

¹ Savage; Food Poisoning and Food Infections, 1920.

² The public health authorities were first notified of this outbreak through the coroner's office.