An Address

ON THE

CLINICAL TYPES OF CONVULSIVE SEIZURES
IN VERY YOUNG BABIES.WITH A SPECIAL CONSIDERATION OF THE SO-CALLED
"IDIOPATHIC CONVULSIONS OF EARLY INFANCY,"
AND THEIR TREATMENT.*

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THERE are few clinical phenomena more familiar to us as medical practitioners than the convulsions of infancy, and very few which present more features of interest. We have learned many things about their causation and results, but there is still a great deal waiting for us to find out by careful clinical observation, as well as by the, to some extent, more exact methods of the laboratory. You will therefore, I hope, agree with me that they form a theme to which we may profitably turn our thoughts for a short time.

As the subject of infantile convulsions in general is rather large and many-sided to be considered adequately in one lecture, I propose to limit my remarks to a consideration of the causation, diagnosis, prognosis and treatment of those varieties of convulsive seizure which may be met with in very young children—those of three months old and under—and to draw your attention particularly to the clinical features, treatment and etiology of a type of so-called "idiopathic convulsions" in little babies which presents a number of points of especial interest.

One small detail regarding the incidence of fits in infants at this age may be noticed in passing—namely, that, in spite of the frequency of birth injury to the brain, which acts as a cause of convulsions mainly in the first week, and the occurrence of certain other forms of convulsions in young babies which are rarely present in later infancy, fits are less common in the early weeks of life than in older infants. Probably the main reason of this is that spasmophilia, which has such a strong effect in producing a convulsive tendency, is never seen before the third and only rarely before the fifth month.

The accompanying table and chart show the frequency with which first convulsions occurred at different age periods in 4,348 children under 4 who attended my outpatient clinic many years ago.

Percentage of Cases of Convulsions beginning in different Six-month Periods, among 4,348 Out-patients under Four Years old.

It will be seen that the fits began during the first three months in only 3.5 per cent. of the cases, during the second six months in nearly 8 per cent., during the third six months in nearly 9 per cent., and during the later six-month periods in between 7.1 and 7.6 per cent. of the cases.

I have recently been studying the notes of 200 cases of convulsions in infants of 3 months and less. These have brought out some points of practical interest; and it is chiefly to the information derived from them that I wish to direct your attention now.

CAUSES OF CONVULSIONS.

The causes of convulsions in very young babies are numerous and of many kinds. These, so far as I have

been able to diagnose them in my cases, may be divided into three groups.

A. Local Injury or Disease of the Brain or its Membranes.

1. Birth injury (intracranial haemorrhage, etc.) 37
2. Meningitis ... 7
3. Hydrocephalus ... 4
4. Other brain lesions (acquired and congenital) 5
5. Congenital idiocy from arrest of cerebral development (with *petit mal*) ... 18

B. Cerebral Disturbance due to Acute Disease of Organs other than the Brain.

1. Food disorders (functional and organic dyspepsia) ... 46
2. Jaundice of various kinds ... 2
3. Genito-urinary (infection of the urinary tract with *Bacillus coli*, etc.) ... 5
4. Bronchitis and bronchopneumonia ... 8
5. Choroiditis, otitis, congenital heart disease ... 4
6. Unknown causes ... 7

C. Cerebral Disturbance connected with Various Forms of General Infection and of Debility.

1. General tuberculosis ... 7
2. Congenital syphilis ... 10
3. Debility ... 2
4. Whooping-cough ... 3
5. "Idiopathic convulsions" ... 35

The number of cases due to each of the various causes in this list cannot be held as representing accurately the relative frequency of the different causes as they occur in ordinary practice, for they have been taken almost only from hospital and consultation practice, in which the cases due to more serious and unusual causes necessarily bulk much more largely than those which are obviously the result of passing conditions, such as indigestion.

Let us consider these various causes one by one in detail.

A.—LOCAL INJURY OR DISEASE OF THE BRAIN OR ITS MEMBRANES.

1. Birth Injury.

Injury to the brain during labour, which is often accompanied by intracranial haemorrhage, is a frequent cause of death at or soon after birth. Although most of the children with such injuries are stillborn or die within a few hours, there are many who survive, and in later life a large proportion of these present physical or mental signs of severe and permanent brain lesions. Convulsions are among the earliest and most striking symptoms in these cases.

When convulsions arise from this cause they almost always begin within the first few days of life, and certainly a large majority of the babies who take fits before the end of the first week are suffering from some intracranial lesion of this sort.

Thirty-seven of my 200 cases of convulsions evidently belonged to this group. In more than three-quarters of these (29) the fits began on the first, second, or third day, and only in five were they deferred beyond the first week. Boys were much more often affected than girls (29 to 13).

The diagnosis of brain injury as a cause of convulsions in any case depends on the character of the labour—on whether it was premature, on the date of the onset of the symptoms, and on the child's condition at and soon after birth, especially on the occurrence of asphyxia, with difficulty in getting respiration established. The diagnosis is strongly confirmed if the fontanelle is found to be bulging soon after birth, or if the fluid obtained by lumbar puncture is blood-stained.

Much valuable light on the morbid anatomy and etiology of birth injuries to the brain may be obtained from such recent work as that of F. J. Browne¹ and Eardley Holland² on the causes of stillbirth. The subject is too large to be fully considered here, but I should like to draw your attention to some of the statistics in Dr. Browne's able and practical paper, and especially to his conclusions as to how the risk of such injuries may be lessened, or altogether prevented, by care on the part of the obstetrician.

In many circumstances the adventure of birth is inevitably a more or less perilous matter for the baby; but it seems certain that by forethought and patience, and by choosing, when possible, those methods of delivery which have been found to be safest for the child, a great deal can be done to lessen or prevent the risk of intracranial

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injury. Dr. Browne's conclusions seem to me of great practical value, and he has kindly allowed me to quote them here.

Cerebral haemorrhage was found in 29.5 per cent. of 200 cases of infants who were either stillborn or died soon after birth. The condition was ten times as common after breech as after vertex presentations. In vertex cases it was met with mainly in difficult forceps deliveries, but it was also apt to occur in ordinary easy forceps cases if the labour was hurried for any reason. It was never found in any case of full-time vertex presentation when the labour had been natural.

One of the commonest lesions found in stillborn children is tearing of the dura mater. This lesion was present in 37 per cent. of the 94 cases in which it was looked for; but in only 40 per cent. of these had it caused any haemorrhage. It was about sixteen times as liable to occur in breech as in vertex presentations; indeed, some damage to the dura mater was found in all breech cases, whether easy or difficult, full-time or premature. In vertex cases tentorial tears were found, mostly after difficult forceps deliveries, but they occurred sometimes in spontaneous deliveries, when the labour had been prolonged and moulding of the head had therefore been excessive. Tentorial tears were never found after normal vertex deliveries.

Prematurity appeared to be a serious cause of cerebral haemorrhage; and premature babies, especially those between 7 and 7½ months, seemed to be about sixteen times more likely than full-time infants to suffer from this lesion. In them the haemorrhage was not due to tears of the dural septa, but was probably to be accounted for by overfilling and bursting of the fragile cranial veins, along with decreased coagulability of the blood. It occurred about seven times as often in breech as in vertex cases. A haemorrhagic diathesis played, apparently, no important part in the causation of the bleeding.

Intraventricular haemorrhage occurred in 8.5 per cent. of the cases examined. It was found in premature cases only, and seemed to be due to the same conditions as have already been mentioned as accounting for other forms of intracranial haemorrhage in premature babies. No symptoms were noticed in these cases which could have given grounds for a diagnosis of the condition during life; and there was no reason to think that any of them could have been permanently cured by operation for removal of the clots.

It is significant that only two of Dr. Browne's cases were considered to have received adequate antenatal supervision. The points which Dr. Browne recommends to be kept in view for the prevention of cerebral haemorrhage during delivery are the following:

1. *Avoidance of breech deliveries*, by cephalic version of breech presentations at a period when this is always possible—namely, not later than the seventh or eighth month.

2. *Avoidance of Difficult Forceps Deliveries*.—This is only possible when there is careful antenatal supervision of every case.

3. *Avoidance of induction of premature labour*, certainly till after eight months; better still till after eight and a half months.

4. *Manual dilatation of the parturient canal* for the passage of the premature infant, and preservation of the membranes unruptured as long as possible.

5. Finally, after the study of a large number of cases, it was found impossible to avoid the conclusion that it was only by *adequate antenatal supervision of every pregnant woman* that the present high morbidity rate from cerebral haemorrhage could ever be seriously diminished.

Prognosis.—A serious prognosis must, of course, always be given when there is evidence of a severe birth injury to the brain having occurred. Many of these cases recover to a surprising degree; but the outlook is always anxious, even when the grave symptoms have apparently passed off completely. In several children who have seemed to make a satisfactory recovery, I have found some degree of mental defect or instability, or a tendency to epileptic seizures, in later childhood. Spastic rigidity of the limbs not infrequently develops, and this condition, even when it is very slight in degree, is likely to persist through life.

Treatment.—Early operation to remove the intracranial clots has been recommended by Cushing and other

surgeons; but it is very doubtful whether this is often of permanent benefit. Certainly, in most cases, nothing can be done beyond careful attention to the general nutrition and treatment of any symptoms that may arise.

2. Meningitis.

In seven of my cases the fits were evidently due to this cause. In the last four of them meningococci were found in the cerebro-spinal fluid, and the others were probably due to the same cause. Six of the children died and one recovered with hydrocephalus.

3. Hydrocephalus.

In four other cases internal hydrocephalus of uncertain origin was present. Two of these died in infancy and the others survived to adolescence; one of them was feeble-minded and the other idiotic.

4. Other Brain Lesions.

Other brain lesions of a more or less obscure nature were found in five cases.

5. Congenital Brain Defect with Petit Mal Attacks.

One sees many cases of this type during the earlier part of the first year of life. Eighteen of my 200 cases in very young babies belonged to it. A large majority of the patients (13) were girls. In most of the children the attacks began very insidiously, and their grave significance was generally overlooked at first by the parents, who referred to them as "turns," or "faints," or "starts."

The seizure generally consists in a sudden momentary jerk forward of the head and shoulders, with the arms extended and somewhat adducted and pronated. The child loses consciousness for a few seconds, and on coming round often cries bitterly as if in pain. After the attacks have gone on recurring for a year or two they are apt to be accompanied or replaced by regular epileptic seizures. Sometimes, though by no means always, the parents have noticed from the first that the baby has not been as bright as she should have been. Always, as she grows older, she becomes less and less intelligent, the mental deterioration being much more rapid when the fits are numerous, and some slight improvement usually occurring, during the earlier stages of the case, when they are few in number or absent for any time. The child is very late in holding up her head, if she is ever able to do so; and the cranium remains far too small.

Etiology.—The causation and pathology of this condition is, so far as I know, quite unknown. The fits are probably connected with a congenital arrest of development of the brain cells. The mental defect is due mainly to the same cause, but it is always aggravated, as already mentioned, by continuance of the attacks.

The *prognosis* is extremely bad. The children all become quite idiotic, although they may live to adolescence or even to adult life.

The *treatment* also is most unsatisfactory. Bromides and chloral are rarely even of temporary advantage. Occasionally a tonic or some regulation of the diet may cause improvement in the symptoms for a time; and now and then a feverish illness, such as measles, may have a similar effect. I have also known a temporary lessening of the number of the attacks to follow a change of climate or more open air.

B.—CEREBRAL DISTURBANCE DUE TO ACUTE DISEASE IN ORGANS OTHER THAN THE BRAIN.

1. Digestive Causes.

Indigestion of some kind accounted apparently for forty-six of my cases, and it is probable that a considerable majority of the cases of convulsions met with in young babies after the first fortnight is due to digestive derangements, either functional or organic. Fits are easily set up in infants who are debilitated by any kind of food disorder—especially by food intoxication with diarrhoea. When there is catarrh of the stomach with dilatation and retention of decomposing food, severe and repeated convulsions are common. This often occurs, for example, in cases of pyloric stenosis which are being unsuccessfully treated by medical measures.

In most cases of dyspepsia with convulsions the *treatment* consists in regulation of the diet—the "short cut" to recovery being the employment of a wet nurse.

In cases with gastric catarrh from prolonged retention of the stomach contents, washing out of the stomach at suitable intervals generally stops the fits at once.

2. Jaundice.

In two of my cases there was profound jaundice. In one of them its cause was obscure; the other was a case of congenital obliteration of the bile ducts.

3. Genito-urinary Irritation.

This is a frequent cause of severe nervous disturbance in babies, and sometimes gives rise to fits. If the urine is concentrated, highly acid and depositing uric acid crystals, as sometimes happens when a baby is taking too little fluid, high fever is not uncommon, and fits may occur. This was seen in two of my cases. In another the fits were apparently due to severe irritation from phimosis. In two cases the cause was genito-urinary infection with *Bacillus coli* (pyelitis or pyelonephritis). In infants over 6 months—especially in girls—this form of infection often causes rigors, and fits are rare. When the disease occurs in the early months of life, however, convulsions are much commoner than in older babies, and rigors rarely, if ever, occur.

4. Acute Bronchitis or Bronchopneumonia.

These accounted for six cases.

5. Choroiditis; Otitis; Heart Disease.

Choroiditis and otitis media seemed to be the causes in one case each, and congenital heart disease in two cases.

6. Unknown.

In seven cases no definite local lesion could be found, but there may probably have been some unrecognized source of reflex irritation.

C.—CEREBRAL DISTURBANCE CONNECTED WITH VARIOUS KINDS OF GENERAL INFECTION, OR WITH DEBILITY.

1. General Tuberculosis.

Fits are not uncommonly the chief symptom in the rare instances in which very young babies die of general tuberculosis. In these the infection often comes from a phthisical parent, and it usually starts in the respiratory tract. In three of my seven cases of this nature there were only a few convulsions. In the other four they were very numerous, and in this and in their other characters the symptoms somewhat closely resembled those met with in the "idiopathic" group. They were also usually checked for a time by the administration of chloral.

2. Congenital Syphilis.

In only 5 per cent. of the 200 cases did the children have evidence or a clinical history of syphilis. When we consider the large number of syphilitic babies seen in the early weeks of life and the frequency with which they are suffering from other causes of convulsions, such as severe dyspepsia and debility, it seems probable that a syphilitic taint has not in itself a very strong tendency to give rise to this symptom.

3. Debility.

In two of the cases nothing was found to account for the convulsions except extreme debility. They appeared as a terminal symptom.

4. Whooping-cough.

In the rare instances in which babies take whooping-cough in the early months of life fits are not infrequently met with. In these cases bromide has little or no effect, but the convulsions are generally stopped by the administration of chloral.

5. The Idiopathic Convulsions of Early Infancy.

The last type of convulsions to which I wish to direct your attention is one which has not received a special name, and for which no central, reflex, or other cause has as yet been discovered. We may therefore speak of these fits provisionally as "idiopathic convulsions." They generally occur during the early weeks of life; and they are not very common—for, in the last twenty-five years, I have only seen thirty-five cases in very young babies and two in rather older infants. They present, however, as we shall see, certain very interesting features, both in

connexion with their treatment and with their possible etiology. Before dealing with the clinical features in more detail, the symptoms which occur in a typical case may be described shortly as follows:

Typical Case.—The patient, who is usually a boy, has been bottle-fed, and has hitherto seemed well in every respect. He has not suffered from any severe food disorder; there has been no reason to suspect the presence of a cerebral birth injury, and his mental condition has been normal. When two or three weeks old the child begins to have slight twitchings of the face and limbs, which recur at frequent intervals. These soon develop into regular convulsive seizures of short duration. After they have gone on for a day or two, the fits become very numerous—twenty to fifty in the day—and they may continue so for weeks, if the case is not successfully treated. During the short seizures the child is unconscious, and in the intervals he is usually drowsy.

The administration of bromide, even in full doses, has little or no effect in stopping the attacks; but if chloral is given cautiously and continuously in a sufficiently large amount, the fits not only cease rapidly, but do not return when the drug is discontinued; and the child grows up perfectly healthy in mind and body. There are reasons for suspecting that, if the treatment is not carried out effectively, the child's mental condition may be permanently damaged.

Details of the Clinical Features.

Sex.—A large majority of the patients were boys (25:10).

Family History.—In five of the cases there was a history of fits, or of great nervousness, in other members of the family. Three of the children were twins.

Character of the Labour.—Satisfactory details of the confinement were only obtained in a few instances, but, so far as could be ascertained from the mothers, the birth had been normal and non-instrumental in 19 of the cases. In 12 forceps had been used, but there had been little or no difficulty in any of these. The infant had shown signs of slight asphyxia at birth in three instances. Two of the births had been premature. In four cases no details were available.

Form of the Feeding.—In two of the cases this was not noted. In two others the children were on the breast, but it was almost certain that in both of these cow's milk had also been given. In the remaining 31 cow's milk, or some food made from it, was being regularly used. In no case had any connexion been noticed between a change of food and the beginning of the symptoms.

General Condition.—In two of the cases the child's general condition was not noted. One of the infants showed marked debility; in another the symptoms began during an attack of influenza. All the other patients (31) were said to have been in good health, and several of them were thought to have been above the average in vigour when the fits began.

Digestion.—In about half of the children there had been no indications of indigestion at all. In the others, slight dyspeptic symptoms, such as flatulence, occasional vomiting, loose motions, or constipation, were remembered by the mothers, but in no case had these been severe.

Onset of Symptoms.—The date of the first sign of the convulsions varied from the fifth day—in three instances—to the end of the twelfth week. (As I have already mentioned, I have seen two cases in which similar convulsions began later—in the sixth month in both instances. I shall not, however, include them here.)

Date of Onset of the Convulsions.

During 1st week in 6 cases			During 7th week in 3 cases		
" 2nd	" 5	"	" 8th	" 2	"
" 3rd	" 7	"	" 9th	" 1 case	"
" 4th	" 4	"	" 10th	" 1	"
" 5th	" 2	"	" 11th	" 1	"
" 6th	" 2	"	" 12th	" 1	"

In most instances the earliest symptoms had consisted in a slight twitching of the face, arm, or leg, and this was usually unilateral at first; there was also some rigidity of the affected limbs in some of the children. These symptoms rapidly developed into regular short convulsions which soon became general, although they were often unilateral at first. After the first few days the character of the attacks remained the same, but their number increased

rapidly until there were often twenty to fifty, or even more, in the twenty-four hours. The period of unconsciousness accompanying the fits was very short, and the child was not noticeably cyanosed; but he generally remained somewhat drowsy during the intervals.

The *diagnosis* of this type of fits depends on the clinical features—short slight general convulsions, repeated at brief intervals, occurring in a child who has been born after a fairly natural labour, has seemed hitherto normal in body and mind, and has had no severe digestive derangement, and no other cause of reflex irritation sufficient to account for fits, and who makes a rapid and complete recovery under chloral.

Treatment.

Bromides, as already mentioned, are of no use. Chloral is very successful, provided it is given cautiously and in sufficient doses.

The chloral is administered by the mouth, and the best dose to begin with is one grain every two hours. This almost always lessens the number of the convulsions, and sometimes stops them altogether. If they still continue a grain and a half must be given, and the dose may have to be increased to two grains every two hours for a short time in children of more than a month old. This dose cannot usually, however, be given for more than a few hours, as it soon makes the child too drowsy to swallow. When the fits have ceased for a day or two days the chloral should be given at increasing intervals—three, four, and six hours—and then gradually stopped.

In the large majority of cases, if no complication occurs, the child gradually awakes, his appetite returns, and he has no more fits, though he sometimes remains more or less drowsy for weeks. In eight of the thirty-five cases, owing, apparently, to the amount of the chloral given not being sufficient, the fits returned when the drug was discontinued. Sometimes they began again at once, and sometimes not for a week or two. In all but one of these, however, when the chloral was resumed, the fits ceased and did not return.

Result of the Treatment.—This was almost always most satisfactory, provided the necessary precautions had been observed. Of the thirty-five cases, one was lost sight of at once, three died rapidly of pneumonia, and two a month or two after apparently successful treatment; in neither of these were details of the fatal symptoms obtainable. Two of the children became mentally defective. Of the remaining cases—twenty-seven in number—fifteen recovered, but after doing well for several months were lost sight of. Of the twelve others, whom I have recently seen, or of whom I have lately heard details, two died from acute infectious illness in later childhood; and ten—whose present ages vary from 13 months to 24 years—have remained quite well in body and mind. None of them, so far as I have been able to ascertain, have ever had any more fits, and none have shown any symptoms of asthma, or severe indigestion, or any sign of an idiosyncrasy to ordinary articles of diet.

Dangers of the Treatment.

There are two dangers connected with the treatment by chloral. One of these is the risk of *inhalation-pneumonia* being set up if the child is carelessly fed while deeply under the influence of the drug. While taking the doses necessary to stop the convulsions, the baby has usually some difficulty in swallowing his milk, and when the maximum doses are being given this becomes greater. Great care must therefore be used in the feeding. If the mother or nurse is warned about this the feeding can usually, without difficulty, be carried out in such a way that no harm results. If, however, little care or skill are used the choking and spluttering which occur are very apt to result in some of the milk finding its way into the lung and setting up pneumonia. This occurred in 3 of my 35 cases.

The other danger which must be guarded against is of quite a different kind, and arises when the chloral is given in too small amounts. When this is done the drug may only depress the child's vitality without preventing injury to the brain cells by the toxin of the disease, and the damage done may permanently injure the child's mental condition. It can scarcely be claimed that this is proved by the experience of the two following cases, but it is at least a probable theory to account for the mental deterioration which resulted in them.

One of these children, who was a typical and severe example of the disease, did well at first in the usual way on the usual doses, and the fits stopped. The medicine was then promptly discontinued, whereupon there was a return of the convulsions, and they again became very numerous. Full doses were again ordered, but the fits continued to recur frequently and severely, and before long it was evident that the child's mental condition was gravely affected. After this no improvement took place, and the infant rapidly became quite idiotic, and remained so till she died some years later. The failure of the treatment in this case may have been due simply to its having been one of unusual severity from the beginning. It was, however, discovered after it was too late that, at the most important stage of the case, the nurse, owing to some misunderstanding, had been giving less than a third of the dose of chloral that was ordered.

In another well marked case, which was not so severe as this one, it was suspected that the full amount of the medicine ordered had not been given. The convulsions, however, stopped after a time, and the baby was reported to have made a good recovery. When I saw him again, seven or eight years later, he was found to be an imbecile of a mild grade, and he is now in an institution for the mentally defective.

Etiology.

The causation of these cases is extremely obscure; but it certainly looks as if the convulsions were set up by the action on the brain cells of some sort of poison formed in the body. The most remarkable fact about the attacks is that in a large majority of the cases they are *permanently* stopped by a merely *temporary* chloralization of the patient. Any attempt to solve the problem of their causation must take this striking fact into consideration.

A search for some analogous phenomena which might possibly help to explain the action of the drug in these cases has only led to the discovery of one fact which seems to have a bearing on the question. This is the observation, described by C. Richet,³ Besredka,⁴ and others, that when a guinea-pig is put deeply under ether, alcohol, chloral, or certain other narcotics, before the second dose of a serum which is usually rapidly fatal, the expected anaphylactic shock is generally suppressed altogether, and after a period of unconsciousness the animal "awakes vaccinated," as Richet expresses it.

This phenomenon has been repeatedly observed by later workers, but I have not been able to find any printed statement as to how long the effect of the "vaccination" which follows has been found to continue. We must look to the laboratory workers to clear up the difficulties of the subject; certainly, as Besredka says, it seems to be "pregnant with possibilities."

There can be little doubt that the very interesting and as yet only partially understood process of anaphylaxis plays an essential part in the causation of some, and perhaps of many, morbid phenomena—especially in early life. There is, however, reason to suspect that too many symptoms are sometimes attributed to it. It therefore becomes those of us who have had no practical laboratory experience in such matters to speak of them with caution and diffidence. Perhaps, however, we may be allowed a little mild theorizing. The result of these experiments in guinea-pigs certainly suggests the question whether the clinical phenomena of this particular type of convulsions may not possibly be due to a peculiarly modified form of anaphylaxis, or some similar process, set up by the ingested cow's milk. While we do not as yet possess facts enough to enable us to answer this question in the affirmative, it may, I think, be fairly claimed that we have enough information to justify our saying that this is at least the most hopeful direction in which to look for an explanation of the facts.

Two somewhat different clinical types of severe acute poisoning from cow's milk, which apparently depend on anaphylaxis, may be met with in young infants, though fortunately they are both *extremely* rare. Of the most characteristic and least common of these, I have only had to do with one case.

*CASE.**

The patient was a female infant whose mother and sister were subject to similar symptoms. The mother, though otherwise healthy, had frequently suffered, during the four years

*I am indebted to Dr. R. A. J. Harper for the notes of this case.

before this baby's birth, from attacks of urticaria, local oedema, and hay fever after eating acid fruits or bananas, and had also once had severe asthmatic symptoms. One sister was subject to urticaria after eating bananas, and had had an attack of acute rheumatism. The other two sisters had shown no sign of idiosyncrasy to food of any kind.

During the first few months of life the baby, who was on the breast, had on several occasions had a meal of cow's milk without any bad effect. When seven months old she was given a little rice pudding with cow's milk, which was followed at once by urticaria and asthma. From that time on she had violent symptoms whenever she was given cow's or goat's milk in any form or raw white of egg. Half a teaspoonful of milk was sufficient to bring on an attack within a few minutes. The breathing became difficult and there was sneezing and coughing, with running of the eyes, rhonchi in the chest, and an urticarial rash. The baby seemed seriously ill and collapsed, and she remained so for two or three days. Once while she was on the breast she developed similar symptoms after her mother had eaten a banana, although the mother herself on that occasion was not affected. Cow's milk diluted in various ways, Glaxo, Cow and Gate milk, Nestlé's, Allenbury's, Benger's and other "infant foods," and also goat's milk, were all found to affect the child in a similar way. Peptonized milk did so also, but to a rather less severe degree. Raw white of egg caused the same symptoms, but the child could take boiled and fried eggs with impunity. With raw egg albumin she had also had diarrhoea and vomiting.

The baby was small but apparently healthy, and showed no physical signs of disease. She was fed, with some difficulty, on patent groats with water, boiled eggs, potatoes and other vegetables, fish, chicken, minced beef, gravy, and so on. When about eighteen months old she began to tolerate small quantities of cow's milk, and she has since, I understand, gradually improved.

The other type of case, though not often met with, is less uncommon, and many instances of it have been described.⁵ The symptoms may set in within fifteen minutes, but are frequently delayed for one to five hours after the milk has been taken. They may be caused by even a few drops of it. The infant turns pale, drowsy, and uncomfortable, refuses food, vomits and has diarrhoea, with some degree of fever, or sometimes a subnormal temperature and collapse; and occasionally there is glycosuria or albuminuria. In these cases there is generally no sign of urticaria or of respiratory distress. The child rapidly recovers when put on breast milk only. Often the idiosyncrasy does not show itself for some weeks after birth; and in many cases it diminishes or ceases in the course of a few months, although in others it may last into later childhood. If the children are carefully looked after the condition is rarely fatal.

It is to be noted that none of my cases of "idiopathic convulsions," so far as I have been able to ascertain, has shown any subsequent tendency to convulsions, to food idiosyncrasy, to asthma, or to skin eruptions. Should it be shown that the above hypothetical explanation of these cases is possibly correct, the further question naturally arises as to whether, when symptoms of poisoning from cow's milk, white of egg, oatmeal, or other common foods occur in young children, they might not be stopped permanently if the patient were subjected to a thorough chloralozation for some days, while continuing the offending diet; and even whether a tendency to asthma and eczema beginning in infancy might not be permanently arrested in a similar way.

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AN international congress for protection of mothers and infants will be held in Paris from July 6th to July 8th, 1922, when the following subjects will be discussed: (1) Protection of mothers before and during confinement; (2) protection of mothers during lactation; (3) protection of the infant separated from its mother; (4) assistance and instruction. Applications for membership should be made to La Ligue contre la Mortalité Infantile, 49, Rue de Miromesnil, Paris.

BILATERAL RIGIDITY IN MIDDLE MENINGEAL HAEMORRHAGE.

BY

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THE clinical picture presented by middle meningeal haemorrhage has received during the past forty years the attention of many skilled observers. Their labours have been so fruitful that in its most typical form the middle meningeal syndrome has the distinction of being one of the most clearly defined in the whole range of medicine. As is usually the case when the picture of a settled type of case becomes clear and easily recognized, the surgeon and the physician have an opportunity of noting with greater profit the variations from type which individual cases present. The frequency of the "free" or "lucid interval," the reasons for its absence, the occurrence and significance of ocular palsies and deviations, of paralyzes of the limbs, of aphasia, and the incidence of Jacksonian fits, have all received their measure of investigation and have been allotted definite values.

Two cases recently under my observation presented certain features to which attention has not hitherto been specifically drawn in this connexion; in place of monoplegia or hemiplegia there was a very pronounced degree of general muscular rigidity. Here and there in the literature of middle meningeal haemorrhage a case is recorded in which it was noted that the limbs were rigid.

A bare half-dozen are to be found amongst Wiesmann's collection of 257 cases.³ Further examples occur in Duret's comprehensive monograph,⁴ but these include severe fractures of the skull, with cerebral laceration and intradural haemorrhage. These references are so incomplete that one gets only a hazy impression of the precise nature of the condition; the authors have simply recorded the fact in a few words without further comment.

In the histories which follow I have attempted to give a fuller account of the appearance and posture of the patient. The muscular fixation was seen to involve the trunk and all four limbs, and was such that passive alteration of position was difficult, and at times impossible, without the employment of considerable force. The clinical picture is so striking that, once seen, it is not likely to be forgotten. It seems to me to be important that attention should be directed to these generalized rigidities, and that the processes attending them should be accurately observed. Kinnier Wilson¹ has recently described a similar rigidity appearing in pathological circumstances other than those here described, so that one cannot say that the essential rigidity seen in my own two cases is in any way pathognomonic of middle meningeal haemorrhage. It is, nevertheless, important to note that hypertonicity and a peculiar attitude of body have been observed in these cases of meningeal haemorrhage no less than in the cases described by Wilson, since the one throws a ray of light, if an oblique one, upon the other.

CASE I.—Fall on Head: Linear Fracture in Left Temporal Fossa: Middle Meningeal Haemorrhage: Bilateral Rigidity of Trunk and Limbs: Operation: Death: Necropsy.

On March 25th, 1921, J. D., male, aged 37, a window cleaner by trade, was at work cleaning some first story windows in a stone courtyard. He lost his balance and fell head foremost to the ground. Being temporarily stunned he lay for a minute or two and then got up and walked home, a distance of a couple of hundred yards. He sat down for a little while, telling his wife that he felt rather shaken, but soon declared that he would go and fetch his ladder and pail. This he did, but on arriving home a second time seemed decidedly worse, and went and laid himself down. He went to sleep, but when his wife tried to rouse him some little time later she found it impossible to awaken him. From this time (perhaps one and a half hours after the accident) onwards he remained unconscious. Later in the evening he was admitted to the Salford Royal Hospital under my colleague Mr. Garnett Wright, who kindly asked me to see the case. On admission he was unconscious, restless, moving arms and legs freely, and inclined to be noisy.

Symptoms and Physical Signs.

When seen by me, seven hours after the accident, the picture had changed. He lay flat upon his back, the arms rigid and extended by the sides. The hands were half-clenched and the forearms hyperpronated, so that the backs of the hands were facing the outer aspects of the thighs. The legs were stiffly extended and the toes pointed downwards, the ankles being