PREVENTION OF INTRACRANIAL INJURIES OF THE NEWBORN

BY

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During a period of six months I performed necropsies on 107 consecutive natal and neo-natal deaths at the above hospital. Excluding fourteen of these cases which had perforations performed for varying causes, forty-three of the remaining ninety-three cases were found to have intracranial lesions—that is, in 46 per cent. of all these consecutive deaths there were intracranial injuries. The lesions present were lacerations of the tentorium cerebelli in most cases, and usually associated with haemorrhages or oedema of adjacent parts; occasionally the falx cerebri was also lacerated.

There is not the slightest doubt that a large proportion of infants who survive have intracranial injuries; the greater number of these will have no more than small tears of the tentorium and oedema of adjacent parts, but others may survive where relatively large haemorrhages have occurred, and I have seen a great amount of blood removed in two cases where I performed repeated bi-daily cisternal punctures for four days and the infants at the time of discharge appeared to be normal. Although the presence of these injuries in the newborn has been recognized as far back as 1829, their tremendous importance as regards infantile mortality has not been stressed until the last few years, and even now some modern obstetrical textbooks do not seem to appreciate their great importance.

The purpose of this short article is not to determine the treatment of these injuries, but to find methods by which these injuries may be prevented.

Mechanical Causes of Injury

Numerous theories have been brought forward as to the method of production of these injuries, but it is more commonly agreed now that a mechanical factor is the direct cause of the injury, due to alteration in shape and compression of the foetal head. This is accompanied by stretching of the supportive apparatus, particularly the tentorium cerebelli. This mechanical effect of alteration in shape and compression is most marked in cases where there is excessive moulding, or where the change in shape of the foetal skull is accomplished too quickly by an operative delivery.

If there is excessive moulding, the effect of lateral compression is to raise the vault of the cranium, so increasing the strain on the falx and tentorium. The prevention of this excessive moulding can be carried out only by efficient ante-natal care in watching for disproportion between size of foetal head compared to maternal pelvis, and performing induction of labour before term or, if necessary, Caesarean section at term. This is one big factor in preventing intracranial injuries.

Breech Deliveries

Another extremely common cause of these injuries is in extraction of the after-coming head in breech deliveries. Bourne in 1922 found that breech delivery is ten times more likely to give rise to cerebral injuries as compared with vertex deliveries. In my series every breech mortality (six) had, at necropsy, intracranial injuries. To prevent these injuries in breech deliveries it is most important that the still unmodelled head should not be drawn violently through the pelvis and so cause sudden compression and, in this way, rapid stretching of the cranial supports. Also the routine procedure of performing a

lateral episiotomy in every case of breech delivery is a great help in preventing the sudden application of, and then sudden release of, compression on the foetal head due to an unstretched perineum.

Again, in breech deliveries care should be exercised in applying suprapubic pressure on the after-coming head, especially as this pressure is applied to the foetal skull in a vertical direction, and so is more liable to produce stretching of the cranial supports. Some obstetricians favour the routine application of forceps on the after-coming head as opposed to applying any suprapubic pressure on the head, thus avoiding pressure in a more dangerous direction.

Misuse of Forceps

As regards the use of forceps, these alone should not increase the risk of intracranial injuries unless associated with other factors as marked disproportion, or the wrong application of forceps, especially a fronto-occipital application. If forceps are being used, it is advisable to use axis traction, even in low forceps deliveries, to avoid premature extension of the head, which causes pressure of the occiput against the pubic arch with increased possibilities of intracranial injuries.

An unexpected cause of injury may result from the use of forceps of wrong size. One well-known firm sent to me a new pair of axis traction forceps with the widest diameter between the blades of exactly $2\frac{1}{2}$ inches instead of the usual $3\frac{1}{2}$ inches. Pulling on the forceps with this ridiculously small diameter would certainly cause intracranial injury, so it is a wise plan to check the separation of the blades from time to time, and also the shape of them, as after much use it is surprising how they can alter their correct shape.

Other Causes of Intracranial Injury

Another common cause of intracranial injury is the use of pituitrin in a case with incompletely dilated cervix or with a rigid perineum. The use of pituitrin in these cases may quickly force the foetal head through this obstruction, with resultant cranial injury. Again, precipitate labours are a frequent cause of similar damage, as the sudden compression followed by sudden release results in injury.

In premature labours one must be exceedingly careful in the delivery of these cases, as premature infants are notoriously prone to develop intracranial injuries, no doubt due to abnormal fragility of the vessel walls, and of the dura, etc., due to incomplete development of fibres of dura.

In delivering a normal case in the interval between uterine contractions, with the head on the perineum, one must be careful not to push too forcibly the foetal forehead against the pubic arch by a finger on the perineum or in the rectum, as such pressure may be enough, especially in a premature infant, to injure the supportive apparatus.

An indirect method in the prevention of these injuries is applicable in those cases with an irregularity or change in rate of foetal heart sounds. Usually these changes are due to compression on the foetal skull, and the treatment is not to apply forceps and deliver as rapidly as possible, and so increase the likelihood of intracranial injury, but to use chloroform, which will lessen the uterine contractions.

Efforts at Resuscitation

The last important point in the prevention of these injuries, or in this case I should say in preventing the increase of intracranial damage, is the manipulations during resuscitation. It is most important to remember

that practically all asphyxiated newborn infants which do not respond to treatment within the first minute or two are suffering from intracranial injuries, and that the older and violent methods of resuscitation, if used, will further increase these injuries by causing increased haemorrhage. If an asphyxiated infant is going to respond to any form of resuscitation, I am sure no more treatment is needed than to remove any mucus from the throat, and to encourage attempts at respiration by gentle traction on the tongue with tongue forceps at the rate of about twenty pulls per minute, together with the injection of a cardio-respiratory stimulant such as coramine or icorol.

Should the infant attempt to breathe, carbon dioxide is here of some use. The older methods of throwing the child from one hand to the other or of violently flexing its body should never be allowed to be used on an asphyxiated newborn infant, as all such infants should be considered as having potential brain injuries, and small haemorrhages may soon prove fatal if unnecessarily aggravated by vigorous manipulations during resuscitation.

RECURRENT VOMITING ATTACKS IN CHILDHOOD

WITH SPECIAL REFERENCE TO ALLERGIC FACTORS *

BY

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Recurrent attacks of vomiting, or so-called "bilious attacks," occur with sufficient frequency in childhood to be of real importance in clinical practice. Though not usually causing prolonged illness, they are often most inconvenient to the children concerned on account of their repeated interference with school life, while they may at times be severe enough to necessitate admission to hospital.

Wyllie and Schlesinger¹ have recently discussed a group of cases of this type under the heading of "The Periodic Group of Disorders in Childhood." Such a title has certain advantages, the chief being the emphasis it lays upon the tendency of these attacks to recur. "Cyclic vomiting" is a term often used for the attacks, but this rather implies a regularly recurring cycle, and any absolutely regular periodicity of the attacks is actually very rare, if indeed it ever does occur. Again, these children are often said to be suffering from attacks of acidosis. This is mentioned in order to draw attention to the fact that there is usually no justification whatever for such a diagnosis, although acidosis may accompany severe attacks of vomiting. It is of course well known that at the time of attacks these children do suffer from varying grades of ketosis. The term "ketosis" is, however, by no means synonymous with acidosis, and in fact true acidosis is not usually met with in childhood except as a relatively rare accompaniment of such diseases as diabetes, nephritis, and acute gastro-enteritis.

The aetiology of the recurrent attacks of vomiting under discussion is by no means clear. They are probably a manifestation of some metabolic disturbance, and Josephs² has shown that they may be associated with a hypoglycaemia. This being so, the accompanying ketosis is therefore explicable on the basis of an insufficient supply of carbohydrates being available for the proper metabolizing of fats. In these patients the clinical improvement effected by restricting fats and giving glucose is evidence favouring this explanation. It should, however, be mentioned that not all cases show a hypoglycaemia, and that

the theory of hypoglycaemia as an important aetiological factor has not been definitely established. Whichever is primary, the ketosis or the vomiting, the fact remains that between attacks these children are generally healthy, with, so far as one can judge, no evidence of ketosis or of any other metabolic disturbance, and one is still ignorant as to the exact factor which initiates an attack. It has been shown that some at least of the children can tolerate a definite ketosis produced artificially without an attack being precipitated.

It is recognized that gastro-intestinal symptoms may be a manifestation of allergy, and Rowe³ regards gastro-intestinal food allergy as occurring most frequently in infancy and childhood. It appears quite possible, therefore, that allergy may play a part in the production of these recurrent attacks of vomiting, and that sensitivity to some foodstuff may be a determining factor in their onset. With this aspect of the problem particularly in view, the following study was undertaken.

Investigation of Forty Cases

So far as possible, all children were selected for study who came to my out-patient sessions in the Children's Department of the London Hospital between January, 1932, and August, 1933, suffering from recurrent vomiting attacks. Personal and family histories were investigated, all significant data in regard to clinical findings were recorded, various investigations were carried out, treatment was prescribed, and progress was followed in each case. An attempt was then made to correlate the findings in the entire series, and to draw conclusions therefrom. As in every case reliance had to be placed on the history obtained from the mother and upon her account of subsequent progress, it was obvious that accuracy in certain of the data was open to question. This, however, is invariably the case in any clinical study of this nature involving observation of out-patients.

The series here presented consists of forty cases; as is inevitable, certain other patients who were originally included defaulted before adequate observations could be carried out, and had therefore to be omitted. Of this series all the children attended quite regularly except three, each of whom for some time failed to come up as requested. The maximum period of observation was twenty-seven months and the minimum five months. The average period of attendance during which observations were made was sixteen and a half months; only two children were observed for less than six months. Reports of progress were subsequently obtained in regard to four of the children after attendance had lapsed.

Character of Attacks

It soon became apparent that there was no absolute uniformity in the duration of individual attacks, and, generally speaking, no regular periodicity in their occurrence. Often a child who was, at the time of first attendance, said to have an attack every two or three weeks, was found thereafter to have such attacks only at very infrequent intervals. Even in the history given by the mothers any account of regularity of attacks was infrequent. It is really impossible to make any useful analysis of the frequency or duration of these attacks. In most cases the vomiting was accompanied by headache, which was sometimes the most outstanding feature of the attack. This will be discussed later.

The average age at the time the children were first examined was just under seven and a half years. In twelve cases the attacks were said to have commenced before the age of three years, in thirteen cases the onset apparently occurred between the ages of three and five years, and in fifteen cases between the ages of five and ten years.

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