

BIRTH TRAUMA IN NEWBORN—AN AUTOPSY STUDY

by

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Introduction

In cases of difficult delivery the head is more often injured than other regions and severe intracranial lesions are liable to cause death as a result of pressure exerted by extravasated blood on the medulla. Only a minority of infants with symptoms and signs attributed to cerebral injury die. Frank haemorrhage is relatively rare, and only about a quarter of the fatal cases with the so called 'Cerebral' features turn out to have post-mortem evidence of macroscopical brain trauma. (Potter, 1962; Clairaux, 1958). The injury in other cases, and probably in many who recover, is caused by anoxia and cerebral oedema. A serious lesion is the fracture of one or more of the skull bones. The fracture may result in disruption of underlying dural sinuses with massive extravasation of blood. The most common intracranial lesion is laceration of tentorium cerebelli extending antero-medially and involving the unsupported straight sinus or rupturing the tributaries of the great vein of Galen (Stowens,

1959). Haemorrhage in the subarachnoid space is usually anoxic in origin. Intracranial haemorrhage in premature infants is probably related to the physical character of the brain substance. The soft parenchyma gives relatively little support to the blood vessels and therefore they are more exposed to the effects of trauma. The same degree of trauma might have little or no influence on the vessels of a full term infant (Stowens, 1959).

Much of the variation in the published statistics of incidence is due to differing assessments of the importance of small subdural and subarachnoid haemorrhages. Confusion as to prognosis and late complications is inevitable, because of the uncertainty which always exists clinically regarding the occurrence, extent and site of haemorrhages. The widely different estimates of fatal birth injuries are difficult to compare and possible comparisons serve little purpose (Morison, 1970). The better the obstetric care taken, the less likely are disasters of this kind to arise. This contention is well supported by the observations of Kurilecz and Down (1966), who record a dramatic fall in the incidence of their studies. The present study describes the incidence and clinicopathological correlation in this group and emphasises the need for nicety of clinical

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judgement regarding the obstetric procedures to be adopted.

Material and Methods

Autopsies were performed on 165 newborn infants dying in perinatal period at the Medical College Hospital, Aurangabad, during the period of two years from March 1970 to April 1972. Detailed obstetric histories were recorded in all these cases and complete autopsy was performed. Birth trauma was considered as the cause of death in cases where there was haemorrhage within the cranial cavity accompanied by evidence of damage to intracranial structures.

Results

Birth injuries accounted for 11 (6.6 per cent) deaths, out of these 8 were mature and 3 premature. Two were stillborn and 9 died few minutes to 14 hours after birth. In 9 cases there were obstetric complications preventing normal delivery and necessitating manipulations resulting in injuries to dura and the dural sinuses, leading to gross intracranial haemorrhage. The obstetric complications in these cases were breech delivery, forceps delivery, internal podalic version, prolonged labour, face presentation and spontaneous delivery. At autopsy other organs showed evidence of anoxia. In all the 11 cases injuries were intracranial, in 7 cases haemorrhage was in the posterior cranial fossa associated with unilateral or bilateral tentorial tears and in 4 cases it was subdural, limited to the frontal and parietal regions. Out of the 7 cases of tentorial tear, 3 were bilateral and 4 unilateral and in all blood and large blood clots were found in the posterior cranial fossa. In four cases source of subdural haemorrhage could not be traced due to

extreme softness and partial autolysis of the brain. One out of these 4 was mature and associated with obstetric complications mentioned above, and 3 were premature with history of normal delivery. In the latter sudden expulsion of foetus and/or the physical property of brain (extreme softness) was probably the cause of subdural haemorrhage.

Discussion

Birth trauma was responsible for causing 6.6 per cent of deaths in the form of intracranial haemorrhage. This incidence is comparable to the corresponding figures published in recent times from other centres for example Clairaux (1958)—10 per cent, Bound *et al.* (1956)—11 per cent., Kurilecz and Down (1966)—4.6 per cent. Nevertheless, there is some evidence that birth trauma is declining in its importance as a cause of perinatal deaths (Bound *et al.* 1956). A sharp fall in the incidence of intracranial haemorrhage in the past few years has been due to tremendous progress in the obstetric care, anaesthesiology and resuscitative measures. This contention is well supported by observations of Kurilecz and Down (1966) who recorded a fall in incidence from 4.6 per cent to 2.6 per cent in a 6 year period. The same has also been demonstrated by Potter (1962). Cruickshank (1930) found gross intracranial haemorrhage at autopsy in 20 per cent of 800 live born infants and 65 per cent of these cases died in the first 3 days of life. At the Chicago-Lying-In Hospital, the total mortality rate from birth trauma was 5.6 per 100 births from 1931 to 1941 and fell to only 0.5 per 1000 births from 1947 to 1949 (Stowens 1959).

In the present study a large majority (81.8 per cent) of the cases of birth trauma were associated with difficult

labour. Similar observations were recorded by Clairaux (1958). Bound *et al*, (1956), Mehdi *et al*, (1961), who suggest that birth trauma chiefly involves babies in whom obstetric complications prevent normal delivery. Analysis of the postnatal age showed that about 82 per cent of the deaths due to birth trauma occurred between few minutes to 14 hours after birth, it seems, in fact as Bound *et al*, (1956) have suggested, that birth trauma (cerebral) by itself is not an important cause of intrauterine death. These traumatic lesions may be considered as they affect the bony capsule of the skull, the supporting membranes of the brain and the brain itself. Emphasis must fall on haemorrhage and especially on subdural haemorrhage, and on the need for a clear appreciation of the site and extent of the haemorrhage.

The most common intracranial lesion was laceration of tentorium cerebelli in 63.6 per cent of cases of birth trauma in the present study. During delivery the greatest stress falls on the tentorium in the areas just below its junction with the falx anteriorly and this is by far the most usual site of tentorial tears. Laceration of the tentorium cerebelli was reported to be the most common intracranial lesion in neonatal deaths by Clairaux (1958), Mehdi *et al*, (1961), Bound *et al*, (1956). In a number of cases tributary veins passing from the brain to the dural sinuses may be damaged if the dura is torn. The vessels are usually ruptured near their point of entry in the sinuses and the haemorrhage caused thereby is frequently profuse. The vessels themselves collapse after injury and the actual site of the bleeding may be very difficult to identify (Clairaux, 1958). More often no source of haemorrhages may be seen (Potter, 1962). This may explain the absence of any

obvious source of subdural haemorrhage in about 36 per cent of the cases in the present study. Subdural haemorrhage was found in the premature babies even though the obstetric history was normal. Such observations were also reported by Bound *et al*, (1956), Stowens (1959), Mehdi *et al*, (1961). This is probably due to the physical character of the brain substance. The soft paranchyma gives relatively little support to the blood vessels and therefore they are more exposed to the effects of trauma. The same degree of trauma might have little or no influence on the vessels of a full term infant (Stowens, 1959). In any discussion of the significance of trauma in prematurity it is very essential to distinguish intracranial haemorrhage due to trauma from that due to anoxia. Traumatic intracranial haemorrhage does occur in premature infants. However, such haemorrhage is only rarely associated with extensive tears of the tentorium or falx, and comes from tears of bridging veins and sometimes from tears of the vein of Galen and cerebral veins. Not only are the haemorrhages often smaller, but it is often more difficult to decide whether they, or an associated anoxia, have been responsible for death. The data presented by Haller *et al*, (1956) provides strong support that in the premature infant subdural haemorrhages, where the role of trauma is great, are relatively fewer than subarachnoid, intraventricular and cerebral haemorrhages, to which asphyxia makes an increasingly important contribution.

Evidence of anoxia was found at autopsy in almost 80 per cent of cases of intracranial haemorrhage. This may be explained on the basis of obstetric complications associated in these cases. If there is a delay in the 2nd stage of labour, the foetus may become distressed and the

cerebral veins engorged. The distended sinuses are poorly equipped to withstand a sudden strain during delivery and consequently give way.

It is quite obvious that there is need for nicety of clinical judgement regarding the procedures to be adopted. The state of the foetus in various stages of distress must be given due consideration before restoring to heroic methods of delivery and subjecting it to further stress.

Summary

Birth trauma was responsible for causing 6.6 per cent of perinatal deaths in the form of intracranial haemorrhage at Medical College Hospital, Aurangabad. The clinico-pathological correlation in this group emphasises the need for nicety of clinical judgement regarding the obstetric procedures to be adopted.

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