ANOXIA AS A MAJOR CAUSE OF PERINATAL MORTALITY— AN AUTOPSY STUDY

by

ZAKIA SULTANA,* M.D.,
V. H. TALIB,** M.D.,
S. D. PATIL,*** M.D.

and

K. D. SHARMA,**** M.D., M.Sc. (Med.)

Introduction

Anoxia is a commonest cause of neonatal deaths. If carried to the extreme, all foetal deaths might be regarded as the result of anoxia (Clairaux, 1958). By anoxia is meant interference of oxygen supply to the foetus. The fault which results in a deficient oxygen supply reaching the foetus may be in the mother, in the placenta, or in the umbilical cord (Potter, 1952; Bound *et al*, 1956; Clairaux, 1958; Mehdi *et al*, 1961).

Evidence of anoxia was summarized by Potter (1952). In practice it is found to take two main forms (1) petechiae or ecchymosis on visceral surfaces and serous membranes, (2) massive inhalation of amniotic contents.

Whether or not the lesions are prominent in the foetus depends largely on the manner in which the oxygen supply is interrupted (Clairaux, 1958). A complete cessation of circulation through the umbilical cord may lead to death of the foetus without pathological signs being visible at necropsy. On the other hand, foetal anoxia resulting from premature separation of the placenta does produce characteristic lesions. The latter probably result from anoxic damage to the capillary endothelium and take the form of petecheal haemorrhage (Clairaux, 1958). Severe congestion of the liver spleen, kidneys, adrenals and brain is almost always found in cases of deaths due to anoxia. Large haemorrhages also occur in adrenal medulla, brain, and beneath the hepatic capsule (Potter, 1952; Clairaux, 1958; Stowens, 1959; Mehdi *et al*, 1961; Maheshwari *et al*, 1971).

The studies showing a correlation between some abnormalities of pregnancy and labour and maternal factor and the cause of death of the infant are few (Bound *et al*, 1956; Mehdi *et al*, 1961; Ghosh *et al*, 1971; Shielakaran *et al*, 1972). However, the analysis of obstetric and maternal causes leading to anoxia deaths reveals that a large majority are associated with trauma and stress of labour, toxaemia, antepartum haemorrhage, maternal diseases, such as congestive cardiac failure, anaemia, pyrexia, syphilis; and diabetes (Potter, 1952; Bound *et al*, 1956; Mehdi *et al*, 1961; Ghosh *et al*, 1971). Conditions of the umbilical cord like prolapse of cord, cord round the neck and true knots in the cord are often designated as

*Lecturer.
**Reader.
***Reader.
****Professor.
Department of Pathology and Bacteriology, Medical College, Aurangabad (Maharashtra).

causal factors in intrapartum deaths (Bound *et al*, 1956). In the etiology large number of maternal factors play their part but the common denomination is probably, abnormal prolongation of the normal state of anoxia present during labour or interference with the supply of oxygen and removal of carbon dioxide and metabolites during parturition. In the present study anoxia was found to be the major cause of perinatal mortality. The purpose of this paper is to correlate the maternal and obstetric conditions with perinatal deaths ascribed to anoxia.

Materials 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. Diagnosis of birth anoxia was confined to those cases in which there was a definite clinical history consistent with anoxia and there was no other pathological finding which would have made the inclusion of these cases possible in some other group.

Results

The lesions attributable to anoxia accounted for 40 or 24.1 per cent of perinatal deaths (stillbirths 32, and neonatal death 8). Eight were mature and 32 were premature. On analysing the obstetric history of this group it was found that a large majority—24 were associated with trauma and stress of labour (Shoulder presentation 2., breech presentation 4., brow presentation 4., face presentation 1., compound presentation 3., cord prolapse 2., true knot 1., tight cord round the neck 1., prolonged labour 5., uterine inertia, 1). Out of the remaining cases, 11 were associated with antepartum haemorrhage and 5 with toxaemia of pregnancy. The gross and microscopic changes observed at autopsy varied with the speed of onset of anoxia. The morbid anatomical changes were minimal in anoxia of sudden onset and marked and widespread in cases of anoxia of gradual onset.

Anoxia of Sudden Onset: Out of the 5 cases falling under this category, 2 had obstetric history of cord prolapse, in 1 case true knot, and in 1 case tight cord round the neck were observed at birth. In one case there was history of arrest of the aftercoming head during breech delivery. All of these five were still births. At autopsy all appeared pale with air passages clear, the lungs on naked eye examination showed few petecheal haemorrhages. Pieces of lungs from all the cases sank in water. Other organs on gross examination showed slight or no congestion in all these cases. Microscopic examination of the tissues revealed collapsed lung tissue, but the alveoli were lined by flattened epithelium. There was mild congestion of pulmonary capillaries, but no evidence of aspiration of liquor amnii or inflammatory reaction was seen. Microscopic examination of other organs revealed slight congestion only.

Anoxia of Gradual Onset: In this group there were 35 perinatal deaths, out of which 27 were stillbirths and 8 died within few minutes to 6 hours after birth, 16 or 44 per cent of these were premature. From external appearance a large majority were blue but few appeared pale. In some the body was meconium stained. On opening the body, serous membranes showed petecheal haemorrhages in almost all the cases. The air

passages in majority of the cases were clear but in 5 cases plugs of meconium and meconium stained liquor were found in the trachea. In 30 cases lungs showed complete atelectasis and pieces of lungs in all these sank in water. In the remaining 5 cases, atelectasis was partial. Other organs showed varying degrees of congestion and petecheal haemorrhages, especially in the thymus. Subcapsular haematoma was found in 3 cases in the liver. In most of the cases brain was very much congested. The severe cerebral congestion in 4 cases was associated with subarachnoid haemorrhages. Massive intra-ventricular haemorrhage was found in two premature infants in this group, one was stillborn and the other died few minutes after birth.

Microscopic examination in this group showed intense congestion of the lungs in almost all the cases. The alveoli were unexpanded in 27, but in 8 variable degrees of expansion and atelectasis were observed. In all these cases alveoli were fully mature and lined by flattened epithelium. Lungs from 8 cases showed small amounts of amniotic sac contents in alveolar ducts and bronchi, all of these 8 were stillborn. Microscopic examination of other organs showed intense congestion in all and punctate haemorrhage in majority of the cases. Liver, kidney and myocardium in 21 cases showed cloudy change. In all the cases the brain was very much congested and small perivascular haemorrhage were present in the cortex.

Discussion

Anoxia accounted for 24.1 per cent of the perinatal deaths (32 stillbirths and 8 neonatal deaths). The incidence is comparable (Table 1) to that reported by Mehdi *et al*, (1961), Bound *et al*, (1956), Rathnawathi and Reddy (1953), Maheshwari *et al*, (1971). MacGregor (1939) and Hadley *et al*, (1958) put anoxia as the most common cause in perinatal period.

Analysis of obstetric and maternal causes leading to anoxia deaths reveals that a large majority are associated with trauma and stress of labour (24 cases—60 per cent); toxaemia of pregnancy (5 cases—12.5 per cent) and antepartum haemorrhage (11 cases—27.5 per cent); such association has also been reported by Bound *et al*, (1956); Mehdi *et al*,

TABLE I

Comparison of Incidence of Anoxia in Perinatal Deaths Reported by Some of the Authors

Authors	No. of cases studied	Percentage of deaths due to anoxia
1. Rathnawathi & Reddy (1953)	162	29.0
2. Bound et al, (1956)	337	27.0
3. Clairaux (1958)	525	45.6 (stillbirths)
	365	15.0 (neonates)
4. Mehdi et al, (1961)	400	26.7
5. Maheshwari et al, (1971)	250	45.0 (stillbirths)
		22.0 (neonates)
6. Ghosh et al, (1971)	375	40.8
7. Shiela Karan et al, (1972)	100	46.0
8. Present study	165	24.1

(1961); Ghosh *et al,* (1971); Shiela Karan *et al,* (1972). The group trauma and stress of labour includes obstetric complications such as shoulder presentation, face presentation, breech presentation, cord prolapse, true knot of the cord, cord round the neck, compound presentation, prolonged labour, uterine inertia and obstructed labour, all of which may prove traumatic to the baby. The incidence of stress and trauma of labour as a maternal and obstetric complication was higher in mature babies of intrapartum anoxia group, but not in the corresponding premature group, indicating that mature babies are more prone to anoxia during complicated delivery than the prematures under the same obstetric conditions. Similar observation was recorded by Bound *et al,* (1956).

The incidence of antepartum haemorrhage is significantly high in anoxia group and also in maceration group. The nature of the bleeding showed a marked difference in the two groups. In the anoxia group it was scanty and extended over a period of days or weeks. This is in accordance to the observations of Bound *et al,* (1956). Toxaemia of pregnancy is a known cause of premature labour, but once born, the toxaemic premature baby is no more at risk than the premature baby born of a non-toxaemic mother (Bound *et al,* 1956). All of the 5 babies of anoxia group associated with toxaemia of pregnancy were stillborn, indicating that the risk of toxaemia to the life of the baby appears to operate only in the antepartum period.

At autopsy, the extent and type of lesion depends largely on the manner in which the blood supply is interrupted. A sudden complete cessation of circulation through the umbilical cord may lead to death of the foetus without pathological signs being visible at necropsy. On the other hand, foetal anoxia resulting from premature separation of the placenta does produce characteristic lesions, which probably result from anoxic damage to the capillary endothelium and take the form of petecheal haemorrhage (Clairaux, 1958). In the present study deaths classified as anoxia of sudden onset showed minimal pathological signs, whereas those due to anoxia of gradual onset showed gross and widespread lesions in the form of patecheal haemorrhages beneath the serous membranes, haematoma of liver, subarachnoid and intraventricular haemorrhages and aspiration of amniotic sac debris.

In the present study, intraventricular haemorrhage was found in 5 per cent of deaths ascribed to anoxia. This is in contrast to the 76 per cent found by Clairaux, (1958). Massive intraventricular haemorrhage is the result of ruptured subependynal haemorrhage. Blood may track upwards over the brain in the subarachnoid space and subarachnoid haemorrhage is common accompaniment of intraventricular haemorrhage. Haemorrhage and extravasation of blood in the subarachnoid space is not to be confused with haemorrhage resulting from birth trauma which is almost invariably subdural in origin.

When confronted with stillbirths showing signs of anoxia it may be difficult to decide whether death occurred before or during labour (especially if maceration is absent or minimal). It may be impossible to make a decision without a record of the foetal heart sounds. It is babies of intrapartum anoxia group (those dying after the onset of second stage of labour) that most commonly require differentiation into those that have had a separate existence and those that

have been born dead. With experience, histopathological examination of lungs makes this decision fairly easy, except in cases living less than one or two minutes. The atelectasis seen in the stillborn babies is characterised by separation of the alveolar wall to a uniform degree and by absence of vascular congestion (Bound *et al*, 1956).

Summary

Autopsies were performed on 165 newborn infants dying in perinatal period at the Medical College Hospital, Aurangabad. The lesions attributable to anoxia accounted for 40 (24.1 per cent) of perinatal deaths. The associated maternal and obstetric conditions were analysed and discussed.

Acknowledgement

We are thankful to Dean, Medical College, Aurangabad for his permission to publish this paper.

References

1. Bound, J. P., Butler, N. .R. and Spector, D. G.: Brit. Med. Jour. 2: 5003, 1956.
2. Clairaux, A. E.: Neonatal pathology in Modern tends in pediatrics. 2nd series. Holzel, A. and Tizard, J. P. M. Ed. Paul, B. Hoeber, Inc. New York, 1958.
3. Ghosh, S., Bhargava, S. K., Sharma, K. B., Bhargava, V. and Saxena, H. M. K.: Ind. Pediat. 8: 421, 1971.
4. Hadley, G. G., Gault, E. M. and Gralam, M. D.: J. Pediat. 52: 139, 1958.
5. MacGregor, A. R.: Arch. Dis. Child. H. 14: 323, 1939.
6. Maheshwari, H. B., Kuldeep Teja., Savita Rani and Sharad Kumar: Ind. Pediat. 8: 417, 1971.
7. Mehdi, Z., Naidu, P. M. and Gopal Rao, V.: Ind. J. Med. Res. 49: 847, 1961
8. Potter, E. L.: Pathology of foetus and Ind. Med. Assoc. 22: 265, 1953.
   new born, Year book publishers, Chicago. 1952.
9. Rathnawathi, C. and Reddy, D. J.: Jour. Ind. Med. Assoc. 22: 265, 1953.
10. Shiela Karan, Bhargava, P. M., Suraindra, Y. A. and Seetha.: Ind. Pediat. 9: 99, 1972.
11. Stowens, D.: Pediatric pathology. Williams & Wilkins Co., Baltimore, 1959.