

# A CLINICO-PATHOLOGICAL STUDY OF PERINATAL MORTALITY

by

N. N. ROY CHOWDHURY,\* M.O., Ph.D. (Cal), F.R.C.S. (Edin),  
F.R.C.O.G., R.A.C.S.

and

K. SIKDAR,\*\* D.G.O., M.O. (Cal), M.R.C.O.G.

Thousands of foetal wastages during perinatal period throw a challenge to the obstetrician to find the probable causes either in the mother or in the foetus. This is only possible if perinatal mortality study is based on histopathological findings. In this communication a study of perinatal deaths in Eden Hospital during 1977 and 1978 was undertaken.

## Material and Methods

There were total 17,431 deliveries and 1,484 perinatal deaths, amongst whom 732 were stillbirths and 752 were neonatal

deaths. Autopsy examination was undertaken in 237 (16 per cent) cases only. The pathological classification of post-mortem findings were based on autopsy findings as presented by Wahal and Gupta (1967) and divided into pulmonary, non-pulmonary and undetermined causes.

## Clinical Analysis

Amongst 17,431 births, 17,191 were single births and 120 were twin pairs (240 babies) as shown in Table I.

Table I shows that perinatal mortality was 4 times higher in twin pregnancy.

TABLE I

Deliveries	Viable births	Live births	Still births	1st Wk. deaths	Total P.N.M.	Still-births/1000 births	P.M.R./1000 births
Single	17191	16498	693	709	1042	40.3	81
Multiple	240	201	39	43	42	162.5	341.6

TABLE II

## Maternal Age and Perinatal Mortality

Age in years	No. of total births	Still-births	1st wk. deaths	Perinatal deaths	
				Total	Percentage
Upto 20	2722	168	137	305	11.3
21 - 30	11085	338	387	725	6.5
31 - 40	3123	164	174	338	11.1
Above 40	501	62	54	116	21.1

\*Professor of Obstetrics and Gynaecology, Medical College, Calcutta.

\*\*Registrar, Eden Hospital, Medical College, Calcutta.

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Amongst 82 twin babies, who died, 24 were 1st twin and 58 were 2nd twin, showing thereby that 2nd twin is at higher risk.

It has been observed that perinatal mortality was commoner in young mother and to those who passed 40 years.

The perinatal deaths were commoner when gestational period was less than 38 weeks.

TABLE III  
*Parity and Perinatal Mortality in Relation to Number of Hospital Admission in Each Parity*

Perinatal deaths	Primi. (6950 caess)	P <sub>1</sub> +0 (4815 cases)	P <sub>2</sub> +0 (2959 cases)	P <sub>3</sub> +0 (1331 cases)	P <sub>4</sub> 0 (596 cases)	P5 and above (539 cases)
Stillbirths	305	101	104	95	52	72
Neonatal deaths	307	97	98	95	59	96
Total deaths	612	198	202	193	111	168
Percentage	9.1	4.1	6.8	14.4	18.7	27.4

It is evident from the above Table that mortality was higher in the 1st born, dropped in 2nd and 3rd and began to rise after 4th attaining higher as number of parity rose.

*Socio-Economic Condition and Perinatal Mortality*

Amongst booked cases, there were 161 perinatal deaths, compared to 1342 amongst unbooked cases, showing that perinatal mortality was higher amongst booked cases probably due to malposition, malpresentations etc. in them.

TABLE VI  
*Perinatal Deaths According to Sex of Babies*

Sex	Still births	Neonatal deaths	Perinatal mortality
Male	421	438	859 (57%)
Female	311	314	625 (43%)

It is observed from the above Table that perinatal mortality was higher in male babies.

TABLE IV  
*Perinatal Deaths in Relation to Gestational Period*

Stillbirths		Neonatal deaths		Perinatal deaths	
Less than	More than	Less than	More than	Less than	More than
428	304	431	322	859 (57%)	626 (43%)

TABLE V  
*Perinatal Deaths According to Birth Weight of Babies*

Stillbirths		Neonatal deaths		Perinatal deaths	
<2500 gms.	>2500 gms.	<2500 gms.	>2500 gms.	<2500 gms.	>2500 gms.
464	268	496	257	960 (63%)	525 (37%)

TABLE VII  
Factors Associated with Perinatal Mortality

Factors	Still- births	Neona- tal deaths	Perinatal deaths	
			Total	Per cent
Toxaemia	135	130	265	18.0
Antepartum haemorrhage	140	86	226	15.0
Placental factors (small placenta)	7	12	19	1.3
Cord factors (knots)	3	4	7	0.49
Difficult and prolonged labour (with uterine rupture)	103	96	199	14.0
Cord prolapse	43	18	61	4.27
Malpresentation	78	68	146	10.2
Premature rupture of membranes	18	12	30	2.0
Postmaturity	12	8	20	1.4
Foetal malformation	9	27	36	2.5
Heart disease	7	9	16	1.1
Anaemia	40	18	58	3.9
Diabetes	14	11	25	1.75
Hypertension	4	5	9	0.6
Haemolytic disease of newborn	2	18	20	1.4
Intranatal asphyxia, neonatal diseases and unknown	117	230	347	25.2
Total	732	752	1484	100.0

The above Table shows that maternal toxaemia, antepartum haemorrhage, difficult labour, malpresentation were associated with higher perinatal mortalities.

#### Causes of First Week Deaths

Acute gastroenteritis 29, asphyxia neonatorum 79, aspiration pneumonia 34, bronchopneumonia 16, congenital heart diseases 16, haemorrhagic disease of the born 9, neonatal jaundice 12, prematurity 262, premature asphyxia 62, respiratory distress syndrome 30, septicaemia 113, other complications of prematurity, e.g. pulmonary haemorrhage, etc. 22, meningitis 8, birth injury 15, congenital malformation 27, unknown causes 25.

#### Congenital Malformation

In 27 neonatal deaths, anencephaly 11,

hydrocephalus 3, ectopia cordis, meningocele 3, absence of chin and palate 1, omphalocele 2, huge enlargement of abdomen 1, achondroplasia 1, Edwards syndrome 1, atresia of gastrointestinal tract 3.

Amongst 9 still births, hydrocephalus 2, amencephalus 3, sacrococcygeal teratoma 1, conjoined twin 1, foetus papyraecus 1, hypoplastic urinary tract 1.

The above Table shows that abnormal presentations carry higher perinatal loss.

Postmortem examinations of dead foetus were undertaken in 237 cases in this series.

#### Pulmonary Factors (72 cases)

(1) Pulmonary anoxia—20 (28%) which includes 18 cases of immaturity of lung parenchyma and 2 cases of pulmonary malformations. (2) Massive aspiration of vernix caseosa—20 (28%).

TABLE VIII  
*Methods of Delivery and Perinatal Mortality*

Methods	Total deaths	Total individual deliveries	Perinatal loss in each (%)
Spontaneous cephalic delivery	1003	13009	0.77
Forceps	89	1816	5.00
Caesarean section	149	1962	7.5
Breech delivery	201	595	33.0
Destructive operations	34	34	100.00
Other methods	8	15	60.0

TABLE IX  
*Distribution of Different Presentations in Perinatal Mortality*

Presentation	Total deaths	Total deliveries during this period	Total Mortality (Per cent)	Perinatal mortality (per cent)
Vertex	1188	16576	7.2	80.0
Breech	209	695	35.0	14.0
Transverse	45	88	48.0	3.0
Face and Brow	20	45	48.0	1.5
Compound	22	27	88.0	1.5

TABLE X  
*Causes of Deaths in 237 Cases Based on Autopsy*

	Pulmonary	Non-pulmonary	Causes undetermined
Stillbirths	17	82	21
Neonatal deaths	55	35	27
Total	72	117	48
Per cent	30.3	49.3	20.3

(3) Massive intra-alveolar haemorrhage—9 (12.6%). (4) Pneumonia—14 (18.8%) and Hyaline membrane formation—9 (12.6%) cases.

#### *Autopsy Findings in Cases of Immaturity of Lung Parenchyma*

The lungs were immature or canalar type characterised by continuous alveolar lining of cuboidal epithelium and thick intra-alveolar septa with excessive

amounts of undifferentiated mesenchyma in which alveolar capillaries were not only few but also separated from the alveolar space by appreciable connective tissues.

#### *Autopsy Findings in Pulmonary Malformation Group*

Lungs were of moderate hypoplastic type. Histopathological study showed mature lungs. One of them had achon-

droplastic deformity and the other had huge enlargement of abdomen. One of these 2 cases showed visceral deformities such as polycystic kidney, multiple cystic liver and hypoplastic gastrointestinal tract. On histology, much of the liver was replaced by irregular masses of connective tissue containing innumerable bile ducts.

#### *Autopsy Findings in Massive Aspiration of Vernix Caseosa*

The lungs were firm, solid and congested in stillbirths but in neonates few patchy areas of aeration was present. On histology, alveolar spaces were filled with vernix caseosa, showing eosinophilic granular material mixed with twisted squamous cells.

#### *Autopsy Finding in Massive Intra-alveolar Haemorrhage*

Lungs were deep red in colour with extensive subpleural haemorrhage on histology, the alveoli and alveolar septa contained large number of R.B.C.

#### *Autopsy Findings in Pneumonia*

Lungs were firm, heavy, greyish pink in colour and the trachea and bronchi contained thick dirty mucus. Histology showed bilateral and diffuseness of the inflammatory process; adjacent alveoli was collapsed. In 3 cases hyaline membrane was found and in 2 cases alveoli contained vernix caseosa.

#### *Hyaline Membrane Formation*

The lungs were found deeply congested and mostly unexpanded though they floated in water. A few subpleural petechial haemorrhages were also seen. Histology revealed that lungs were canalicular or alveolar in type with alternating patches of ectasia and inectasia. In the ectatic areas alveolar walls were covered by an undulating ribbon like lining of deeply eosinophilic material which also often extended into the respiratory ducts. This material was seen in the alveoli in the form of coiled masses. The alveoli septa shows leucocytosis.

#### *Autopsy Findings in Intrauterine Anoxia Cases*

(1) Visceral congestion; (2) patchial haemorrhage; (3) Subarachnoid or intraventricular haemorrhages; (4) Exudate in the body cavities.

On histology—(1) Intense engorgement of vessels of lungs with/without perivascular and interstitial pulmonary haemorrhage; (2) Intense engorgement of liver sinusoids; (3) Cloudy changes in the kidney; (4) Congestion of brain heart and thymus.

#### *Birth Injury*

Autopsy showed subdural haemorrhages and haemorrhages in the base of the brain. Tardious spots were present in heart and lungs. Histology showed manifestations of anoxia in all organs.

#### *Non-Pulmonary Factors (117 cases)*

Causes	Stillbirths	Neonatal diseases	Total and Per cent
Intrauterine anoxia	67	23	90 (76%)
Birth injury	6	4	10 (8.5%)
Congenital malformation	10	4	14 (12.2%)
Duodenal perforation	—	1	1 (0.8%)
Haemolytic disease of newborn	—	2	2 (1.7%)

### Congenital Malformations

**Hydrocephalus:** The cerebral cortex seemed to be thinner. Histology of the brain remained unaltered.

**Atencephalus:** Cranial vault was found to be completely missing. The thymus was enlarged, lungs hypoplastic and adrenal glands were extremely small. The adrenal glands on section showed the picture of infants of several months old, containing medullary tissue than glands of normal neonates and the cortex lacked wide inner-zone found in foetus.

**Ventricular Septal Defects:** Involved the entire membranous portion of septum.

**Hypoplastic Urinary Tract:** Both the kidneys and bladder were hypoplastic. Section shows kidney composed mainly of connective tissue with few tubules and glomeruli.

**Huge Enlargement of Abdomen:** Uterus was elongated. There were bilateral hydronephrosis and distended bladder. Histology showed dilatation of kidney tubules and hypertrophy of muscular layer of bladder.

**Sacro-Coccygeal Teratoma:** A bilateral gluteal tumour 10" x 8" was detected. Histology showed undifferentiated teratoma.

**Conjoined Twin:** A thoracopagus. Autopsy showed single liver.

**Haemolytic disease of Newborn:** All viscera were yellow, spleen enlarged. Section of spleen showed reduction of white pulp and lymphoid hypoplasia.

Section of liver showed areas of erythropoiesis.

**Duodenal Perforation:** Two small perforations 2 m.m. in diameter found at duodenum, section of which showed defect in muscular layer.

**Atresia of Gastrointestinal Tract:** Two cases had ileal atresia and other was a case of unperforated anus with recto-vaginal fistulae.

**Cause Undetermined:** A thorough clinical and pathological examination in 48 (20.3%) cases showed no abnormality. In few of them autopsy examination showed characteristic of anoxia without any explanation for this death.

### Summary

Postmortem examination failed to identify the causes of deaths in 48 cases. Reduction of perinatal mortality depends on identifying these unknown factors along with other measures.

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### References

1. Wahal, K. M. and Gupta, J. S.: Ind. J. Med. Res. 4: 325, 1967.