THE OBSTETRICIAN AND THE HIGH RISK INFANT*

H. DE V. HEESE, B.Sc., M.D., M.R.C.P. (EDIN.), D.C.H., Department of Paediatrics, University of the Witwatersrand, Johannesburg

The major responsibility of the obstetrician and the paediatrician is to present the mother with an infant who has the potential to develop into normal adulthood. A low perinatal mortality is frequently quoted as a measure of the excellence of antenatal care, management of labour and neonatal care. It is but a crude criterion, and the emphasis should be on morbidity rather than mortality. Morbidity encompasses a wide spectrum of disease. It includes neurological syndromes such as severe cerebral palsy and syndromes which may only become evident in the school-going child.

Interest in the newborn in many quarters in South Africa can best be described as bekrompe. Hospital care is sanctioned for the mother for prolonged periods, regardless of cost, so as to deliver her of a live-born infant, but all interest is then lost as to whether suitable staff and facilities are available for the further care of the infant. Apart from the misery to the parents and family of a brain-damaged or abnormal child, the ultimate cost to the State or community is not kept in mind. This cost stretches over the whole life-span of the individual. It includes hospitalization, medical and surgical expenses, special schooling, institutionalization and financial loss of earning capacity. Thus, the prevention of brain damage in only one infant per year would justify the money spent on paediatric staff and equipment provided for a maternity unit.

THE 'HIGH RISK INFANT'

The Delivery

6

There are 6 essential requirements for the successful management of the 'high risk' foetus and infant. It is the responsibility of the obstetrician in the first instance to ensure that the following conditions are fulfilled at the hospital or obstetrical unit where the delivery of the infant is to take place:

- 1. The infant should be delivered in an institution orientated towards the maximum care of the newborn.
- 2. A paediatrician should be in attendance at the delivery.
- The hospital must be adequately staffed with paediatricians experienced in the care of the sick neonate.
- Trained nursing personnel must be available. They play a vital—perhaps the most important—role in the successful management of the 'high risk infant'.
- 5. Facilities must be available for the adequate resuscitation of the infant and for intensive care during the early neonatal period.

*Paper presented at the Interim Congress of the S.A. Society of Obstetricians and Gynaecologists (M.A.S.A.), Hermanus, April 1968. A laboratory must be attached to the unit to provide accurate acid-base estimations, arterial oxygen tensions and other vital biochemical investigations at short notice.

The importance of the foregoing is shown in Table I, where the mortality in infants of diabetic mothers in 3 maternity units is compared. Specialized facilities existed at Groote Schuur Hospital, whereas these facilities were not available to the staff in the other units.

The further responsibilities of the obstetrician in the management of the 'high risk infant' are as follows:

- (a) The management of the pregnancy and delivery of the foetus or infant recognized to be 'at risk'.
- (b) The recognition during the immediate neonatal period of the 'infant at risk'.

MANAGEMENT OF THE RECOGNIZED FOETUS AT RISK

Certain problems always arise and, from experience, the following are perhaps the most important:

The Hazards of Premature Delivery

Prematurity does not present much of a problem provided the infant weighs more than 2,000 G. For infants between 1,500 and 2,000 G, the over-all mortality for 'at risk' infants is probably between 10 and 20%, whereas the mortality and morbidity for infants less than 1,500 G rise steeply with a fall in weight.

As far as gestational age is concerned, the hazards of respiratory distress and hyaline membrane disease are very real in all infants delivered under 37-38 weeks of gestation. In infants of less than 33 weeks, who develop respiratory distress, the outlook is poor. They may die of hyaline membrane disease or intracranial haemorrhage. The hazards of oxygen toxicity to the eyes and possibly to the lungs are also greater the lower the gestational age. In this respect, a gestational age of 33-34 weeks seems to form a sharp line of demarcation between infants who may recover completely and those who may be left with residual damage.

Caesarean Section versus Vaginal Delivery

There are many recorded physiological differences between infants delivered by caesarean section and those delivered *per vaginam*. Some of these differences are listed in Table II. In the relatively well foetus or infant these differences are probably of small significance, whereas in the infant with pulmonary disease it is just possible that they may play an important part in neonatal mortality and morbidity.

The reasons for these differences are, in many cases, not clearly established and the role of anaesthesia needs further

TABLE I. INFANTS OF DIABETIC MOTHERS: MORTALITY 1964 - 1966

Hospital and maternity unit	No. of infants	Stillbirths	Neonatal deaths	as % of live-births	as % of infants delivered
Peninsula Maternity 1964 - 1965	21	2	3	16	24
New Somerset 1964 - 1965	9	2	1	14.3	33
Groote Schuur 1964 - 1966	78	3	5	6.7	10.3

study. There is, however, a difference between the lung function and the acid-base status during the first 24 hours of life of infants born by caesarean section and infants born after spontaneous vaginal delivery.¹

TABLE II. RECORDED DIFFERENCES BETWEEN INFANTS DELIVERED per vaginam and by CAESAREAN SECTION

Parameter	Vaginal delivery	Caesarean section		
Crying vital				
capacity	Normal	Severely depressed		
Functional				
residual				
capacity	Normal	Normal		
Serum proteins	Normal	Depressed		
Serum Ca++	Normal	Depressed		
Serum K+	Normal	Raised		
Haematocrit	Normal	Depressed		
Response to				
glucagon	Normal	Depressed		
Cardiac output	Normal	Depressed		
Acidosis	Mild	Moderate to severe		

Studies carried out at Groote Schuur Hospital² further support the view held by Usher,³ as opposed to the views of Strang *et al.*⁴ that hyaline membrane disease occurs more frequently after delivery by caesarean section.

Delivery of the 'high risk infant' by caesarean section is often associated with 5 serious mistakes which may prejudice the survival and condition of the foetus and infant.

1. The delivery of any infant by caesarean section should never be considered without a paediatrician being present in the theatre before the operation is commenced. The relationship between severe depression or asphyxia at birth, and lack of active resuscitation with the severity of hyaline membrane disease and intracranial haemorrhage^s has been established.

2. Theatre temperature should not be regulated for the comfort of the staff with complete disregard for the hazards of hypothermia to the infant. The lower the neonate's body temperature falls after delivery and during the first few days of life, the higher the mortality and morbidity.^{6,7} A low environmental temperature is the most important factor in the production of body temperatures below 95°F. Premature infants, severely depressed infants with Apgar scores under 4, actively resuscitated infants and the infant with cerebral birth injury and low birthweight for gestational age are particularly prone to hypothermia.

The presence of hypothermia is often not recognized because of (i) the first impression of good health, when looking at the hypothermic infant—the face is red and the hands, feet and mucous membranes are well coloured; (ii) failure to measure the infant's temperature with a low-reading thermometer.

3. An elective caesarean section (or induction of labour) should be avoided on a Friday, Saturday or Sunday. The successful management of a sick infant involves not only the paediatrician and nursing staff, but ancillary services such as biochemical laboratories and radiological services. There is often a shortage of staff over weekends.

4. A paediatrician should be familiar with resuscitation procedures, and a check of equipment must be carried out before the delivery of the infant.

5. Excessive suction of the mouth and the nasopharynx with high-pressure suction apparatus must be avoided.

This pernicious, but common, habit inevitably causes damage to the mucosa and results in portals of entry for bacteria. Obstetricians should now and then inspect the palate, nasopharynx and larynx of infants so iatrogenically traumatized by over-enthusiastic, misguided efforts. Excessive suction also frequently causes laryngeal spasm and difficulty with resuscitation.

Placental Transfusion

Much has been written about the correct moment at which the cord should be tied. There is general agreement that it should be tied early where blood-group sensitization is suspected. Where the cord is tied late, the infant's blood volume can be increased by 35 - 61%.

A recent study^s suggests that uterine contraction is the most important factor as far as the speed and transfer of blood is concerned and that the respiratory efforts of the infant and gravity play no role in the determination of the volume transfused. Whether a large placental transfusion to the baby carries any special advantage is debatable. No differences in the haemoglobin content could be demonstrated at 3 months between babies with early or late clamping of the umbilical cord.⁹ I suspect, though, that in an average delivery, the cord is clamped at approximately one minute and the infant, therefore, receives an 80% or greater transfusion. It would be of interest to record exactly the time the cord is clamped in a large series of normal cases.

Foetal Distress

Having stressed the danger of caesarean section, it must be pointed out that the only sure relief of foetal distress is the prompt delivery of the infant. As far as oxygen administration to the mother is concerned, it is of help where foetal distress is secondary to maternal hypoxia. Where it is due to a block of foetal gas exchange at a placental level, the effects of oxygen administration are uncertain.⁹ Evidence is accumulating that a high maternal arterial PO₂ level may cause vasoconstriction of the umbilical vessels and ductus arteriosus of the infant and also vasoconstriction of the placental spiral arterioles.

Lack of Communication

Lack of communication between obstetrician and paediatrician may add to the problems of the 'high risk infant'. The paediatrician is often warned about maternal diabetes, pre-eclampic toxaemia, illness during the first trimester of pregnancy, and sedation, but other equally vital information is not passed on, e.g. polyhydramnios which is associated with oesophageal or upper intestinal atresia; oligohydramnios which is associated with renal disease; prolonged rupture of the membranes; or renal infection in the mother which is associated with neonatal infection and prematurity.

RECOGNITION OF THE 'HIGH RISK INFANT' DURING THE EARLY NEONATAL PERIOD

A heavy responsibility rests on the obstetrician to recognize certain early signs warning of potential disease in the newborn. Cases are often referred to the paediatrician so late that little can be done to prevent death or morbidity. Obstetricians must be aware of the following:

Interpretation of the Apgar Score

The Apgar scoring system (Table III) is widely practised and is probably one of the most useful measures of the clinical state of the infant at birth.

TABLE III. APGAR SCORING SCALE

Sign	0	1	2
Heart rate Resp. effort Muscle tone Cath. in nose Colour	Absent Absent Limp No response Blue—pale	<100/min, Weak cry Flexed extremities Grimace Body pink, extremities blue	>100/min. Strong cry Well flexed Cry Pink

Certain modifications to the system have been suggested but are not generally accepted, the most important being that signs such as the heart rate and respiratory effort should be loaded. I personally feel that they are the 2 most important criteria, and would like to suggest that a scoring system based on these criteria and certain intrapartum parameters such as the characteristics of the foetal pulse be explored and developed.

Two common mistakes are made in the assessment of the state of an infant at birth:

- (i) Where the observer is emotionally involved with the case, i.e. the obstetrician or midwife, too high an Apgar score is allocated.
- (ii) Where an automatic timer is not available in the theatre, scores are allocated at the wrong time. It is, in practice, extremely difficult under conditions of stress to allocate a score punctually at the required times of 1 min., 3 min. and 5 min.

A low Apgar score is associated with both an increased mortality and morbidity.³⁰ Mortality is usually associated with death within the first 2 days of life and is also related to a low birthweight. Morbidity is associated with respiratory distress in the immediate postnatal period, and later in life with evidence of permanent brain damage.³⁰ The latter correlates best with a low 5-minute Apgar score in infants who survive the first year of life.

All infants with low 3-minute Apgar scores require intensive premature care and intravenous fluids of 15% dextrose solution with added sodium bicarbonate for 36-48 hours after resuscitation.

A note of warning must be issued against the dangers of injecting large single doses of sodium bicarbonate and/or high concentrations of dextrose-solution into the umbilical vessels immediately after delivery. Any procedure on the umbilicus such as the introduction of catheters or administration of intravenous fluids must be carried out with strict aseptic precautions. The hazards of infection and a thrombosis of the umbilical vein and its connections, and portal hypertension in later life, are well documented. Extensive necrosis of the buttock and/or sciatic palsy may also result from the introduction of drugs or concentrated dextrose solution into the umbilical vein. These effects may be transient but are commonly permanent.

Respiratory Distress in the Newborn

The term 'respiratory distress' is employed to describe a clinical picture of abnormal breathing irrespective of its cause.

A diagnosis of respiratory distress is made if 2 or more of the following symptoms and signs are present:

- 1. Respiratory rate of more than 60/min., which is maintained for more than 3 hours.
- 2. Expiratory grunting present after 3 hours of age.
- 3. Cyanosis in room air.

4. Marked costal and sternal recession.

5. Pulmonary crepitations.

Respiratory distress may be caused by pulmonary or extrapulmonary pathology. The extrapulmonary causes include conditions such as cerebral birth injury, congenital heart disease, hypoglycaemia, diaphragmatic hernia and acute haemorrhage. The pulmonary causes present the chief threat to life in newborn infants and may be caused by specific diseases such as pneumonia, pneumothorax and meconium aspiration. However, the commonest cause is hyaline membrane disease.

Hyaline membrane disease occurs in approximately 14-16% of all premature deliveries. It is the commonest cause of death in the neonatal period, and the aetiology of the condition is unknown.

Infants suffering from hyaline membrane disease show evidence of respiratory distress soon after birth.

When faced with an infant with respiratory distress it is important to make a definite diagnosis and institute therapy or refer the sick infant to a neonatal intensive care unit as soon as possible.

So-called 'observation' under these conditions may lead to further deterioration and the onset of asphyxial attacks. The outlook for any infant after the onset of an asphyxial attack is poor, especially where the attack is associated with profound slowing of the heart rate.⁵ Thus, the mortality of 25 infants with severe hyaline membrane disease and a history of severe recurrent asphyxial attacks before admission to the neonatal intensive care unit at Groote Schuur Hospital was 100%.

It must be stressed that moving a small, sick infant from one hospital to another incurs hazards such as hypothermia, anoxia and the possible risk of infection. These potentially fatal hazards are preventable by taking adequate precautions to keep the baby warm and by the administration of humidified oxygen in a high concentration via a face mask to the infant during transfer. The administration of intravenous sodium bicarbonate and 15% glucose before or during the transfer of the infant further improves the ultimate outcome.

The tragedy, however, is that authorities in charge of ambulance services still have to be convinced of the importance of transferring newborn babies and infants in portable incubators. The immediate financial expenditure is still regarded as being more important than the final outcome of the case in terms of morbidity and mortality and ultimate financial cost.

The incidence of asphyxial attacks, morbidity and mortality in infants admitted to intensive care units are also dependent on the standard of medical and nursing care, monitoring facilities and vital auxiliary services such as radiological and biochemical services. The improved results in referred cases of severe hyaline membrane disease obtained in the neonatal respiratory unit of Groote Schuur Hospital for 1962 - 1965 compared with 1965 - 1967 were entirely due to improvements in the already mentioned services and facilities (Tables IV and V).

TABLE IV. INCIDENCE OF HYALINE MEMBRANE DISEASE: ASPHYXIAL ATTACKS AND MORTALITY

			First asphyxial attacks	
	Infants at risk*	Weight (kg.)	recorded in unit	Mortality
1962 - 1965 1965 - 1967	51 49	2·23 2·18	41·2% 25·5%	71·4% 16·7%

*Excluding cases with a history of asphyxial attacks before admission or a birthweight <1.13 kg. or gestational age <31 weeks.

TABLE V. HYALINE MEMBRANE DISEASE: MORTALITY

1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1		and the second sec	Salvable*	le*	
Period	Admissions	Mortality	cases	Deaths	
1962 - 1965	64	30(46.9%)	50	17(34%)	
1965 - 1967	59	12(20-3%)	49	2(4%)	
*Excluding cas	ses with asphyxia	attacks befor	e admission, o	ases with major	

weeks.

All cases of hyaline membrane disease must therefore be referred early to properly staffed and equipped units for management and treatment.

The Low-Birthweight-for-Gestational-Age Infant

Infants of similarly low birthweight can vary widely in gestational age and functional maturity. Approximately 30-40% of infants classified as premature by weight (<2.5 kg.) are born from pregnancies lasting from 38 to 40 weeks. The importance of recognizing low-birthweightfor-gestational-age infants lies in the seriousness of associated complications such as hypoglycaemia, massive pulmonary haemorrhage, hypothermia and metabolic acidosis.

Low-birthweight-for-gestational-age infants can be recognized by their clinical appearance at birth, from intrauterine growth" charts or from certain objective criteria.12,13

The clinical appearance at birth depends to a large extent on the duration, intensity and time of onset of the growthretarding influence. Two clinical types can be recognized:

1. The long, thin, low-birthweight infant with a wrinkled, dry, peeling skin and alert appearance, showing signs of apparent recent loss of weight. The growthretarding influence is thought to occur close to term.

2. The 'stunted', short, lively, low-birthweight infant with well-developed grasp and sucking reflexes. These infants show evidence of failure of both the subcutaneous deposition of fat and growth in length. The growth-retarding influence in these infants is of long duration.

The criteria for the clinical assessment of the gestational age of infants are given in Table VI. The skin-creases on the sole of the foot and the size of the breast nodule are the most reliable physical indices of maturity.

TABLE VI. CLINICAL CRITERIA FOR THE ASSESSMENT OF GESTATIONAL AGE¹²

		Gestational age (weeks)		
	<36	36 - 38	>39	
Sole creases	Ant. transverse	Occasional creases ant. $\frac{2}{3}$	Sole covered	
Breast nodule diameter (mm.)	2	4	7	
Scalp hair	Fine fuzzy	Fine fuzzy	Coarse silky	
Earlobe	Pliable	Some cartilage	Thick cartilage stiff	
Testes Scrotum Rugae	Lower canal Small Few	Intermediate	Pendulous Full Extensive	

Gestational age and maturity can also be assessed on criteria based on neurological development. Although this type of assessment requires experience, Robinson³³ found that certain reflexes have sufficiently predictable times of appearance to provide simple and reliable indices of gestational age (Table VII).

Hypoglycaemia

Primary symptomatic hypoglycaemia may present at any time during the first 7 days of life, with a peak incidence between 24 and 48 hours. The condition is not uncommon and its early recognition is important.

Mortality and morbidity are high unless the infants are actively treated, and mental defect and/or seizures may be sequelae found in later life.

The condition occurs most commonly in infants of low birthweight for gestational age, the smaller of twins, and in infants of diabetic mothers.

An infant may present with any of the following signs: tremulousness and irritability; apathy and limpness; cyanosis, signs of respiratory distress, poor feeding, eye-rolling, a high-pitched cry and a depressed Moro reflex. Apnoeic spells and convulsions occur late and are of serious prognostic significance. None of these symptoms or signs is pathognomonic, and tetany, meningitis, respiratory distress and intracranial haemorrhage must be considered in the differential diagnosis.

The diagnosis is confirmed by the demonstration of a blood-glucose level of below 20 mg./100 ml. in an infant of low birthweight; and under 30 mg./100 ml. during the first 72 hours of life in a full-sized infant born at term with any of the above symptoms. In practice a provisional diagnosis of hypoglycaemia is made in an infant who presents with any of the above symptoms and a Dextrostix reading below 40 mg./100 ml. Therapy should be instituted without delay and consists of the administration of dextrose intravenously.

TABLE VII. REFLEXES AND THE ASSESSMENT OF GESTATIONAL AGE"

Reflex	Stimulus	Positive response	Gestation (weeks) if reflex is:	
		i osnive response	Absent	Present
Pupil reaction	Light	Pupil contraction	<31	29 or more
Traction	Pull up by wrists from supine	Flexion of neck or arms	<36	33 or more
Glabellar tap	Tap of glabella	Blink	<34	32 or more
Neck-righting	Rotation of head	Trunk follows	<37	34 or more
Head-turning	Diffuse light from one side	Head-turning to light	Doubtful	32 or more

1 Maart 1969

The Infant of the Diabetic Mother

Diabetes in the mother carries a high foetal mortality and neonatal mortality and morbidity. Hazards of the neonatal period include:

- (a) Respiratory distress.
- (b) The effects of an increased incidence of congenital malformations.
- (c) Metabolic disturbances, e.g. hypoglycaemia, acidosis.
- (d) Apnoeic attacks.
- (e) Electrolyte disturbances, e.g. hypoglycaemia and hypocalcaemia.
- (f) Congestive cardiac failure.

The management of these infants consists of giving them premature intensive care and the treatment of complications. In the management of this group it is important to recognize that these infants have less extracellular water and less total water than is normal. They are extremely liable to respiratory distress, hypoglycaemia and acidosis. For these reasons, infants of diabetic mothers should receive intravenous fluids consisting of 10% dextrose-bicarbonate solution during the first 24 or 36 hours of life.

The Transfusional Twin Syndrome

The mortality in monozygous twins is given as approximately 25%. This high mortality is partly due to the peculiar vascular anastomoses which may be present.

- 1. Artery-to-artery anastomosis may occur, but is nonconsequential.
- 2. Vein-to-vein anastomosis may cause abnormality in the one twin, but it is usually not serious.
- 3. The most important abnormality is a one-directional arterial venous shunt, giving rise to the following abnormalities: twin 1 is anaemic, retarded and looks malnourished; twin 2 is plethoric, oedematous, has cardiac hypertrophy and also renal hypertrophy. This second twin is 'at risk' and may die of cardiac failure, respiratory distress or corebral thrombosis. Treatment consists of urgent venesection and intensive care.

Single Umbilical Artery

The number of umbilical vessels should be recorded after each delivery. A single umbilical artery has been reported in approximately 0.7 - 0.8% of live-born infants.¹⁴ A similar incidence has also been observed in White South Africans.15

A large percentage of babies with single umbilical arteries are born dead or die soon after birth, and 90% of these have major congenital malformations. Of those born alive, 25 - 50% are found to have abnormalities, which may be single or multiple. Congenital abnormalities, in order of frequency, have been reported as follows: (i) skeletal, (ii) gastro-intestinal system, (iii) genito-urinary system, (iv) cardiovascular system, (v) central nervous system.

The importance of finding a single umbilical artery is that it should alert the observer to look for other and possibly correctable abnormalities, e.g. tracheo-oesophageal fistula within the first few days of life. Abnormalities may, however, only become apparent much later.

SUMMARY

The obstetrician's responsibility in the successful management of the 'high risk infant' is outlined. A plea is made for the recognition of the highly specialized field of neonatal paediatrics. The 'high risk infant' requires the best possible care.

This aim can only be achieved by the development within maternity units of separate departments of neonatal paediatrics which are adequately staffed and equipped.

The major risks of the 'high risk infant' are: (i) to be delivered by an obstetrician who is under the misconception that he knows all about the care of neonates; (ii) to be managed by a paediatrician who has had no training in the highly specialized field of neonatal care; (iii) to be delivered in an institution with poor facilities.

I wish to thank Dr J. G. Burger, Medical Superintendent of Groote Schuur Hospital and Dr R. Nurok, Medical Superintendent of Somerset Hospital, for permission to publish.

REFERENCES

- 1. Heese, H. de V.: Unpublished data. 2. Ma'an, A. F., Evans, A. and Heese, H. de V. (1966): S. Afr. J. Obstet. Gynaec., 4, 13. 3. Usher, R. H., McLean, F. and Maughan, G. B. (1964): Amer. J.
- Obstet. Gynec., 88, 806.
- 4. Strang. L. B., Anderson, G. S. and Platt, J. W. (1957): Lancet, 1, 954. 5. Harrison, V. C., Heese, H. de V. and Klein, M. (1968): Arch. Dis.
- Harrison, V. C., Hese, H. de V. and Klein, M. (1966): Arch. Dis. Chi dh., 43, 116.
 Day, R. L., Caliguiri, L., Kamenski, C. and Ehrlich, F. (1964): Pediatrics, 34, 171.
- 7. Silverman, W. A., Fertig, J. W. and Berger, A. P. (1957): Ibid., 20, 477.
- 8. Yao, A., Hirvensalo, M. and Lind, J. (1968): Lancet, 1, 380.
- Lanzkowsky, P. (1960): Brit. Med. J., 2, 1777

- Diage, J. S. and Berendes, H. (1966): Pediat. Clin. N. Amer., 11, 636.
 Malan, A. F., Evans, A., Smit, W. B. de V. and Heese, H. de V. (1967): S. Afr. Med. J., 41, 698.
 Usher, R., McLean, F. and Scott, K. E. (1966): Pediat. Clin. N. Amer., 11, 845.
- 13. Robinson, R. J. (1966): Arch. Dis. Childh., 41, 437.
- 14. Froehlich, L. A. and Fujikura, T. (1966): Amer. J. Obstet. Gynec., 94. 274.
- 15. Fanaroff, A. A. (1968): Personal communication.