NEWBORNS, CARE AND MANAGEMENT

by

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The care of newborns has been based more on usage rather than on scientific facts. That so many have survived is no argument against a change. Rather it is evidence of the toughness of the human frame, especially of the robust full term newborn. But it may not hold good for others, for those who are not robust and not full term according to their birth weights. And these are just the infants it is becoming increasingly difficult to salvage, both from morbidity and mortality, now that family planning has been extended even to postpartum sterilisation.

First of all, which are the infants who should be particularly looked after? They have been classified as the "high risk babies".

High Risk Babies

The use of "high risk nurseries" is being advocated in most of the larger institutions where babies are born. Those at high risk include babies with low birth weights, and incidentally also those with high birth weights, for in both the neonatal mortality is high. Infants born with the help of instruments also have a higher mortality. Where there has been difficulty in resuscitating the infant also increases the risk. Certain conditions such as hydramnios and congenital malformations carry a high mortality rate for the newborn.

Certain conditions in the mother, such as toxaemia, diabetes and hypertension can be lethal to the baby, so may the use of certain drugs, such as steroids, barbiturates, tranquillisers, addictions to LSD, morphine, etc. and high doses of vitamin K. It has also been suggested that cigarette smoking may produce infants who are small for dates.

Diagnosis of Congenital Defects

While many of these, such as mongolism, are obvious, some have to be searched for, so as not to be missed. Though the incidence of a single umbilical artery may be less than 1.0 per cent, the incidence of congenital anomalies in these cases may be over 30 per cent (Harris and van Leeuwen, 1968). A single umbilical artery may be difficult to pick up unless the cord is examined at its point of attachment to the infant. The anomalies usually associated with this defect are 16-18 trisomy and malformations of the lower urinary tract. 16-18 trisomy is associated with mental defect and certain facial features, (which do not really matter), and anomalies of the central nervous system (which do matter!).

Other congenital anomalies, which may not be obvious, can be diagnosed very simply with a No. 8 catheter. This may be passed through both sides of the nose to ensure that both choanae are patent. Then it may be slipped into the oesophagus to find out if there is any atresia there. Once it passes into the stomach, the contents may be aspirated. If the amount is less than 20 ml. and there is no

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bile in it, then there is probably no obstruction. Finally, the tube may be passed into the vagina and anus to make sure that both are patent.

When should the Cord be Tied

When foals are born naturally in the field, the mare takes some time to deal with the cord. When their birth is assisted by a vet, the cord is tied off early. But the ones who are delivered by vets are more likely to develop respiratory distress (Dunn, 1967-68).

The same holds true for the human newborn, especially the premature one. Ideally, the cord should be tied after delivery of the placenta. The placenta should be placed at the same level as the infant. Then, after all pulsations in the cord have ceased, it may be tied. incidentally, the infant thus also gets an extra 50-65 ml. of blood.

Nature herself uses the placenta as a safety valve. We should also use it for human babies.

Hyperbilirubinaemia in the Newborn

It is commonly presumed that all these cases are due to erythroblastosis. So, when the laboratory tests for this condition come negative, as they often do, the jaundice is presumed to be harmless and allowed to take its course. There is real danger here, for hyperbilirubinaemia may lead perhaps to kernicterus and mental defect.

Incidentally, there are many other causes of hyperbilirubinaemia, deficiency of bilirubin gluronyl transferase, an enzyme in the liver which accelerates bilirubin transfer, administration of vitamin K in doses over 1.0 mg., (a real temptation when all commercial preparations of vitamin K come in ampoules of 5 and 10 mg.) and a short gestational age. Diabetes in the mother and the presence of an inhibiting agent in the mother's milk (? progestin) may also lead to hyperbilirubinaemia.

The treatment is of course exchange transfusion, but for most cases phenobarbitone and phototherapy are all that are required. Phenobarbitone stimulates the formation of bilirubin glucuronyl transferase.

Early Feeding

It has been shown that feeding the newborn early, say 4 hours after birth, keeps down the bilirubin (Wennberg and Schwartz, 1966). If glucose water is given, then hypoglycaemia is avoided. To have the first feed as a clear liquid is of advantage, if it is vomited, no great harm is done even if it is inhaled. About 6 hours after birth, milk may be started.

This schedule should be modified for the infant who is too premature or immature and in whom inhalation is a real danger.

Convulsions in the Newborn

Anoxia is a well known cause of convulsions in the neonate. But the giving of oxygen, especially if it is given continuously, is known to give rise to hyaline membrane disease and even possibly to pulmonary haemorrhage (Shanklin and Wolfson, 1967).

Other causes of convulsions include intracranial injury, jaundice and other infections. Infections in the newborn may be difficult to diagnose. Meningitis, for instance, may occur without a rise of temperature and without neck rigidity. It may be possible to diagnose it only if a lumbar puncture is done.

Hypoglycaemia is another well known cause of convlusions, but there is no hard and fast rule about the level of the blood sugar which precipitates the convlusions. Hypoglycaemia may be suspected if the infant is premature, if he has apnoeic

spells, cyanosis and apathy. Sometimes it is associated with macroglossia and sometimes with leucine sensitivity. In later life there may be mental defect following such hypoglycaemic convulsions. Hypoglycaemia should of course be treated with a glucose solution, by mouth or by I.V. drip, 50 and then 10 per cent strength, but I.V. fructose may be more satisfactory, the release of glucose is slower and there is no after hyperinsulinism. Steroids and diazoxide (a thiazide derivative which has a hyperglycaemic effect) have also been useful.

Pyridoxine dependency is an interesting cause of convulsions in the newborn, occurring usually after the mother has received large doses of pyridoxine for hyperemesis gravidarum, etc. In 1967 there appeared a report of a woman in Czechoslovakia who complained that in 3 successive pregnancies she had felt the foetus convulsing in utero (Bejsovec *et al* 1967). The first two died in status after birth. But the third child was given large doses of pyridoxine immediately after birth and survived. The concept of intrauterine convulsions is difficult to prove or disprove, but these mothers seem to know instinctively which of their children will be normal and which will have pyridoxine deficiency after birth.

Vulnerability to Infections

Immunity is acquired as a result of infection, but an infection in the newborn may prove costly. This is because the infant at birth has only one immunoglobulin, IgG. The others, IgA and IgM, are present in only negligible amounts. IgG is derived from the mother, and the level of IgG in the infant depends on the level of IgG in the mother. After birth, some immunoglobulin may be derived from the colostrum in the early days, but thereafter the infant does not absorb any immunoglobulin from the mother's milk. The necessity for early immunisation becomes obvious.

The routine to-day is to give small-pox and B.C.G. at birth, polio vaccine at 2 and 3 months, triple vaccine at 4, 5 and 6 months and measles vaccine at 9 months.

Infections in the newborn should be avoided, for they are dangerous and invasive at this age. Hypersensitivity is absent and infections tend to become disseminated.

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