Resuscitating fresh stillbirths

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Summary: Guidelines for duration of resuscitation of freshly-stillborn term infants and their long term outcome are unclear. The predictive factors of both cerebral palsy and early neonatal demise include Apgar scores of 3 or less at 10 minutes, perinatal acidaemia and neonatal seizures. We describe the case of severe perinatal asphyxia (umbilical artery pH at birth of 6.75, Apgar scores of 0.1 and 5 0.1 5 at 1.5 and 10 minutes and neonatal hypertonia) in a term pregnancy, where the infant made an uneventful recovery and was discharged home well. Guidelines for neonatal resuscitation are discussed.

Key words: Stillbirth resuscitation; Perinatal asphyxia; Apgar Score.

INTRODUCTION

Severe perinatal asphyxia occurs in 6 per 1,000 live term births, and of these 1/1,000 die or are severly brain damaged ([1, 2]). With hypoxic-ischaemic encephalopathy, hypoxia and hypotension combine to produce neuronal necrosis, with spastic quadriplegia and mental retardation ([3]). Resuscitation of fresh stillbirths poses great problems for the neonatal emergency team because of unclear guidelines on duration and doubt concerning the long term outcome. Most quadriplegic and severely mentally retarded infants originate from successfully resuscitated fresh stillbirths and it is thus essential to define the limits of resuscitation to minimise the risk of infants suffering "a fate worse than death" ([4]).

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CASE REPORT

A 29 year old Para 4004, with an uncomplicated antenatal history, admitted herself in labour at 40 weeks gestation with a substantial antepartum haemorrhage of 300 mls. The bleeding settled following admission and labour was allowed to proceed. Abdominal examination revealed that the fetal lie was longitudinal, the presentation cephalic and the head engaged. On ultrasound there was a live intrauterine singleton gestation without evidence of placenta praevia or placental abruption. On vaginal examination, the cervix was 4 cm dilated. The amniotic membranes were ruptured and clear liquor drained. A fetal scalp electrode was applied and a normal cardiotocograph (CTG) trace obtained. Fortly minutes later, there was a further vaginal blood loss of 100 ml. The CTG trace showed a sustained deceleration of the fetal heart to 80 beats per minute (Fig. 1). Placental abruption was diagnosed and the patient was transferred to the theatre for emergency caesarean section.

At operation, a male infants, weighing 3.2 kg was delivered through a lower segment incision of a Couvelaire uterus. The infant was pale, hypo-

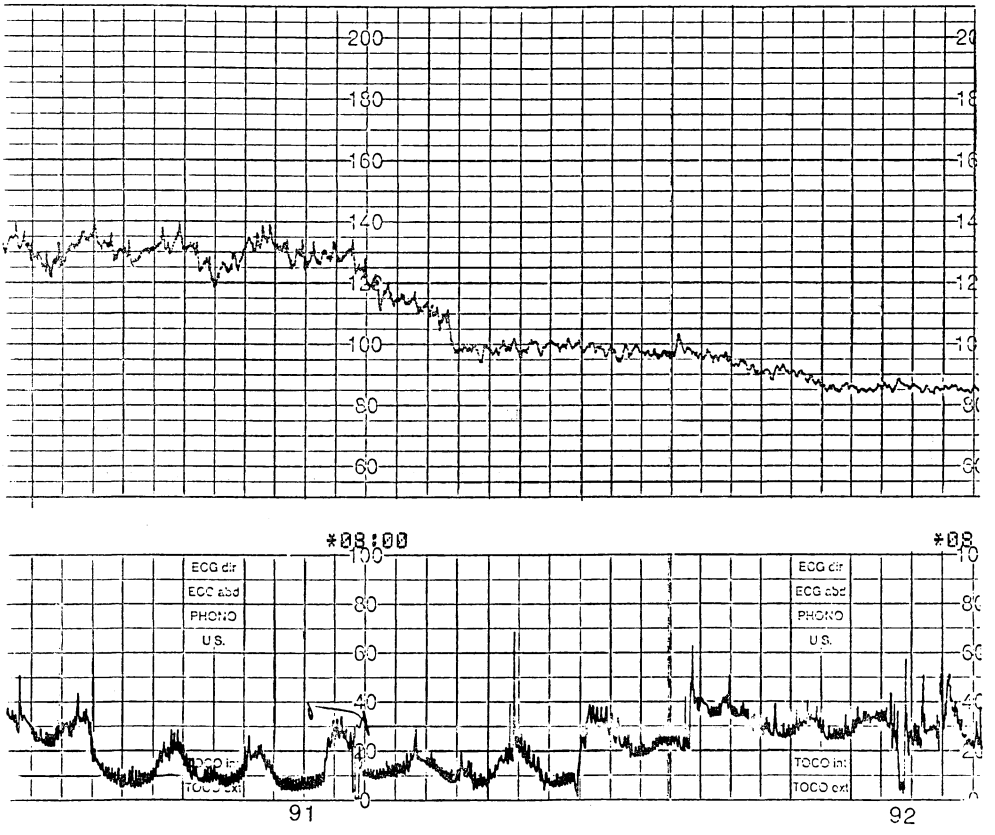

Fig. 1. — Cardiotocograph trace following artificial rupture of membranes.

tonic and lifeless with no recordable apex beat. Despite vigorous resuscitation, including intubation, ventilation, cardiac massage, and the administration of adrenaline, sodium bicarbonate and glucose. it was six minutes before an apex beat was obtained and 10 minutes before the heart rate exceeded 100 beats per minute. Apgar scores and cord gases were severely abnormal (Tables 1, 2).

Fifteen minutes after delivery the infant was transferred to the intensive care unit and placed in a headbox delivering 50% oxygen. He suffered an episode of apnoea and bradycardia and was artificially ventilated until regular respirations were established at two hours old. Repeat blood gases at 1 hour 40 minutes post delivery were much improved (Table 2). Four hours after delivery the infant was noted to be hypertonic with an irritable cry. He stabilised over the next 12 hours and by 16 hours old, examination of the central nervous system was normal. Despite the marked perinatal asphyxia, he had an uneventful neonatal course and was discharged home on the fifth day following delivery feeding and behaving normally. His subsequent six-week check was normal.

DISCUSSION

Perinatal asphyxia may cause brain damage but there is considerable uncertainty about factors that predict a poor neurological outcome. The Apgar score is our standard means of assessing the severity of birth asphyxia ([5]) and is a better predictor of long term outcome at 5 minutes than at 1 minute ([6-8]). However the limits of resuscitation are ill defined. Steiner *et al.* (1975) noted that "the pro-

Table 1. — *Apgar scores at 1, 5, 50, and 15 minutes.*

Minutes	*1*	*5*	*10*	*15*
Heart rate	0	0	2	2
Resp. effort	0	0	1	1
Muscle tone	0	0	0	1
Stimuli response	0	0	0	2
Colour	0	0	2	2
Total	0	0	5	8

Table 2. — *Blood gases from the umbilical artery at birth and at 1 hour 40 minutes after delivery.*

	Birth	1 hr 40 min
pH	6.75	7.24
pCO_2	11.18	4.97
pO_2	6.55	6.15
Standard Bicarbonate	Unrecordable	15.3
Standard Base Excess	Unrecordable	−10.6

gnosis appeared to be uniformly good" when the heart beat was restored within five minutes of birth and the infant breathing spontaneously within thirty minutes thereafter ([9]).

Applying a policy of active resuscitation using these criteria he showed that less than 20% of those who survived were brain damaged. Support for this approach came from many quarters, as it was felt that the quality of life enjoyed by the vast majority of these survivors justified a vigorous approach to resuscitation ([10, 11]).

D'Souza (1978) proposed that babies exhibiting apathy initially with subsequent hyperexcitability and extensor hypertonia, carried the worst prognosis ([12]). Nelson and Ellenberg (1979) in a large prospective study of 40,000 infants showed that the features associated with both cerebral palsy and early neonatal demise were seizures and Apgar scores of 3 or less at 10 minutes or later ([13]). It was calculated that a score of 3 or less at 10 minutes yielded a risk of 10% of cerebral palsy. Later reports suggested that neurological abnormalities were more likely in infants with Apgar scores less than 3 at five minutes in the presence of acidosis (umbilical artery pH < 7.27) ([14]).

In the case described, despite an Apgar score of 0 at 5 minutes, umbilical arterial acidosis (pH = 6.75) and hypertonia at 4 hours, the infant made an uneventful recovery. While the outcome was attributed to efficient resuscitation in a structurally normal term fetus, without prenatal evidence of compromise, it highlights the limited sensitivity and specificity of the guidelines based ou current clinical and biochemical markers of asphyxia. With no recordable fetal pulsation at 5 minutes in the presence of severe acidosis current opinion would support withdrawing resuscitative measures in the case described. Clearly more specific measures of neonatal outcome in the presence of asphyxia would facilitate the decision whether to continue or withdraw resuscitation.

Recently, Doppler studies of cerebral blood flow velocities have shown a strong correlation between abnormal Doppler signals from the anterior cerebral arteries of asphyxiated infants and subsequent adverse outcome ([2]). It has been suggested that high cerebral blood flow velocities are the result of vasoparalysis of arterioles and represent a form of irreversible brain damage giving a high (94%) risk of death or severe impairment ([15]). However this test has yet to be evaluated appropriately in this setting in a clinical trial.

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