

years.<sup>1</sup> The figure is similar in Britain after less radical resection,<sup>2</sup> and in Britain we may well be dealing mostly with patients whose disease has infiltrated into surrounding tissue.

The early disease in Japan may be different from that in Britain because the survival in Japanese patients (95%) is so much better than that in British patients (70%) that it cannot be explained purely on the basis of removing more affected nodes—and even R1 resection in Japan still produces an 88% five year survival.<sup>2,4</sup> Nearly half of the few early lesions that are encountered in Britain are type III lesions (excavated and mimicking benign ulcers) compared with less than 5% in Japan, which again suggests a different type of early disease.<sup>2,4</sup> Another difference may be that only 16% of Japanese patients have lesions in the cardia (which carry a poorer prognosis stage for stage than those in the body and antrum) compared with 30% in one series reported from Britain.<sup>4,9</sup>

The British Medical Research Council is now running a trial to compare extended lymphadenectomy with conventional gastrectomy. Eligible cases are those up to the Japanese stage III, in which the liver and peritoneum are clear of the disease but the serosa has been breached or there is metastasis to the second tier of lymph nodes<sup>5</sup> (MRC cooperative surgical trial for gastric cancer, protocol, Ninewells Hospital, Dundee). In Japan patients with stomach cancer are younger than in Britain; in addition, the Japanese have less obesity and a lower incidence of arterial disease, and postoperative deep vein thrombosis is almost unknown. There will inevitably be a learning curve for surgeons undertaking the tricky dissection of friable, haemorrhagic fat and lymphatic tissue close to vital structures. It may also be difficult to set aside the extra operative time needed without the service to other patients deteriorating.

Despite these difficulties we need to identify which, if any, patients in Britain will benefit from an extended

lymphadenectomy, and the MRC trial should achieve this. But diagnosis of the condition also needs attention. Some encouraging results have come from the West Midlands, where 48 new cases of gastric cancer were detected among 2820 patients with dyspepsia who were screened; eight of these were early cases (A Jewkes, meeting of the British Stomach Cancer Group, Manchester, 1988). This approach screens only patients with symptoms, but in Japan also most patients have symptoms. The resource implications of repeatedly screening all patients aged over 50 with dyspepsia are daunting, but only with a drive toward early diagnosis and, probably to a lesser extent, better surgery may the results of treating gastric cancer be improved. And the Japanese have shown that improvement is possible.

PETER J MILEWSKI  
Consultant in General Surgery

Withybush Hospital,  
Haverfordwest,  
Pembrokeshire SA61 2PZ

JOHN BANCEWICZ  
Reader in Surgery

University Department of Surgery,  
Hope Hospital,  
Salford M6 8HD

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## Regular Review

### Birth asphyxia and cerebral palsy

#### *Birth asphyxia is hard to define and measure but is rarely the cause of cerebral palsy*

Most laymen and many obstetricians and paediatricians believe that cerebral palsy could be prevented by better obstetric care. Not surprisingly, perinatal brain damage has become an important reason for litigation, which will cause increasing problems for health authorities in the next few years.

#### Terminology

Cerebral palsy is not a single entity. It may be defined as "a disorder of posture or movement which is persistent but not necessarily unchanging, and is caused by a non-progressive lesion of the brain, acquired at a time of rapid brain development." It may be, but is not always, accompanied by other neurological impairments such as mental retardation, cortical vision defects, or epilepsy.

The movement disorder may be predominantly spastic, ataxic, or athetoid; it may affect any number and combination of limbs, head, and trunk; there are many recognised causes and many cases in which the cause is not apparent. The causes of cerebral palsy are traditionally divided into prenatal, perinatal, and postnatal.

Mental retardation may result from perinatal or postnatal factors, but in most children with mental retardation unaccompanied by cerebral palsy the cause is prenatal<sup>1</sup>—for example, chromosomal defects, other anomaly syndromes, or noxious influences as in the fetal alcohol syndrome. People with mental retardation commonly show a delay in motor maturation so that they are late to walk and run, but their pattern of motor development is otherwise normal. The term "cerebral palsy" implies that motor function is not merely delayed but is also deviant—that is, following a course never seen in a normal child.

#### Epidemiology

As cerebral palsy is not a single entity and authors vary in their case definitions and completeness of ascertainment, the data on incidence and prevalence are inexact. Estimates vary from two to four cases for every 1000 births. Mental retardation occurs in about 3.7 children in every 1000 births. There is little evidence of any recent decline in the incidence of either cerebral palsy or mental retardation despite improved obstetric standards.<sup>2</sup>

Recent epidemiological research on cerebral palsy suggests that the importance of perinatal factors in causing childhood handicaps has been overestimated.<sup>3</sup> For example, a review of children with cerebral palsy in Western Australia suggested that only around 8% of cases were associated with (though not necessarily caused by) birth asphyxia.<sup>4</sup>

In Sweden Hagberg has emphasised the importance of distinguishing among the various types of cerebral palsy in epidemiological studies.<sup>5</sup> He found that for infants born at or near term the strength of the relation between perinatal problems and cerebral palsy is strongest for the athetoid type but is weaker for spastic hemiplegia and diplegia and weakest for the ataxic type. In many cases review of both the pregnancy and the birth failed to show any potentially damaging factors.

Nelson and Ellenburg studied some 54 000 deliveries that included 189 cases of cerebral palsy.<sup>6</sup> There were correlations between abnormalities of pregnancy and cerebral palsy, but there was little association with abnormalities of labour. Many of the infants with cerebral palsy had other major malformations that must have antedated labour, and some of these showed evidence of "birth asphyxia." Nelson and Ellenburg concluded that asphyxia might wrongly be regarded as the cause of cerebral palsy in such cases and observed that "we probably do not know what causes most cases of cerebral palsy."

### Perinatal problems and asphyxia

Little was the first to describe cerebral palsy—in 1862. He thought that perinatal factors were the main if not the only cause. Freud questioned this view, pointing out that the abnormalities noted in the perinatal period might sometimes be the result rather than the cause of the subsequent impairment.<sup>7</sup> Modern epidemiological evidence suggests that he was right: perinatal asphyxia is not the only or the commonest cause of cerebral palsy. Nevertheless, this is not to deny that it may sometimes be the cause. The next important question is: How may the cases that are caused by "asphyxia" be identified?

The first difficulty is to define asphyxia.<sup>8</sup> It refers in general terms to the end result of reduced oxygen and nutrient supply to the fetal brain. It is postulated that inadequate gas exchange through the placenta leads to increasing hypoxia, which impairs myocardial contractility and reduces perfusion of the brain. Under the combined influence of hypoxia and reduced perfusion the neurones cease to function and intracellular anaerobic metabolism is stimulated, leading to progressive and eventually irreversible neuronal damage.

Beyond a certain degree of hypoxia and ischaemia the regulatory capacity of the cerebral circulation is damaged and blood flow is not restored even if the perfusion pressure is raised. Studies with Doppler ultrasonography in asphyxiated infants show that there are differences in cerebral haemodynamics between those infants who die or are handicapped and those who survive unscathed.<sup>9</sup>

As no direct measures of asphyxia are available many definitions have been used: fetal heart rate abnormalities detected by monitoring; the results of fetal scalp blood sampling; classic indications of "fetal distress" such as the passage of meconium and fetal bradycardia; obstetric events thought likely to cause "fetal distress;" low Apgar scores; and neonatal acidosis as measured in cord blood samples.

Attempts to relate these measures to subsequent neurological impairment have consistently failed; they do not reliably predict which children will have cerebral palsy. Neither is fetal distress, low Apgar scores, or severe acidosis a necessary or a sufficient condition for cerebral palsy.<sup>10</sup>

In babies born at term the most reliable neonatal indicator of later neurological abnormality is the sequence of events known as hypoxic-ischaemic encephalopathy, which is thought to be a sign that the infant has suffered enough "asphyxia" to cause some brain injury.<sup>11,12</sup> The infant who develops hypoxic-ischaemic encephalopathy may require resuscitation at birth. After an interval that may vary from minutes to many hours the infant shows changes in tone, activity, and patterns of primitive reflexes. There may be hyperalertness or declining consciousness. Fits and abnormal movements develop, and there are abnormalities of sucking, feeding, and eye movements. The infant may deteriorate and die, recover completely, or progress slowly and continue to show abnormal neurological signs.

Some researchers have suggested that neonatal fits are so closely associated with hypoxic-ischaemic encephalopathy

- Cerebral palsy is not a single entity, its incidence is not falling despite improved obstetrics
- Only about 8% of cases are caused by perinatal factors; we don't know what causes most cases
- Asphyxia is hard to define and measure and is rarely the cause of cerebral palsy
- Hypoxic-ischaemic encephalopathy is the most reliable indicator of asphyxia
- Neither traditional clinical signs nor electronic monitoring allow reliable recognition of asphyxia
- In response to a legal claim first establish the diagnosis then look at the records

that they may be used as an outcome measure of the quality of obstetric care,<sup>13</sup> but fits are not always associated with the encephalopathy.<sup>14,15</sup> Intracerebral haemorrhage in the early neonatal period is also well recognised as a cause of abnormal neurological findings and may be related to hypoxic-ischaemic encephalopathy or to a traumatic delivery, but it may also arise spontaneously.<sup>16,17</sup> Other possible causes such as malformations or metabolic diseases should be considered.

The severity of hypoxic-ischaemic encephalopathy is a far more reliable predictor of adverse outcome than a low Apgar score or acidosis. The encephalopathy is usually associated with acidosis at birth but not with excessively severe acidosis. Most infants with very severe acidosis at birth survive unscathed.<sup>18</sup> Mild hypoxic-ischaemic encephalopathy, resolving within 48 hours, is usually associated with a normal outcome, but the more severe forms of hypoxic-ischaemic encephalopathy are associated with an increasing risk of death or handicap.<sup>12,19</sup>

In summary, if a full term infant has an uncomplicated neonatal course with no evidence of hypoxic-ischaemic encephalopathy or neurological disturbance then birth asphyxia is unlikely to be the cause of any disability or defect.

Asphyxia in preterm infants is different as the fragile state of the infant at birth and the common need for intensive care because of respiratory complications greatly complicate the analysis of data on handicap in relation to outcome. Most adverse outcomes in preterm babies seem to have a multi-

factorial aetiology, although authors differ on exactly which factors play the greatest part in causing cerebral palsy.<sup>20,21</sup>

### When does asphyxia occur?

Lay people tend to assume that asphyxia occurs in the last part of labour and that prompt delivery would avoid any problems. Nevertheless, the fetus seems well able to withstand sudden acute stressful and potentially asphyxiating events such as prolapse of the umbilical cord or abruption of the placenta—the outcome of these events is generally good, even when the infant has been born with a low Apgar score and has needed resuscitation.<sup>22</sup>

If asphyxia is an acute event we might expect a close relation between the speed of delivery after recognition of fetal distress and adverse outcome. But Painter and others were disappointed to find that, although there was some correlation, prompt intervention for fetal distress failed to prevent an adverse outcome.<sup>23</sup>

The results of animal studies suggest that a single episode of very severe asphyxia has an all or none effect: the result is either full recovery or death. Repeated episodes of sublethal asphyxia produce lesions in the brains of fetal monkeys which approximate those seen in people with cerebral palsy. Thus asphyxia may sometimes be a subacute or chronic process starting in pregnancy rather than an acute insult occurring in labour. Many authors have noted that brain damage occurring in utero may lead to difficulty with resuscitation—and an erroneous diagnosis of birth asphyxia may then be made, as first postulated by Freud.<sup>24</sup>

Antepartum hypoxia may occur in two ways in man, being associated with either prenatal brain damage or an increased vulnerability to intrapartum stress. Firstly, there are well described cases in which the mother has suffered severe shock and hypotension or hypoxaemia—caused, for example, by carbon monoxide poisoning or anaphylaxis—and the fetus suffers cerebral damage in utero.<sup>25</sup> Secondly, antepartum hypoxia may arise with intrauterine growth retardation. These fetuses have reduced oxygen and high lactate concentrations and other evidence of chronic hypoxia in utero.<sup>26</sup> In most cases, however, there is no evidence of prenatal hypoxaemia, growth retardation, or other adverse events in pregnancy. Clearly, much remains to be learnt about the true nature of “asphyxia.”

### How may asphyxia be recognised?

Whatever the precise cause of asphyxia, might the ability to identify warning signs in labour enable the obstetrician to avoid an adverse outcome or at least to reduce the severity of the asphyxial damage? Unfortunately, neither the traditional signs of fetal distress nor the use of electronic monitoring permit the reliable recognition of the asphyxiated infant during labour.

Many obstetricians have used neonatal fits as an indicator of adverse outcome, although in future research programmes accurately diagnosed hypoxic-ischaemic encephalopathy would be preferable. Electronic monitoring may reduce the number of babies suffering neonatal seizures, though it does not seem to reduce the number of children with cerebral palsy. Even when optimal protocols of care are followed both intrapartum death and neonatal fits occur without apparent warning.<sup>27</sup> Although suboptimal care during labour is associated with an increased rate of neonatal seizures, most neonatal seizures are not preceded by poor care and most errors of care are not followed by seizures.<sup>28</sup>

The issue may be reduced to the question: What are the sensitivity, specificity, and positive predictive values of any given abnormality observed during labour in relation to

neonatal fits, hypoxic-ischaemic encephalopathy, or cerebral palsy? Given the substantial investment in fetal monitoring equipment and the immense medicolegal importance of the issue, there are remarkably few data. But clearly fetal monitoring is far from being an exact science,<sup>29</sup> and the obstetrician therefore must exercise clinical judgment in interpreting the records.

The process of “asphyxia” is complex and does not invariably result in the fetus showing signs of distress that may be recognised with present day methods. Even when fetal distress is recognised and leads to speedy delivery the damage may already have been done. This is not to suggest that the obstetrician should not respond to evidence of fetal distress; but when the infant is found to be suffering from cerebral palsy it is unwise to assume that more prompt action would have avoided it.<sup>30</sup>

### Other causes of cerebral palsy

Vascular and haemorrhagic lesions of the fetal or infant brain account for many cases of cerebral palsy. Ultrasonography has shown that porencephalic cysts may develop before birth, often in the territory of a single artery, suggesting that they are caused by a vascular occlusion.<sup>25</sup> These lesions are particularly associated with hemiplegic cerebral palsy. Periventricular cysts have also been shown in utero and are associated with spastic diplegia. Infarction may be caused by embolism,<sup>31</sup> particularly in the surviving member of a monovular twin pregnancy in which the other twin has died or is a fetus papyraceous.<sup>32,33</sup>

Anomalies of cortical development and metabolic disorders must also be considered as causes of cerebral palsy, and some cases have a familial basis<sup>34</sup> and may be inherited as a dominant or an autosomal recessive characteristic or through a sex linked gene.<sup>35</sup> Cerebral palsy may be seen in children with chromosomal and other dysmorphic syndromes and in children with congenital infections, but often we must agree with Nelson and Ellenburg that we do not know the cause.

### Response to litigation

When parents claim compensation after the birth of a handicapped child it is essential firstly to establish a diagnosis. A diagnosis of “brain damage at birth” is not acceptable. Cases must be identified in which the main problem is mental retardation without cerebral palsy, and other diagnoses such as chromosomal or dysmorphic syndromes should be excluded. Major anomalies of other organs suggest a prenatal onset of the child's problems. Deafness and blindness in isolation are rarely if ever the result of birth asphyxia, and other causes should be sought.

Next the neonatal records should be inspected. If there was no evidence of moderate or severe hypoxic-ischaemic encephalopathy it is unlikely that asphyxia was the cause of the child's disability. Other causes of fits or abnormal neurological behaviour should be considered.

Even if the infant is thought to have shown signs of hypoxic-ischaemic encephalopathy, a defence may be based on the evidence that asphyxia is often subacute or chronic and that the abnormal signs in labour are not reliable, provided that the management decisions fell within the accepted bounds of standard practice.

### Can litigation be avoided?

Hypoxic-ischaemic encephalopathy and neonatal convulsions occur even in the best units, and the incidence of hypoxic-ischaemic encephalopathy does not seem to have fallen in the past decade.<sup>36</sup> Whenever neurological abnormali-

ties occur in the neonatal period an accurate diagnosis must be sought, using all available methods. Obstetricians, paediatricians, general practitioners, and nursing staff should refrain from describing "birth damage" as the cause of a child's disability without carefully considering the facts. Once this idea has been implanted in the parents' minds their thoughts will turn to litigation even if a more experienced person subsequently offers a contrary opinion.

An important motive for litigation in some cases is to discover "exactly what went wrong." Much distress may be avoided if obstetricians and paediatricians jointly examine the records of the labour and explain to the parents what happened and why various decisions were made. There is no reason to think that this practice would increase the litigation, and it might well have the opposite effect.

Another motive for litigation is anger with the staff. Patronising remarks by nursing or medical staff, failure to listen carefully to the mother's distress or anxiety in labour, and any suggestion that the mother herself was to blame for a bad outcome are all common complaints among parents who undertake legal proceedings.

Another reason for turning to litigation is the increasing dissatisfaction of parents with the services provided for disabled children and adults by the NHS, education authorities, and social services. Parents think that they owe it to their child to protect his or her future by obtaining a large sum in damages. The "schedule of damages" produced for the plaintiff, although often inflated for obvious reasons, highlights the true costs of disability and rehabilitation.

### Defects with the adversarial legal system

The present adversarial legal system for settling negligence claims benefits a fortunate few, but its overall effects are damaging. Apart from encouraging defensive obstetrics, it has three other disadvantages.

Firstly, parents justifiably claim that the existing services are inadequate. If they succeed in proving negligence they obtain a sum of money that is sufficient to provide all the care and equipment that any disabled person could require. But resources are finite, and the money allocated to settling these claims could be used to bring about the substantial improvements needed in services for all disabled people.<sup>37</sup> Better services might reduce the justification for future litigation.

Secondly, the settlement of claims is by a single payment, which is calculated on the basis of predicted life expectancy and the pattern of care that might be appropriate for disabled children when they reach adult life. Not only is it absurd to predict anyone's life expectancy but also the statistical basis on which these "expert" guesses are made is often fragile or non-existent. This system must result in a financial windfall for some families and inadequate provision for others.

The third problem with the adversarial system is that the issues of causation that have to be considered are extremely complex. A court is not the place to debate scientific uncertainties; furthermore, any attempt to do so is extremely costly.

If the proposal to transfer all liability to health authorities is adopted parents may be even more likely to start legal proceedings. If the present trend continues each health authority may be faced with a six or seven figure claim every 12 to 18 months with respect to asphyxial brain injury causing cerebral palsy. The financial burden will have a serious impact on other services. A solution must be sought as a matter of urgency. One proposal has been a "no fault" scheme limited to perinatally acquired disabilities. Even this would present formidable difficulties, but the present problems cannot be allowed to continue indefinitely.

DAVID M B HALL

Consultant in Community Child Health,  
St George's Hospital,  
London SW17 0QT

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