Do concepts of causes and prevention of cerebral palsy require revision?

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OBJECTIVE: My purpose was to explore the criteria of The American College of Obstetricians and Gynecologists (Technical Bulletin No. 163) for perinatal asphyxia to be linked to subsequent cerebral palsy.
STUDY DESIGN: Analysis of four cases of intrapartum fetal insults with subsequent cerebral palsy and a literature review are presented.
RESULTS: All of the four cerebral palsy cases had sufficient intrapartum causes of cerebral palsy, yet none fulfilled The American College of Obstetricians and Gynecologists' linkage criteria. Complications in the cerebral palsy cases were as follows: maternal intrapartum cardiac arrest, fetal skull fracture with brain infarct, intrapartum fetal stroke, and a newborn delivered after uterine rupture with only central nervous system defects. There are no well-done laboratory or clinical studies that unequivocally support the "criteria" that umbilical artery pH must be <7.00 or the requirements of Apgar score <3, hypoxic-ischemic encephalopathy, and multiple organ dysfunction. Apparent exceptions to these criteria occur.
CONCLUSIONS: The American College of Obstetricians and Gynecologists Technical Bulletin's criteria for cerebral palsy linkage and the role of parturition in cerebral palsy should be reevaluated. A rebirth of obstetric enthusiasm for cerebral palsy research, teaching, and treatment needs to occur. (Am J Obstet Gynecol 1995;172:1830-6.)

Key words: Cerebral palsy linkage, perinatal asphyxia

Cerebral palsy, a disorder of movement and posture caused by a lesion or injury of the immature brain, is the leading cause of childhood deformity and the second leading cause of severe mental retardation.[1, 2] There are more than 300,000 persons with cerebral palsy in the United States. The American College of Obstetricians and Gynecologists (ACOG) in its February 1991 Committee Opinion and in the ACOG Technical Bulletin No. 163, January 1992 *(Fetal and Neonatal Neurologic Injury),* proposed four "must be" requirements before a plausible link could be made between perinatal asphyxia and consequent cerebral palsy.[3] They are as follows: (1) an umbilical artery pH <7.00; (2) an Apgar score of 0 to 3 for longer than 5 minutes; (3) neonatal neurologic sequelae, such as seizures, coma, or hypotonia; and (4) multiorgan dysfunction, involving the cardiovascular, gastrointestinal, hematologic, pulmonary, or renal system. These four requirements have international appeal to obstetricians because, when they are applied, few cases of cerebral palsy appear to be due to perinatal asphyxia. This article explores the validity for such criteria.

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Case reports

The following cases are not typical of neonatal hypoxic-ischemic encephalopathy but demonstrate that intrapartum insults can cause cerebral palsy, in the absence of the above-described criteria for "linkage." The charts reviewed were in litigation.

Case 1. Intrapartum fetal stroke. At age 4 this boy had spastic hemiplegia. His primigravid mother had entered the hospital in early labor. His initial fetal heart rate (FHR) pattern showed loss of beat-to-beat variability, which responded with deceleration after application of a scalp electrode. He was delivered spontaneously after 8 hours of labor and weighed 3110 gm. His Apgar scores by a neonatologist (present because of meconium-stained amniotic fluid) were 6 at 1 minute and 8 at 5 minutes. By 9 minutes he required respiratory and cardiovascular support, and he had seizures at age 8 hours. Although the absent beat-to-beat variability suggests fetal brain stress or injury before labor, repeated computed tomographic (CT) scans showed that he had an infarct of the area served by the middle cerebral artery, probably during parturition. This diagnosis was supported by subsequent magnetic resonance imaging (MRI) studies.[4]

Case 2. Intrapartum maternal cardiac arrest. At age

Table I. Confusing aspects of neonatal acidemia

Subject	*Comment*
Cerebral palsy in fetal monkeys	May occur without acidemia
Human cerebral palsy without acidemia	Case report
Human central nervous system complications	Only with fetal metabolic acidemia
Severe fetal acidemia	Occurs in normal human neonate
Intrapartum recovery of fetal acidemia	Perhaps 6 hr for metabolic acidemia
Maternal pH most important determinant of fetal pH	Seldom clinically measured

3 years this boy had spastic quadriplegia. His mother, a multigravid patient who had a normal pregnancy, had entered the hospital at 38 weeks of pregnancy in active labor. While being given an epidural anesthetic, she had a cardiac arrest. The FHR was <60 beats/min. It took nearly 20 minutes after the initial hypotensive event to obtain acceptable maternal vital signs. By this time his FHR was considered to be normal. Labor was allowed to continue, and he was born 2 hours later. His weight was 2560 gm and he had Apgar scores of 6 at 1 minute and 7 at 5 minutes. On day 1 he was considered normal, but on day 2 he had seizures and was subsequently diagnosed as having hypoxic-ischemic encephalopathy. Post partum his mother had a brief period of posthypoxic mental confusion.[5]

Case 3. Traumatic delivery. A 4-year-old girl was diagnosed as having mild right spastic hemiplegia. Her multigravid mother was undergoing vaginal delivery after a previous cesarean section and was taken to the operating room because of failure to deliver after 2 hours of second-stage labor. There was difficulty in extracting the fetal head at cesarean section. Her Apgar scores were 8 at 1 minute and 9 at 5 minutes. In the nursery a depressed skull fracture of the left parietal bone was noted. CT scans showed an infarct in this same cerebral area; later scans confirmed fibrosis and cysts. While in the nursery the newborn had a questionable seizure but was discharged home on the fourth day of life.

Case 4. Newborn hypoxic-ischemic encephalopathy with neonatal death. A 3845 gm term newborn was delivered by "crash" laparotomy with Apgar scores of 1 at 1 minute and 2 at 20 minutes. Her mother was undergoing vaginal delivery after a previous cesarean section and while in the delivery room had a uterine rupture with the fetus and placenta being extruded into the peritoneal cavity. The newborn was delivered within 15 minutes of the rupture. An umbilical vein pH was 6.95. The newborn had no other organ defects but was declared "brain dead" at age 49 hours and was removed from life support.

Comment

The ACOG "must be" requirements for cerebral palsy to be linked to intrauterine asphyxia are briefly discussed in the context of the cases reported.

Fetal acidemia. The poor predictive value of newborn blood pH for subsequent cerebral palsy has been repeatedly noted.[6] In 1959 Kaiser[7] found that fetal acidemia could be entirely incidental and without pathologic significance; acidemia could be a response to exogenous stimulus such as maternal acidemia and well within the adaptive capacities of the fetus or it could reflect a serious morbid fetal state. Only an estimation of the newborn's condition can make a proper evaluation of acidemia's significance.[7] Table I lists some of the confusing aspects of newborn acidemia.

The ACOG Committee Opinions (No. 137 and No. 138), in discussing the "criteria," use the term *proximate* without definition. If proximate is longer than a few hours, then it is only an assumption that a fetus is unable to correct a labor-induced metabolic acidemia before its birth.[8] Maternal pH values are the single most significant determinant of fetal pH values, and maternal acidemia alone can produce fetal blood gas values indicative of severe asphyxia in an otherwise healthy newborn.[9] Beard and Simons[10] reported that 19% of the cases of fetal acidemia were due to maternal acidemia. Maternal pH should be determined in cases of severe fetal acidemia.

Low Apgar scores. In my case 2 the defense experts testified that despite the mother's cardiac arrest with cerebral symptoms, it was only a coincidence (odds of approximately 1:20,000,000) that her child had cerebral palsy. His normal Apgar scores (according to the ACOG criteria) eliminated the possibility that intrapartum asphyxia caused cerebral palsy.

The requirement for low Apgar scores excludes linkage of intrapartum asphyxia with most cases of cerebral palsy. In a discussion of why hypoxic-ischemic encephalopathy occurs in neonates who were not depressed at birth, Hull and Dodd[11] noted "What seems more probable is that these infants suffered a cerebral insult some time before birth which was not detected by the CTG (fetal monitor) and was only temporary." In "near-miss" sudden infant death syndrome leading to cerebral palsy, Constantinou et al.[12] described a period of "near normality" lasting up to 50 hours after asphyxia-induced brain damage. Normal Apgar scores can be expected in some neonates who are destined to have intrapartum asphyxia-induced cerebral palsy. Unless

Table II. Significance of neonatal seizures

Year	*Authors*	*Etiology*	*Related to cerebral palsy**
1861	Little	Asphyxia—term fetus	Yes
1872	Osler	Teething	Yes
1882	Freud	Extrauterine	Uncertain
1976	Sarnat and Sarnat	Asphyxia	Yes, in stage 3
1979	Eriksson and Zetterstrom	Hypoxia	Uncertain
1981	Dennis	Suboptimal care	Uncertain
1985	Brann	Asphyxia	Perhaps
1985	Niswander et al.	Suboptimal care	No
1985	Keegan et al.	Asphyxia	Uncertain
1987	Minchan et al.	Hypoperfusion?	Uncertain
1989	Patterson et al.	Hypoperfusion?	Uncertain
1990	Murphy et al.	Substandard care	Yes
1994	Richmond et al.	Fetal distress	Uncertain

*Author's interpretation.

Table III. Conflicts with "diving reflex" concept

Event	*Comment*
Human fetal brain oxygen requirement	Much greater than that of experimental animals
Diving reflex itself	May be transitory in growth-retarded fetus
Multiorgan dysfunction	Only central nervous system affected in some delivered at uterine rupture
No supporting laboratory data that it always occurs	Multiorgan dysfunction only a clinical impression
Multiorgan dysfunction not present in all cases of "near-miss" sudden infant death syndrome	Applies to asphyxiated fetus as well

the Apgar score is below 4 for longer than 20 minutes, it is not predictive of subsequent cerebral palsy.[13]

Neurologic sequelae. The cause of seizures in term neonates is often disputed as is their relationship to consequent cerebral palsy (Table II). In 1976 Sarnat and Sarnat[14] defined three stages of neonatal hypoxic-ischemic encephalopathy and their correlation with subsequent cerebral palsy. Whereas mild encephalopathy had previously been considered to have a good outcome, in 1994 Rosenbloom[15] challenged Sarnot and Sarnot's widely accepted concepts by reporting that "mild hypoxic-ischemic encephalopathy" could be associated with subsequent athetoid cerebral palsy. Infants with neonatal seizures are 50 to 70 times more likely to have severe cerebral palsy and 18 times more likely to have epilepsy.[16] Freeman and Nelson,[17] as well as Grant et al.,[18] suggested that whereas neonatal seizures are associated with both intrapartum asphyxia and cerebral palsy, it does not follow that "asphyxia is associated with cerebral palsy."

Multiorgan dysfunction. None of our four cases had evidence of multiorgan dysfunction. Central to the theory of multiorgan dysfunction is the concept that the brain is spared during asphyxia (the diving reflex). It is assumed that if fetal asphyxial brain damage occurs, then other organs must also be impaired.[19, 20] Whereas this is one of the most agreed-on generalities in all of clinical perinatology, there appears to be no laboratory support for this multiorgan dysfunction hypothesis. In "near-miss" sudden infant death syndrome leading to cerebral palsy, some surviving infants had no multiorgan dysfunction.[12] Table III lists some of these contradictory observations. Hence, all four of the ACOG Technical Bulletin No. 163 requirements" can be challenged.

In the early development of electronic fetal monitoring and fetal scalp sampling techniques, it was recognized that these were only indirect indicators of fetal asphyxia and not necessarily without error.[21] Because of these errors in diagnosis, ACOG committees[22] have proposed that the term *fetal distress* be expunged. Similarly, a joint committee of the Little Foundation and World Federation of Neurology has proposed that both *hypoxic-ischemic encephalopathy* and *asphyxia* be limited to neonates where all of "ACOG's requirements" linkage criteria apply.[23]

Gaffney et al.[24] reported that 9 of 10 human infants with prenatal brain lesions had ominous or suspicious FHR changes during labor. Fetal brain lesions can be expected to induce FHR abnormalities[25] (but not necessarily asphyxia) in the otherwise well fetus. Blair and Stanley suggested that brain lesions may cause asphyxia. I could not accomplish asphyxia from induced brain lesions in five fetal sheep (unpublished observations).

Nelson noted that neuroimaging techniques suggest that in utero strokes are a common cause in children with hemiparetic cerebral palsy. Such cases may represent 30% of all cerebral palsy. In Allan and Riviello's review of in utero strokes, 10 of 27 cases had compli-

Table IV. Reports suggesting cerebral palsy despite absent linkage criteria

Authors	*Findings*
Brann	Normal Apgar scores with hypoxic-ischemic encephalopathy
Sykes et al.	Central nervous system damage sometines without neonatal depression
Ment et al.	Perinatal cerebral infarction as an isolated asphyxial event
Kubli et al.	Reversible acidemia in cases of intrapartum asphyxia
Young et al	Transient reversible fetal asphyxia
Hull and Dodd	Intrapartum recovery probably occurs
Constantinou et al.	Postasphyxia window of near normality in "near-miss" sudden infant death syndrome cases with subsequent cerebral palsy
Rosenbloom	Mild neonatal encephalopathy with subsequent cerebral palsy

Table V. Obstetric techniques for future cerebral palsy studies

Technique	*Purpose*
Improved fetal monitoring	Detect fetal stress versus fetal distress
New collaborative study	Use data from current techniques for analysis
Maternal tranquility (Freud)	Reduce fetal stress
Drug protection against asphyxia or for those at risk of cerebral palsy	Prevent cerebral palsy
Analysis of cerebral palsy rate for elective cesarean section versus vaginal birth	Determine importance of intrapartum insults in cerebral palsy
Newer neuroimaging techniques for depressed neonates or those having had fetal distress	Ascertain time of insult producing cerebral palsy

cated labors, 22 of 27 had focal seizures, and, as in my case 1, all had abnormalities on scans. In utero strokes may reflect aberrant fetal blood vessels, embolic or thrombotic disorders, and perfusion failures from serious hypoxemia, involving the middle cerebral or internal carotid artery.

Hope and Moorcraft (1990), Murphy et al. (1990), Glynn and Leon (1992), Rosen and Dickinson (1992), Gaffney et al. (1994), and Hull (1994) have all challenged the current concept that intrapartum asphyxia is the cause of cerebral palsy in $<9\%$ of cases. These authors suggest an upper range of 19% to 30%. ACOG's requirement for low Apgar scores eliminates linkage for cases such as my case 1 and case 2, despite intrapartum causes of cerebral palsy. The ACOG bulletin did not include a classification for trauma (my case 3). Uterine rupture with subsequent cerebral palsy, as in case 4, is now more common with widespread use of vaginal delivery after a previous cesarean section. These cases have not supported the "must be" criteria (Phelan). Table IV lists articles supporting the concept that intrapartum events may result in cerebral palsy, despite absence of some of the above-listed criteria.

The recent analysis suggesting that intrapartum asphyxia is the cause in $<9\%$ of the cerebral palsy cases may be attributable to changing criteria for the diagnosis of intrapartum asphyxia, application of the stringent requirements of the ACOG bulletin, and the use of more sophisticated statistical analysis. As a result, an outpouring of articles now suggests that obstetricians are helpless to prevent most cases of cerebral palsy and that neonatologists or pediatricians are likewise unable to alter the course of the childhood abnormality.[2] While apparently good news to some,[2] as Kitchen argued, "to accept this view absolves the perinatologists of blame, but is an anathema to those striving to improve outcome by ever more strenuous efforts to optimize care."

In addition to the wish to eliminate cerebral palsy, an incentive for obstetric research may be the high malpractice awards to children with cerebral palsy. These awards are not necessarily due to trial by jury or contingency fees for attorneys, for, in their absence, Irish obstetricians have experienced cerebral palsy judgments comparable to those awarded in the United States (MacDonald). Palmer suggests that the problem probably will never be resolved until the causes of cerebral palsy are determined.

For a positive outlook, there are research opportunities (Table V). Clinical diagnosis would be improved by diagnosing the various degrees of fetal *stress* rather than always using the term fetal *distress*. The concept that prenatal events could render the fetal brain more vulnerable to intrapartum injury was suggested by Little more than 130 years ago. It has resurfaced at regular intervals and has laboratory support. Such a vulnerable fetus could have intrapartum brain damage without associated cardiovascular or acid-base changes, thus explaining failures of current fetal monitoring techniques.

There are no well-done laboratory or clinical studies that support the ACOG bulletin's stringent concepts for any one of its four requirements (severe acidemia and

depression, encephalopathy, and multiorgan defects), much less that all four "must be" present. Despite their popularity, these ACOG requirements should be revised and replaced by those based on well-done clinical and laboratory studies. As those responsible for the newborn's good health, just as they have encouraged tort reform, obstetricians should encourage society's efficient and compassionate support of those cerebral palsy victims of in utero insults. A rebirth of enthusiasm for the in utero detection, prevention, and treatment of cerebral palsy needs to occur.

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A complete list of references is available from the author on request.

Discussion

Dr. Julian. T. Parer, San Francisco, California. Dr. Goodlin has challenged the criteria published by ACOG for conditions that must be present before a plausible link can be made in the relationship between perinatal asphyxia and neurologic deficit in an individual patient. Dr. Goodlin uses the wording ". . . proposed four 'must be' requirements . . ., whereas the ACOG Technical Bulletin uses slightly different terminology: ". . . all of the following criteria must be present before a plausible link can be made" and in another area "unless all of the characteristics are present . . ., a plausible link . . . is lacking." I think an examination of the terminology is important and all the more so because Dr. Goodlin admits that his illustrated cases were in litigation, and legal professionals are self-confessed wordsmiths.

Dr. Goodlin's approach is to describe four cases that have exceptions to the ACOG criteria, and he concludes that the criteria should be revised and replaced by those that are based on well-done clinical and laboratory studies. In another part of this article he states that the criteria ". . . should probably be revised." Dr. Goodlin's four cases consist of examples of fetal damage in utero through a variety of causes, none of which are progressive asphyxia brought about by the accumulation of an oxygen debt caused by decreasing uteroplacental or umbilical placental function. Thus his first case consists of an infarct of the fetal middle cerebral artery, the second is a maternal cardiac arrest that was reversed before delivery of the baby, the third is a traumatic delivery resulting in a skull fracture during head extraction at a difficult cesarean section, and the fourth is fetal asphyxia as a result of a uterine rupture, which presumably represents a catastrophic stepwise reduction in both uterine and umbilical blood flow. None of our screening criteria for fetal asphyxia are expected to predict the first three events any more than they can predict a catastrophic gunshot wound to the pregnant uterus.

The strongest case made by Dr. Goodlin is the criticism of the fourth criterion, that is, the requirement for multiorgan system dysfunction. Dr. Goodlin states that in none of the four cases was there multiorgan dysfunction, although no data are presented. It is difficult to agree with him that case 4, the extremely depressed and acidotic fetus born after uterine rupture and apparently vegetative for 2 days before life support was

turned off, did not have substantial organ damage that may not have been detected in the brief life of the baby. In any event I tend to agree with Dr. Goodlin that of all the criteria this appears to be the least well documented in prospective series.

However, the Committee Opinion is not without some value, and a restatement of the criteria may salvage some of this value. The following are possibilities.

To invoke persistent intrapartum global fetal asphyxia lasting until delivery as a cause of subsequent cerebral palsy the following apply: (1) FHR pattern with absent variability, (2) umbilical arterial blood pH <7.0 and usually <6.8, (3) umbilical arterial base excess <15 mEq/L and usually <20 mEq/L, (4) Apgar score <3 at 5 minutes and usually ≤ 3 at 10 minutes, (5) abnormal neurologic signs in the immediate newborn period and usually seizures in <12 hours, and (6) multiorgan damage, which is often seen in these newborns (e.g., cardiac, renal, coagulopathy).

A corollary of these criteria, that is, factors that virtually rule out persistent intrapartum global fetal asphyxia lasting until delivery as a cause of subsequent cerebral palsy, include the following: (1) normal FHR pattern with normal FHR variability until a few minutes before birth, (2) normal umbilical arterial blood gas values (pH >7.10, base excess >-12 mEq/L, (3) Apgar score >7 at 5 minutes, and (4) absence of neurologic signs in the first day of life.

Dr. Goodlin has provided a service by his critical approach to this subject and has pointed out both flaws in the ACOG committee's recommendation and defects in our knowledge. His plea in his article for further specific investigative work is particularly pertinent.

I would appreciate his response to the question of whether progressive, nonacute asphyxia causing fetal brain damage may better fit the ACOG criteria.

Dr. Robert Resnik, San Diego, California. Dr. Goodlin has presented four cases and a literature review as a challenge to current ACOG guidelines, which outline criteria viewed prerequisite to implicating birth asphyxia as a cause of neonatal hypoxic encephalopathy. He refers to a Committee Opinion and a Technical Bulletin published in 1991 and 1992, respectively, and it should be noted that similar guidelines were reaffirmed in a Committee Opinion of ACOG in April 1994.[1] Clearly, the etiology of neonatal brain dysfunction and cerebral palsy remains an enigma. Dr. Goodlin's four cases undoubtedly represent examples of intrapartum asphyxia or trauma resulting in brain damage, but the role of intrapartum factors in the genesis of these disorders has never been questioned—only the extent to which brain damage is attributable to intrapartum factors or other earlier pregnancy complications.

Although the guidelines recommended by ACOG may be imperfect, there is a considerable body of evidence that minimizes the role of the intrapartum period as a major cause of neonatal brain damage. A National Institutes of Health review reported in 1985, which took into account all available epidemiologic data reported up to that time, concluded that the large majority of cases of cerebral palsy were of unknown cause and that intrapartum factors were a minor contributor.[2] These conclusions were confirmed by Blair and Stanley,[3] who reviewed all cases of children born with spastic cerebral palsy in Western Australia between 1975 and 1980 and concluded that only 8% of the cases could be attributed to intrapartum asphyxia.

Obstetricians, neonatologists, pediatricians, and developmentalists have long sought that measure or device which would help to identify intranatally, or at the time of birth, those infants at risk for brain damage. FHR monitoring,[4, 5] umbilical artery pH, and Apgar scores[6] have lacked both sensitivity and specificity in predicting disease and have not lived up to their promise, because of the fact that they are based on the erroneous assumption that intrapartum asphyxia is the leading cause of neonatal brain damage. In point of fact, the preponderance of studies published in the last 15 years suggest that it is a relatively minor contributor.

The predominant neuropathologic finding associated with cerebral palsy is periventricular leukomalacia, a lesion initially associated with coagulation necrosis in the white matter lateral to the ventricles, followed later by cavitary lesions. The fact that this lesion may occur well before the onset of labor has been documented by Bejar et al.,[7, 8] who used neonatal brain ultrasonography to document the role of prematurity, amniotic fluid infection, and twin-to-twin transfusion as significant etiologic factors. These studies have provided new insight into the etiology of neonatal brain disease and cerebral palsy and emphasize the important role of antepartum factors.

The ACOG guidelines are imperfect but serve an important role. As they state, most cases of cerebral palsy caused by intrapartum asphyxia are associated with a pH <7.0, a low Apgar score at 10 to 20 minutes, and multi–organ system dysfunction. Conversely, the literature clearly supports the fact that most neonates with a pH >7.0, normal Apgar scores at 10 to 20 minutes, and even neonatal seizures are entirely normal at follow-up. Dr. Goodlin is surely correct when he calls for more investigation. However, our current knowledge in this area is just emerging from the dark ages, and the current ACOG guidelines seem quite defensible on the basis of what is currently known.

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Dr. Ralph W. Hale, Washington, D.C. I don't believe it is necessary to comment on the scientific rebuttal to some of Dr. Goodlin's statements because they have been adequately presented by Dr. Parer and Dr. Resnik. I would like to point out that the very first statement in the Technical Bulletins of the College is that these are not standards, these are educational documents that are designed to help educate the Fellows in a wide variety of areas. The references that are reported in these are evidence based—evidence based in that they are reviewed carefully and are researched according to the Canadian system. This is a very effective system on which to base the validity of the references. Obviously, no document can be all inclusive. The ACOG Technical Bulletins and Committee Opinions don't attempt to be all inclusive but do attempt to be a guideline that is educationally based, which physicians can use to aid them in their practice. There obviously will be exceptions, and I think that the exceptions that Dr. Goodlin presented were very appropriate.

Dr. Raymond J. Jennett, Phoenix, Arizona. One of the advantages of belonging to this Society has been to come and hear the iconoclastic wisdom of Dr. Goodlin, for which we should all be grateful. He has pointed out that Dr. Little listed prolonged labor as one of the associated factors of cerebral palsy. I don't know how Dr. Little could have peered into the late twentieth century and known that it was again the fashion to allow prolonged labors under the title of active labor management, which too often means pushing the fetus with large doses of oxytocin until it decompensates and then adding a forceps delivery or a vacuum extraction. We are not training our residents that there is such a thing as an abnormal pelvis, that there is such a thing as an abnormal position, and that many of these things can be additive and impair cerebral circulation. Strokes can happen, vascular impairment can happen, and all of these things should be looked at and not just in the matter of asphyxia and acidosis.

Dr. Goodlin (Closing). One of the things that is happening with vaginal birth after cesarean section and uterine rupture is that there are now enough cases available of cerebral palsy in infants who were delivered by emergency laparotomy for a registry. The word is that these cases of cerebral palsy, in which the time and duration of the insult were known down to seconds, are not following ACOG's Technical Bulletin dicta.[1] Because the ACOG's Technical Bulletin is widely quoted, I have noted it. These same rules have been adopted by the handbooks that pediatricians and obstetricians use. The idea that prenatal events increase the vulnerability of the fetal brain so that the least little thing can cause brain damage without signs of fetal distress is certainly one that should be explored.

There can be a period of newborn "normality" after asphyxia. Before 1970, it was believed to occur for the fetus, and then a "consensus happened" and it doesn't occur anymore. However, it does occur, and so the Apgar scores can be normal after intrapartum asphyxia.

There is a cerebral palsy that is relatively mild, and when you look back at the record, the labor and neonatal course were both normal. Consensus states that this cerebral palsy had to be induced prenatally. Conversely, severe cerebral palsy occurs 10% to 20% of the time. There is severe fetal distress and the neonatal course is very abnormal. These cases are to be considered of intrapartum origin. Low has been writing about neonatal asphyxia for more than 25 years. He and his associates have described cases of minimal asphyxia with minimal brain damage.[1] It seems entirely conceivable to me that the mild cases of cerebral palsy could also have been caused by mild intrapartum asphyxia when the brain had become vulnerable. I don't think our standard criteria are able to make these diagnoses. To answer Dr. Parer, I don't believe that asphyxia has to be progressive, as shown by the ruptured uterus data.

Dr. Resnik discussed periventricular leukomalacia. Obstetricians continually talk about it as occurring only before 34 weeks. We now know that periventricular leukomalacia occurs at term, so everything that we hang our hat on has contrary opinions.[2]

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