...beaten the mother may elicit traumatic memories or flashbacks for children.

IMPLICATIONS AND CONCLUSION

Pediatricians have long considered parental and family dysfunction and child and sexual abuse within their clinical purview. We have not yet, however, focused on the needs of children who witness violence. We no longer can afford to ignore this problem. Children are being traumatized and are learning maladaptive lessons about the use of violence in relationships. From a policy perspective, it is important that data be generated to provide accurate information regarding the number of children who witness domestic violence. This could be done, for example, by tabulating police reports of domestic violence. We also need further study to understand how children are affected by witnessing violence, especially why some children seem to be more resilient to its effects than others. At a clinical level, all care givers, parents, health professionals, and police need to work together to identify children who witness violence and to ensure that they get the counseling services and interventions they need. These services represent secondary prevention by breaking the cycle of children who witness violence and later engaging in violent behavior as adults. Training and reimbursement for pediatricians and other child clinicians should have high priorities. Reimbursement for these services in the present health care climate may be difficult, because this intervention, like many other pediatric interventions, potentially will accrue savings for social service and/or education and not for the health sector. Finally, we need to focus our efforts on primary prevention: educating our patients, supporting stronger sanctions for batterers, and teaching children that intimate relationships don't have to be violent.

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ACKNOWLEDGMENTS

This work is supported in part from grants from the Maternal and Child Health Bureau (MCJ 259169) and the Massachusetts Committee on Criminal Justice.

We thank Jeanne McCarthy for her help in preparing the manuscript.

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Validity of Brain Death Criteria in Infants

Okamoto and Sugimoto report a 3-month-old infant who developed apnea presumably secondary to hypoglycemia and then had a cardiopulmonary arrest. She regained sinus heart rhythm and spontaneous respirations 40 minutes after the apnea was first noted. Examination revealed evidence of neurologic function, including spontaneous respiration. There was subsequent deterioration in the infant's status and the patient met the clinical criteria for brain death on hospital day 3 and this persisted for 48 hours. Ancillary tests, including electroencephalogram (EEG) and brainstem auditory-evoked responses supported the clinical diagnosis. The infant survived for an additional 71 days after meeting the criteria for brain death formulated by the Task Force on Brain Death in Children. Supportive measures were continued because brain death in infants has not been accepted as legal death in Japan. There was no change in her clinical status for the next 6 weeks. However, after 3 weeks, cerebral angiography revealed blood flow to the brain. Somewhat surpris-

Received for publication Dec 24, 1994; accepted Dec 27, 1994.
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ingly, on day 43, the infant developed respirations, 2 to 3/minute with a tidal volume of 40 to 50 mL. They were insufficient to remove the respirator and the respiratory pattern did not change with physiologic or pharmacologic challenges.

The authors suggest that the circumstances of this case should be carefully considered when applying the currently recommended criteria to determine brain death in young children. This implies that the criteria might have failed. I think they did not. There was no recovery of any neurologic function other than 2 to 3 ineffectual respirations/minute. This was insufficient to maintain cardiac function without the use of a ventilator and resulted in the infant being supported for an additional 10 weeks until she developed pneumonia.

An abbreviated summary of the current criteria are: 1) the co-existence of coma and apnea; 2) the absence of brainstem function as defined by nonreactive pupils, absence of spontaneous eye movement, including oculovestibular testing, absence of movement of bulbar musculature, and no respiratory movements off the respirator including the performance of an apnea test; 3) the absence of hypothermia and hypotension; 4) flaccid tone and no spontaneous movement other than of spinal cord origin; and 5) the examination should remain consistent with brain death throughout the observation and testing period. The criteria are not recommended for the first 7 days of life. From 7 days to 2 months, the Task Force recommended two examinations and EEGs separated by at least 48 hours. From 2 months to 1 year, the examinations and EEGs should be separated by at least 24 hours, and over 1 year, the observation period should be at least 12 hours. The most important factors when considering brain death are the proximate cause of coma to insure the absence of a remediable condition and that irreversibility exists. Therefore, in conditions such as hypoxic-ishemic encephalopathies, the observation period is to be extended.[1]

The authors cite three cases that they imply are examples of criteria failure. The first case is that reported by Pasternak and Volpe.[2] This infant was the product of a 35-week gestation, weighed 2.3 kg, and was delivered by emergency caesarean-section because of abruptio placentae. Intraparenchymal and intraventricular hemorrhages were present. The infant subsequently deteriorated and at 72 hours of age was noted to have fixed pupils and absent corneal reflex, absent extraocular movements, and absent facial movements. There was no spontaneous lower cranial nerve function. Respiration and spontaneous extremity movement were also absent. Touching the infant below the angle of the mandible produced flexion myoclonus of all four extremities. EEG activity was present. At 120 hours of age neurologic function began to return. This infant did not meet the guidelines for the determination of brain death in children. First, it is unclear whether oculovestibular (caloric) testing was done or whether only the oculocephalic reflex was absent. Sensation below the angle of the mandible is most likely subserved by cervical root 2 but could be in the distribution of the third division of the trigeminal nerve. There is no indication that an apnea test was performed. Thus, all brainstem function may not have been absent. In addition, the guidelines for the determination of brain death in children are not applicable to children less than 7 days of age. Thus, this infant did not meet the criteria for brain death.

The next case cited as an example of failure of current criteria is that of a 6-week-old infant reported by Green and Lauber.[3] The authors state that this infant had an absence of brain function and a flat EEG but recovered both function and EEG activity the following day. The clinical description given by Green and Lauber was "The pupils were fixed and dilated. The extremities were flaccid with absent stretch reflexes. There is no response to painful stimuli and no spontaneous respiration." An EEG was reported to show no cerebral activity. On the following day, the EEG did show activity and that same day the child moved extremities spontaneously, withdrew from painful stimuli, and regained pupillary light reaction. Subsequent neurologic function included sucking and chewing movements and occasional eye opening. On the 20th day of hospitalization the child regained spontaneous respiration, was able to move his eyes, and suck. He remained this way for 3 weeks until the time of death. This child also did not meet the current criteria suggested by the Task Force. There is no documentation of the child's temperature or blood pressure at the time of the examination. There is no mention of oculovestibular testing and no mention of apnea testing. Also, the Task Force recommends two examinations and two EEGs separated by at least 48 hours for infants who are 6 weeks old.

The third case cited by Okamoto and Sugimoto is that of a 3-month-old infant reported by Kohrmann and Spivack.[4] The infant met the current criteria of brain death and these persisted for 24 hours and included two EEGs which were consistent with electrocerebral silence. The infant's phenobarbital level was 21 ug/mL.

Approximately 4 hours after the second examination, the infant began to make sucking movements. The infant survived for another 5 weeks before suffering a cardiac arrest and dying. In the interim, computed tomography demonstrated extreme cystic encephalomalacia. This infant fulfilled the criteria for 28 hours, which exceeded the guidelines by 4 hours. Sucking motions were regained, but not spontaneous respiration. Thus, the three cases are not examples of infants who had significantly reversible conditions after meeting the criteria of brain death.

The authors recommend that cerebral angiography should be aggressively performed to confirm brain death except in cases with massive cerebral contusion or completed rostral-caudal herniation. They indicate that nonfilling cerebral angiography may be irrefutable evidence of brain death although the converse is not true. There is no data to support that recommendation and its application may not be reasonable. Clearly their case and those reported by Altman[5,6] and Ashwal[7-9] would indicate that the presence of flow does not confirm the viability of the

brain. Cerebral blood flow in the latter cases was determined by methods other than angiography. However, one may assume that if flow was demonstrated by various isotope methods, cerebral angiography would have also demonstrated flow. The absence of intracranial flow in adults appears to be reliable. In adults, the intracranial pressure increases significantly with brain death and the perfusion pressure to the brain is thus reduced and flow is inhibited. However, the same circumstances do not apply to young infants in whom brain death may occur without marked increased intracranial pressure. Another factor to consider is that the lower limits of blood flow necessary to support the immature brain have not been determined. Altman et al[10] have demonstrated flows of 7 to 11 mL/100 g/minute by positron emission tomography are sufficient to sustain the brains of premature infants. Minimal blood flow necessary to support the brainstem in the developing brain is uncertain. Absolute lack of flow to the cerebrum and the brainstem would need to be demonstrated if this information is to be used to support a diagnosis of brain death. We do not know what minimum flow to the brainstem can be demonstrated by angiography. Can we be assured that cerebral angiography can visualize low flows to the brainstem of infants? Until this data is available, the authors' recommendations cannot be supported. Confusion could be created by demonstrating flow in infants who are dead but who do not have increased intracranial pressure and thus maintain patency of some vessels for a period of time. This may cause unnecessary prolonged support.

In conclusion, the authors remind us to be continuously careful and cautious in our application of brain death criteria in young infants. This is appropriate. The return of no clinical neurologic function other than 2 to 3 ineffectual respirations/minute due to the survival of only a small group of neurons in the medulla should not be considered an example of reversible deficits and failure of current brain death criteria. If anything, the criteria were validated. Reconsideration of the criteria based on this case is not warranted.

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AAP Recommendations on Cow Milk, Soy, and Early Infant Feeding

ABBREVIATIONS. IDDM, insulin-dependent diabetes mellitus; BB, BioBreeding (rat); NOD, non-obese diabetic (mice), BSA, bovine serum albumin; SD, standard deviation; HC, hydrolyzed casein; SPI, soy protein isolates.

The report of the American Academy of Pediatrics (AAP) Work Group on Cow Milk Protein and insulin-dependent diabetes mellitus (IDDM) states that early exposure to cow milk protein may be an important factor in the initiation of the β-cell destructive process in some individuals and recommends that, with the exception of cow milk-based infant formulas, high-risk infants not be fed products containing cow proteins during the first year of life.[1] In addition, the feeding of soy-based formulas was discouraged based on studies reported from our laboratory. The idea that IDDM might be food-induced has important possibilities concerning prevention, but the relationship is just not as simple as first thought.[2]

It may be an oversimplification to focus solely on breast-feeding and exposure to a single diet component, cow milk, in the first year of life. There are inconsistencies in the current data likely reflecting variation in the presence or level of diabetogens in different foods or interactions with other factors such as infectious agents. A recent meta-analysis[3] suggests part of the variability in the case-control studies is attributable to inaccuracies in long-term dietary recall, lower response rates in controls versus cases, and failure to account for genetic susceptibility. Thus, the case-control data show a statistically significant but weak association between early infant feeding and diabetes development.

A major assumption in the milk-IDDM hypothesis has been that dietary exposure in the first year of life is important. However, the majority of studies have examined diet only in the first 3 to 6 months of life, which may prove to be only part of the story. One study has shown an effect of weaning foods[4] on

Received for publication Apr 20, 1995; accepted Apr 24, 1995.
Reprints not available.
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