Brain Death in Infants and Children

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In 1987, guidelines for the determination of brain death in children in the United States were proposed by a task force consisting of representatives of several major professional medical and legal societies (Table 1). These guidelines emphasized the importance of evaluation of the patient’s medical history and clinical examination in determining the cause of coma so that remedial or reversible conditions can be differentiated from other nonreversible causes. In addition, age-related observation periods and specific neurodiagnostic tests were recommended for children younger than 1 year. For children older than 1 year, the task force determined that the diagnosis of brain death could be made solely on a clinical basis and that laboratory studies were optional. Since their publication in 1987, these criteria have been generally accepted and have served as explicit guidelines for physicians who are asked to diagnose brain death in children. When these guidelines were developed, criteria for term infants younger than 7 days and for preterm infants were excluded because of the lack of sufficient data. More recent studies indicate that the criteria used in infants younger than 2 months can also be applied to preterm and term infants.

**Epidemiology**

During the past decade, the reported incidence of brain death in older infants and children has ranged from 0.65% to 1.2% of patients admitted to the pediatric intensive care unit (PICU). In one study, the percentage of brain death among deaths overall was 31.4% in children older than 1 month and 6.3% in neonates. Data from Loma Linda University Children’s Hospital revealed that 28% of children who died in the PICU had been declared brain dead. In contrast, only 2.1% of neonates declared dead were also declared brain dead.

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**Table 1 Ad Hoc Task Force guidelines for determination of brain death in children**

A. History: Determine the cause of coma to eliminate any reversible conditions
B. Physical examination criteria:
   1. Coma and apnea
   2. Absence of brain stem function
      a. Mid position or fully dilated pupils
      b. Absence of spontaneous oculocephalic (also called the doll’s eye reflex) and caloric-induced eye movements
      c. Absence of movement of bulbar musculature, corneal, gag, cough, sucking, and rooting reflexes
      d. Absence of respiratory effort with standardized testing for apnea
   3. Patient must not be hypothermic or hypotensive
   4. Flaccid tone and absence of spontaneous or induced movements, excluding activity mediated at spinal cord level
   5. Examination for brain death should remain consistent throughout the predetermined period of observation
C. Observation period according to age:
   1. 7 days to 2 months: 2 examinations and EEGs 48 hours apart
   2. 2 months to 1 year: 2 examinations and EEGs 24 hours apart; or 1 examination and an initial EEG showing ECS, combined with a radionuclide angiogram showing no CBF; or both
   3. More than 1 year: 2 examinations 12 to 24 hours apart; EEG and isotope angiography optional

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On the basis of the number of deceased infant and child donors reported to the Organ Procurement and Transplantation Network, it is estimated that approximately 1800 children younger than 17 years are declared brain dead each year in the United States. This estimate is based on the known number of brain-dead children who are evaluated for organ donation and the fact that only 55% of all brain-dead children become organ donors. Most children who are declared brain dead and who become donors are in the 11- to 17-year-old age group (Figure 1). The proportion of children declared brain dead from 1990 to 2001 remained approximately the same across all age groups (Figure 2). The actual number of children who died between 1993 and 2003 declined by about 22%. The percentage of children who were declared brain dead and then become organ donors in 2001 was approximately the same as the percentage was in 1993 (Figure 3).

Brain death most commonly occurs after acute brain injuries (Table 2). The most frequent cause of brain death in children is traumatic brain injury (30%), most often caused by child abuse and motor vehicle accidents. Asphyxial injury (14%) is also common and occurs after near drowning (9%), as a complication of shock, from strangulation or suffocation, or from sudden infant death syndrome (5%). Brain death due to meningitis may occur in patients who have massive cerebral edema with onset of herniation within 12 to 24 hours of hospitalization. Miscellaneous causes of brain death involve rare metabolic diseases, perioperative injuries of the central nervous system, and acute obstructive hydrocephalus.

In most children who are in a coma after a serious injury of the central nervous system, brain death is usually declared and confirmed within the first 2 days of hospitalization. Once the diagnosis of brain death is confirmed, most children are removed from life-support systems or are referred for organ donation within 2 days. Rarely, brain-dead children have been maintained on ventilator support for prolonged periods, but even in those patients, cardiac arrest occurs within a mean of 17 days after brain death is suspected. Longer survival (eg, chronic brain death) has been reported in children who met the criteria for brain death but for various reasons were maintained on ventilator support for months or years.
Clinical Examination

By definition, all patients who are declared brain dead are comatose and apneic, and they lack brain stem reflexes. These findings may not be present at the time of admission in all children, but they usually evolve during the first days of hospitalization. Serial examinations are frequently helpful. It is essential to ensure that reversible conditions associated with altered metabolic states, exposure to toxic agents, fluid and electrolyte abnormalities, hypothermia, hypotension, or medication effects (particularly pupillary constriction) are treated (Table 3).

Hypothermia occurs in about 50% of children who are comatose after catastrophic brain injury. Thus, patients should be rewarmed before the neurological examination and neurodiagnostic tests are completed.

Coma

Coma is defined as a state of deep, unarousable, sustained pathological unconsciousness with the eyes closed that results from dysfunction of the ascending reticular activating system either in the brain stem or both cerebral hemispheres. Assessment of coma may be difficult in infants and children. Although there is no absolute way to be completely certain that a neonate or young infant has lost all conscious awareness and is “unreceptive and unresponsive,” as stated in the original task force criteria, testing by tactile, visual, and auditory stimulation is comparable to the testing used for an older child. When attempting to diagnose brain death, even in neonatal and very young patients, the clinician is assessing the complete loss of all responsiveness rather than trying to detect subtle conscious behaviors. In most instances, and regardless of the age of the child, the bedside clinical examination is satisfactory for accomplishing this goal. The absence of any form of repetitive, sustained, purposeful activity on serial examinations must be documented; likewise, brain death must be differentiated from other states of unconsciousness, such as the vegetative state. If the results of neurological examination remain unreliable or uncertainty exists about whether the child is unresponsive, confirmatory neurodiagnostic studies such as electroencephalography (EEG) and measurement of cerebral blood flow (CBF) are required.

Loss of Brain Stem Function

In preterm and term neonates, clinicians must consider the fact that several of the cranial nerve responses are not fully developed. For example, the pupillary light reflex is absent before 29 to 30 weeks’ gestation, and the oculocephalic reflex may not be elicited before 32 weeks’ gestation. Term and preterm infants are difficult to examine because their smallness makes it technically difficult to assess cranial nerve function adequately. Assessment of pupillary reactivity can be compromised at the bedside by difficulties in gaining access to the infant in an incubator, as well as by corneal injury, retinal hemorrhages, and other anatomical factors such as swelling or partial

Figure 3 Data showing that the percentages of children being declared brain dead and who became organ donors in 2001 was similar to the percentages in 1990. The top line (squares) is an estimate of the percentage of the total number of brain-dead children per total number of deaths, and the bottom line (diamonds) is the percentage of the total number of brain-dead children who became organ donors per total number of deaths.

Table 2 Causes of brain death in 590 infants and children

<table>
<thead>
<tr>
<th>Causes</th>
<th>% of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head injury</td>
<td>30</td>
</tr>
<tr>
<td>Near drowning</td>
<td>9</td>
</tr>
<tr>
<td>Central nervous system infection</td>
<td>16</td>
</tr>
<tr>
<td>Asphyxia</td>
<td>14</td>
</tr>
<tr>
<td>Sudden infant death syndrome</td>
<td>5</td>
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<tr>
<td>Metabolic disorders</td>
<td>5</td>
</tr>
<tr>
<td>Cerebrovascular disorders</td>
<td>5</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>16</td>
</tr>
</tbody>
</table>

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fusion of the eyelids. Because of the smaller amount of pigmentation and the smaller size of the pupils in newborns, difficulty in visualizing changes in the size of the pupil can make assessment of the loss of pupillary reactivity troublesome. Assessment of ocular motility likewise can be difficult in small intubated patients, and often the examiner will need assistance, particularly for ice water caloric stimulation. From a procedural point of view, performing this test in newborns is not substantially different from performing the test in older children. Assessing the caloric response adequately is more difficult in neonates with a small external ear canal; therefore, both the oculocephalic (doll’s eye) reflex and the oculovestibular (caloric) reflex should always be examined.

Although the corneal reflex is perhaps the easiest brain stem reflex to examine in neonates and infants, it is often the least reliable. Contact irritation, dehydration and maceration of the cornea, use of lubricant drops, wearing of eye patches for treatment of hyperbilirubinemia, and use of analgesic medications often adversely affect tactile sensory receptors on the surface of the cornea. Testing for this reflex is important, however, because its presence indicates preserved brain stem function.

Assessment of lower cranial nerve function is also limited and is usually confined to examination of the gag reflex. When infants are intubated (by either the oral or the nasogastric route), testing of the gag reflex usually can be accomplished only by stimulating the endotracheal tube (eg, by passing a suction catheter through the endotracheal tube).
Apnea

The normal physiological threshold for apnea in children (ie, the minimum carbon dioxide tension at which respiration begins) has been assumed to be the same as that in adults (PCO2 > 60 mm Hg). Most studies indicate a gradual increase in PCO2 over a 5- to 10-minute period, typically while arterial Po2 is maintained at greater than 200 mm Hg and 100% oxygen is supplied tracheally. Recent reports concerning apnea testing in children have raised questions about (1) the effects of lesions compressing the brain stem, (2) potential recovery of brain stem respiratory drive, and (3) the PCO2 threshold in children. In 1993, Ammar et al. reported data on 5 children, ages 9 months to 7 years, with severe brain stem dysfunction, including loss of pupillary reflexes and apnea, caused by surgically resectable lesions in the brain stem. These children experienced return of spontaneous respirations and substantial neurological function after surgery despite having met most but not all criteria for brain death before the surgery. Ammar et al. suggested that treatment of lesions compressing the brain stem might reverse severe neurological deficits that mimic brain death. In another report, a 3-month-old infant who met the criteria for brain death (Table 1) started taking 2 to 3 irregular breaths per minute on day 43 of hospitalization, but died 71 days after becoming comatose. At issue is whether such an occurrence should be considered a return of respiratory function and, if so, whether return of irregular breathing is an “improvement” in the absence of other evidence of brain stem function.

A third report involves the case of a 4-year old child who had a pilocytic astrocytoma in the posterior fossa and experienced a cardiac arrest. This child met the clinical criteria for brain death, except for the results of apnea testing, which showed minimal respiratory effort after 9 minutes and 23 seconds, when the child’s PCO2 was 91 mm Hg. This child’s spontaneous breathing was insufficient to maintain life, and assisted ventilation was necessary. The authors thought that this child’s higher PCO2 threshold was the result of hypoxic-ischemic injury. This case raised questions as to whether this phenomenon was unique to children and whether the current standard of a PCO2 of 60 mm Hg is correct.

Apnea testing in children is similar to that in adults; apneic oxygenation is used after mechanical ventilation is discontinued. Normalization of PCO2 and core temperature and preoxygenation for 5 to 10 minutes before the apnea challenge is begun are recommended. Careful monitoring of heart rate and blood pressure during observation of the chest cage for movement is needed. Most studies recommend that PCO2 levels be measured at 5-minute intervals and continued for 15 minutes if PCO2 has not reached 60 mm Hg and PO2 has not decreased less than 50 mm Hg. Prolonged bradycardia or the development of hypotension during testing is due to irreversible brain stem failure, acidosis, or hypoxemia, and if any of these occurs, mechanical ventilation should be resumed. Children should be given a few manual respirations with a resuscitation bag before mechanical ventilation is resumed, because the ventilator rate is often lowered to normalize carbon dioxide levels.

Electroencephalography

Guidelines for recordings used to determine brain death were developed by the American Electroencephalographic Society in 1994. The role of EEG in confirming brain death in infants and children, as well as in neonates, has been reviewed by Moshe and Schneider. Problems with obtaining an EEG in infants and children include shorter inter-electrode distances; external artifacts in newborn ICUs and PICUs; rapid cardiac and respiratory rates of infants and children compared with those of adults; shorter distances between the heart and the brain, making the electrocardiographic contribution disproportionately large in children; reduced amplitude of cortical potentials in preterm and term neonates; and useful methods for confirming the clinical diagnosis of brain death. However, during the past decade, reliance on confirmatory testing has decreased and reliance on repeated clinical examinations that indicates coma, apnea, and lack of brain stem function has increased.
persistent EEG activity. Most of these EEG patterns show low-voltage theta or beta activity or intermittent spindle activity. Such activity in functionally dead brains may persist for days. Data from several studies indicate that the initial EEG in brain-dead children is isoelectric in 51% to 100% of patients (mean 83%). In most children who initially have EEG activity, follow-up studies usually show evolution to ECS.

Typically, when the initial EEG in children shows ECS, a repeat EEG will also show no activity. Several cases of recovery of EEG activity have been reported. In these reports, the findings were either inconclusive or the patients had retained some brain stem or cerebral function and thus did not meet the clinical criteria for brain death. Since the report by Green and Lauber almost 30 years ago of 2 infants who experienced return of some EEG activity after an initial ECS recording, only a few additional reports of infants in whom EEG activity returned have been published; none of these infants recovered. Thus, concerns about the return of EEG activity have been overemphasized.

ECS may occur soon after an infant or a child has had a cardiac arrest. In infants in whom the initial EEG (typically obtained 8-10 hours after cardiac arrest) showed ECS, a repeat study 12 to 24 hours later may show diffuse low-voltage activity. Most of these infants die of complications associated with the acute catastrophic injury; the remaining survivors usually evolve to a permanent vegetative or minimally conscious state. Similar observations have been reported in adults.

Overall, the available data in children suggest that evidence of ECS on the initial EEG is sufficient to support the clinical diagnosis of brain death. However, according to the 1987 task force guidelines, it is not necessary to obtain an EEG in children older than 1 year, so long as the findings on neurological examination remain unchanged for the age-appropriate observation period.

In children, the most common medications causing reversible loss of brain electrocortical activity include barbiturates (eg, phenobarbital), benzodiazepines, narcotics, and certain intravenous (thiopental, ketamine, midazolam) and inhalation (halothane and isoflurane) anesthetics. Data from a study in 92 children indicated that therapeutic levels of phenobarbital (ie, 15-40 μg/mL) do not affect the EEG.

**Determination of Cerebral Blood Flow**

Neuroimaging techniques used to document the absence of CBF include cerebral angiography, radionuclide angiography, transcranial Doppler sonography, computed tomography with injection of contrast material or xenon inhalation, digital subtraction angiography, single-photon emission computed tomography, and positron emission tomography. Of these, radionuclide angiography remains the most widely used in children because the necessary equipment is portable and the procedure is relatively sensitive and easy to perform. Documentation of the absence of CBF is considered confirmatory of brain death and has been reviewed elsewhere in detail.

**Radionuclide Imaging**

Radionuclide imaging in children is accurate and reproducible and has been favorably compared with other methods of detecting the presence or absence of CBF. The absence of CBF in brain death is due primarily to low cerebral perfusion pressure (which can be calculated by subtracting intracranial pressure [ICP] from mean arterial pressure) and secondarily to release of vasoconstrictors from vascular smooth muscle and
brain parenchyma. Figure 5 illustrates an example of the absence of CBF shown by radionuclide imaging.

A certain percentage of children who are brain dead may have evidence of CBF shortly after diagnosis. In studies reported by Drake et al., 15 of 47 brain-dead children had evidence of intact CBF as determined by radionuclide imaging. About two thirds of the patients had loss of CBF when restudied 2 to 3 days later. This loss of CBF occurred regardless of whether these patients had ECS or some EEG activity when the first study was done. In a more recent report, 16 of 18 clinically brain-dead preterm and term infants had some CBF. Greisen and Pryds also reported 2 newborn infants with ECS who were thought to be brain dead but had preserved CBF as shown by xenon scanning. Overall, CBF clearly may be present in infants and children who are clinically brain dead. In most patients, additional radionuclide studies 24 to 48 hours later will most likely, although not uniformly, show the loss of CBF.

Transcranial Doppler Sonography

Transcranial Doppler sonography has been advocated because it is a portable, noninvasive method of detecting cerebral circulatory arrest. Since 1983, studies in children have validated the specificity and sensitivity of this method. Changes detected with transcranial Doppler sonography in brain-dead patients include loss of diastolic flow, appearance of retrograde diastolic flow, diminution of systolic flow in the anterior cerebral artery with unchanged flow in the common carotid artery, and, finally, the loss of any detectable flow in these vessels.

Digital Subtraction Angiography

In digital subtraction angiography, another technique used to assess intracranial circulation, contrast material can be given intravenously or by intra-arterial injection. As in conventional cerebral angiography, a small amount of nonionic contrast material is injected while digital subtraction imaging of the cerebral vasculature is done. This process allows visualization of contrast material within the major intracranial vessels; lack of such visualization indicates absence of CBF. Few reports of use of this technique in children have been published; a case report described use of this technique in a brain-dead neonate. The authors of a report of the use of intravenous digital subtraction angiography in 110 patients with clinical signs of brain death observed that the first examination documented the absence of contrast enhancement in 105 patients. Repeat examinations in the remaining 5 patients, performed within several hours, also confirmed cessation of CBF.
**Evoked Responses**  Brain stem auditory response (BAER) testing has been extensively studied as an alternative confirmatory method of determining brain death.1 BAER tests are noninvasive tests performed by presenting a repeated auditory stimulus through headphones placed over the patient’s ears and then recording the evoked response from scalp electrodes. Its portability and noninvasiveness seem ideal, but several studies20-24 have raised doubt about the reliability of BAER testing in determining brain death, particularly in children younger than 6 months. More recent studies20-24 have suggested that BAER tests are reliable for confirming brain death in children. In one report, 25 90% of 51 brain-dead children had loss of the BAER (complete loss in 27 patients; loss of waveforms III-VII in 18 patients). Loss of the BAER also preceded development of ECS. This finding suggests that BAER testing might be more useful than EEG for earlier laboratory confirmation of brain death. However, if BAER testing is performed too early, a false-positive result may occur.

Somatosensory evoked potentials may offer greater discrimination in the confirmation of brain death. In recent studies of somatosensory evoked potentials in children, only 62.5% of patients had either complete absence of the somatosensory evoked potential or only a cervical cord (but no thalamocortical) response, suggesting a limitation of somatosensory evoked potentials as a confirmatory test for brain death in children.3

**Brain Death in Newborns**

About 550 newborns of every 4.9 million live births have brain death diagnosed before the age of 1 year. Causes of brain death in 87 newborns younger than 1 month included hypoxic-ischemic encephalopathy (61%), birth trauma (8%), malformations (6%), cerebral hemorrhage (6%), infection (7%), sudden infant death syndrome (7%), nonaccidental trauma (4%), and metabolic causes (1%).27

Preterm and term neonates younger than 7 days were excluded from the 1987 guidelines for determination of brain death in children, and the ability to diagnose brain death in newborns is still viewed with uncertainty because of the small number of reports on brain-dead neonates. Several years after publication of the guidelines, data on 18 brain-dead neonates were published, and the authors6 suggested that brain death could be diagnosed in term infants and preterm infants greater than 34 weeks’ gestational age within the first week of life. Because newborns have patent sutures and an open fontanel, increases in ICP after acute injury are not as significant as in older patients. Thus, the usual cascade of herniation from increased ICP and reduced cerebral perfusion is less likely to occur in newborns than in older infants. However, brain death can be diagnosed in newborns (even when younger than 7 days) if the physician is aware of the limitations of the clinical examination and laboratory testing. These infants should be examined carefully and repeatedly, with particular attention given to brain stem reflexes and apnea testing. An observation period of 48 hours is recommended to confirm the diagnosis. If an EEG is isoelectric or a CBF study shows no flow, the observation period can be shortened to 24 hours. Although preterm infants who are brain dead are rare, the same time frame most likely would be applicable. A few instances of neonates or older infants who showed minimal transient clinical or EEG recovery have been reported, but none appear to have regained meaningful neurological function, and all died within brief periods.3

Because of significant physiological and cerebrovascular differences in the neonatal response to injuries resulting in brain death, a much higher incidence of newborns with EEG activity or cerebral perfusion has been observed in previous studies.3 In addition, some newborns with ECS showed preserved CBF, and conversely, others without CBF showed EEG activity. In neonates, even though CBF and mean arterial blood pressure are much lower than in older children, increases in ICP after acute injury are less dramatic. Recent data on 30 newborns who underwent EEGs and radionuclide perfusion studies indicate that one third of the infants with ECS had evidence of CBF, and 58% of those with no CBF had evidence of EEG activity.27

Data on 37 of 53 brain-dead newborns in whom EEGs were performed revealed the following findings: ECS (n = 21), very low voltage (n = 13), burst suppression (n = 1), seizure activity (n = 1), and normal activity (n = 1). Almost all patients whose first EEG showed ECS had ECS on the second study, and most patients who did not show ECS on the first EEG did so on a repeat study. The data suggest that only a single EEG showing ECS is necessary to confirm brain death, provided the results of the examination remain unchanged.3

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CBF data indicate that CBF is absent in 72% of brain-dead newborns. In some infants who initially had evidence of CBF, repeat studies showed an absence of flow. The median duration of brain death in the neonates with CBF (4 days) did not differ significantly from that in neonates without CBF (3 days). These findings, as well as those described earlier, again emphasize the limitations of both CBF determinations and EEG findings for confirmation of brain death in neonates.

Organ Procurement in Brain-Dead Children

Because brain death in children is more often due to severe asphyxial injury than is brain death in adults, the question has been raised about whether similar injury to other organs would preclude transplantation. Use of inotropic agents to support blood pressure and cardiac function may be necessary in such children, particularly if brain death was related to a preexisting global asphyxial injury that may have caused hypoxic myocardial injury. Recent data, however, suggest that organ transplantation, including heart transplantation, can be successfully accomplished with organs obtained from infants and children. Likewise, although brain-dead victims of child abuse have infrequently been considered organ donors because of legal issues, many centers are currently trying to obtain surrogate consent and, with cooperation from the medical examiner’s or coroner’s office, have been able to successfully recover organs from such donors. Surrogate consent is not necessary in every state. Often parents retain the right of consent even if they have been arrested for the abuse.

The loss of neuroendocrine function must be treated if successful organ donation is to be accomplished. Perhaps the most common problem is diabetes insipidus, which can be easily controlled with low doses of vasopressin (3-5 units intramuscularly or 0.05-0.1 units/kg intravenously as needed). Some patients may also require supplemental corticosteroid therapy, because the hypothalamic-pituitary-adrenal axis may be impaired. Adequate respiratory support to maintain organ function is also important. In addition, treatment and prevention of infection will minimize cardiovascular instability. In most instances, the time necessary for this type of monitoring and support is relatively short (2 days) before organ recovery occurs.

Providing support for the grieving family and for nursing and other medical staff is extremely important and is perhaps one of the most vital roles a physician can play. The responsibilities of the physician involved in the declaration of brain death must always be clearly demarcated from the responsibilities of physicians involved in or responsible for organ procurement. It is also important for the medical staff to provide information to the patient’s family on a continuing basis. The more informed a family is about the medical status of the child, the better equipped the family members will be to have a discussion about organ donation.

Responsibilities of PICU Nurses in Determination of Brain Death

Although nurses in the PICU are not involved in the formal declaration of brain death, they provide specialized nursing care, monitor specific clinical data, and coordinate specialized studies used by clinicians to determine brain death. In a child with a brain injury, specialized nursing care may include maintenance of cardiovascular and ventilatory support and thermoregulation. Pertinent clinical laboratory tests include metabolic studies, toxicology studies, and assays of medication levels. Neurodiagnostic tests specifically used to determine brain death, such as an EEG and CBF studies, should be coordinated at specified intervals depending on the age of the child. Specialty nurses in the PICU play a key role in the declaration of brain death. These nurses provide the specific clinical information used by the clinician to determine whether the 3 standard clinical criteria for brain death—coma, absence of brain stem reflexes, and apnea—have been met.

In the PICU, the bedside nurse is often first to initiate neurological evaluation for brain death when a patient’s level of consciousness or brain stem reflexes deteriorate. Coma is evidenced by a lack of cerebral responsiveness to verbal or tactile stimuli. Before cerebral responsiveness is assessed, all reversible conditions of coma, such as hypothermia (core body temperature <32°C), drug intoxication or poisoning, use of neuromuscular blocking agents, severe electrolyte imbalance, severe acid-base abnormalities, and severe metabolic or endocrine imbalance, must be ruled out. If barbiturates were used to induce coma, the levels of the drugs must be subtherapeutic in order for brain death to be assessed. In the PICU, specialized nursing care involves frequent monitoring of vital signs, including ICP, cerebral perfu-
sion pressure, and ventilation, as well as level of alertness and score on the Glasgow Coma Scale, which is used to measure verbal, motor, and visual responses, for confirmation of brain death.

Testing brain stem reflexes includes assessing pupillary reaction to light, the corneal reflex, and the oculocephalotubular and gag reflexes. These reflexes must be absent in order for brain death to be declared. Testing of pupillary reaction to light most commonly yields pupils in mid position at 4 to 6 mm. However, the position may vary from 4 to 9 mm, and pupils may be round, irregular, or unequal. The corneal reflex can be tested by gently wiping the cornea with a strand of cotton. Absence of this reflex is evidenced by a lack of reaction (eg, blinking) or a lack of any sign of pain (eg, grimacing). The gag and cough reflexes can be tested by introducing the tip of a tongue blade into the back of the oral cavity. There should be no gag or cough reflex.

Adverse changes in level of consciousness or brain stem reflexes should be reported immediately to a physician. Once the physician has examined the patient, confirming and documenting findings of coma and absence of brain stem reflexes, an apnea test, the third and final criterion for confirming brain death, may be performed. Although institutions may vary somewhat in the parameters used to perform an apnea test, 3 conditions should exist before the test is done: the patient’s core body temperature should be at a minimum of 36.5°C; systolic blood pressure should be 90 mm Hg or greater, with a positive fluid balance to maintain organ perfusion; and PaCO₂ should be 40 mm Hg or greater if the patient was preoxygenated. The apnea test is positive when no respiratory movement is noted and the posttest PaCO₂ is 60 mm Hg or greater. Together, these findings support the clinical determination of brain death.

Coma, absence of brain stem reflexes, and the presence of apnea are the standard clinical criteria that confirm brain death. However, when any uncertainty remains or further confirmation is needed, diagnostic testing may be used. Cerebral angiography, electroencephalography, transcranial Doppler sonography, and somatosensory and brain stem auditory evoked potential and CBF scans (technetium Tc 99m brain scan) are commonly used.

Cerebral angiography will indicate no intracerebral filling at the level of the carotid bifurcation or circle of Willis. Electroencephalography should indicate no electrical activity during a recording period of at least 30 minutes. Transcranial Doppler sonography findings consistent with brain death are indicated by high vascular resistance and high increased ICP. The findings should include lack of diastolic or reverberating flow, systolic-only flow or retrograde diastolic flow, and small systolic peaks in early systole. Somatosensory and brain stem auditory evoked potentials yield no responses. Finally, CBF studies should show no uptake of radionuclide in brain parenchyma (hollow skull phenomenon). Of the studies used for determination of brain death, EEG and CBF examinations are the most easily accessible in the PICU setting and therefore the most commonly used.

If and when a physician determines that diagnostic testing is needed, the PICU nurse should continue providing specialty care that maintains the desired hemodynamic, ventilation, and hydration status required for the results of neurological testing to be valid. In addition, the nurse should coordinate laboratory and neurodiagnostic studies at specified intervals. In general, for children older than 1 year, 2 neurological examinations are performed 24 hours apart. Younger children may require more time between neurological examinations and diagnostic testing. An algorithm for the diagnosis of brain death is given in Figure 6.

The role of the PICU nurse includes being knowledgeable and skilled in delivering specialized care to children who have experienced brain death. Although this role involves a variety of technical and psychological factors, consistent application of the nursing process will provide continuous care that meets each patient’s unique needs.

Conclusions

The diagnosis of brain death in children is based on the same principles used in adults. Although the neurological examination is difficult because of the size of the patient, the immaturity of certain developmental reflexes being tested, and pathophysiological differences due to the presence of open sutures and fontanels in neonates and infants, the 3 fundamental criteria for brain death (coma, apnea, and absent brain stem reflexes) are the standard clinical criteria for confirming results on neurodiagnostic tests. As is the case in many
other areas of medicine, clinical judgment and serial examination allow a definitive diagnosis to be established. A better understanding of the pathophysiology of brain death in neonates and infants should help determine whether recommended age-related periods of observation are based on differences in developmental neurophysiological or cerebrovascular regulation because it remains unknown whether newborn infants might have potential for later and significant recovery of brain function.

References