BRAIN DEATH POLICY AND PROCEDURES

I. University of North Carolina Hospitals

A. Policy or Determining Pediatric Brain Death

Two professional standards must be met to certify Brain Death. 1) The physician must be professionally competent and trained to interpret the clinical evidence identified as being necessary criteria for Brain Death, see Section C. For the UNC PICU, those deemed appropriate are pediatric intensivists, neurosurgeons, and neurologists 2) The physician must have no conflict in performing this function for the patient. (Potential conflict may exist in professional, economic, religious, and/or social arenas.) In the situation where organ donation may be involved, the preferred method will be for either neurosurgery and/or neurology to perform both exams. However, the minimum shall be one exam by either neurosurgery or pediatric neurology services with the second exam being performed by the pediatric intensivist.

B. Relationship of Brain Death Determination to Organ Procurement

An increasing need for procuring organs and tissue from deceased patients is recognized. In general, the shorter the time between death and harvesting, the higher the likelihood of successful transplantation. The decision to declare Brain Death must occur independent of decisions regarding the appropriateness and feasibility of organ donation. It is the responsibility of the attending physician to discuss Brain Death with the patient’s family. This communication should be facilitated by a health care professional responsible for the patient’s well-being (nursing, house staff, social work, and clergy). The approval of the attending physician is required to both notify Carolina Donor Services and initiate the approach to patient’s family for donation consent. This discussion is best accomplished with additional information from third party organ procurement specialists.

C. Definition.

Brain death has occurred when cerebral and brainstem functions are irreversibly absent.

Absent cerebral function is recognized clinically as the lack of receptivity and responsivity with no psychological, autonomic or somatic response to any external stimulation. Absent brainstem function is recognized clinically when pupillary light, corneal, oculocephalic, oculovestibular, oropharyngeal, and respiratory reflexes are irreversibly absent.

Procedures for testing these reflexes are noted below. Peripheral nervous activity, including spinal cord reflexes, may persist after brain death, however, decorticate and decerebrate posturing is inconsistent with brain death.
Irreversibility is recognized when the cause of coma is established and is sufficient to account for the loss of brain function, and the possibility of recovery is excluded by observation for an appropriate period of time.

It is important to recognize the cause of coma, if possible, since absence of brain function resulting from head trauma has different implications than that caused by metabolic abnormality or intoxication. Reversible causes of coma that may mimic irreversibility include sedation, certain intoxications, renal, hepatic, and other electrolyte abnormalities, hypothermia, neuromuscular blockade and shock. If the cause of coma cannot be established, these reversible conditions must be ruled out by appropriate laboratory studies.

The period of observation will also vary, depending on the circumstances and possible causes. In most cases, in which the cause is established and appropriately accounts for coma, a period of 6 hours is reasonable.

The established adult criteria for brain death appear valid in infants and children, although caution should be exercised in applying them to premature and newborn infants. A more prolonged period of observation may be necessary in this population. (See worksheet.)

If cause and irreversibility are established and the clinical examination yields unequivocal findings, confirmatory tests are unnecessary. They may, however, be appropriate in determining primarily the irreversibility of brain death, and possibly accelerating the determination of that irreversibility. When appropriate, confirmatory laboratory testing may prove helpful include electroencephalography, radionucleotide brain scan, transcranial ultrasound/Doppler studies, and brainstem evoked potentials.

II. CLINICAL CRITERIA FOR BRAIN DEATH

A. Prerequisites: The following criteria should be documented in the medical record in addition to the patient's exam.

1. Irreversibility of brain injury demonstrated with the exclusion of reversible conditions.
2. Absence of hypothermia (core temperature over 32°C).
3. Absence of cardiovascular shock or profound metabolic derangements, i.e. Na < 125 or K < 2.
4. Absence of depressant drugs.
5. Two observations at intervals of at least 6 hours by an intensivist, neurologist, or neurosurgeon (who is a member of the active staff), or another physician licensed to practice medicine, applying appropriate standards of medical practice.

NOTE: The duration of observation has been gradually shortened. Six hours is the current usual standard, though 12 to 48 hours may be required- if doubt exists about the cause of irreversibility of coma in adults and children.
B. CLINICAL CRITERIA FOR BRAIN DEATH

1. Absent brain stem function:
   a. Fixed and dilated pupils.
   b. No ocular-vestibular response to 100cc of ice water instilled over 30-60 seconds.
   c. Absent corneal and pharyngeal reflexes including cough on tracheal suction.
   d. The absence of respiratory movement during apnea testing with an FiO₂ of 1.0.

   NOTE: The patient should have a normal to mildly elevated PaCO₂, 40-60 torr, at the initiation of apnea testing and should have “apneic ventilation” performed for a period of time sufficient to induce a 20 torr increase in PaCO₂. Appropriate safety steps are taken and include pre-oxygenation with 100% oxygen and monitoring of arterial blood pressure and SaO₂. Severe hypotension or desaturation is reason to terminate testing.

2. No behavioral or reflex response to noxious stimuli that imply function above the level of the foramen magnum. (NOTE: Spinal reflexes may be preserved, and their presence does not preclude diagnosis of death.)

   NOTE: Areflexia is common after severe brain injury. However, as the duration of absent cerebral function lengthens, it becomes more likely for spinal reflexes to reappear.

C. Special Cases

1. Ancillary laboratory tests: Laboratory tests are not required where the diagnosis is certain and the clinical criteria are met. The EEG and/or imaging of blood flow can be useful diagnostic and prognostic tests were there is uncertainty about either the etiology or the clinical findings.

2. Therapeutic drug-induced coma: Barbiturates and hypothermia currently, have a role in ameliorating damage to the brain of some severely ill patients. in the presence of therapeutic drug intoxication or hypothermia, the diagnostic and prognostic utility of the EEG diminishes. Absent cerebral flow as measured by imaging studies will be required in these instances and should be performed promptly when brain death is suspected.

D. Death Certificate

The death certificate is to be completed by the patient’s attending physician or another physician member of that service.
CONFIRMATORY STUDIES FOR THE DETERMINATION OF ELECTROCEREBRAL INACTIVITY:

EEG

All personnel involved should be aware of methods of enhancing the value of the EEG in the determination of electrocerebral inactivity. The laboratory personnel performing an EEG should be careful in noting any possible technical artifacts which may be contaminating the record. Examples of these are respiratory or intravenous drip-related artifacts, movement about the room by Intensive Care personnel, EKG artifacts and the like. Also, Intensive Care Unit personnel should be aware that they may be able to enhance the diagnostic accuracy of the test by restricting their movements near the patient during the test. All parties should understand that temporary muscular blocks such as succinylcholine or pancuronium are often invaluable to the EEG recording, since the drugs can temporarily attenuate muscle activity which is obscuring the electroencephalographic record. To emphasize this further, in the presence of even low amplitude muscle artifact, and EEG diagnosis of electrocerebral inactivity may be impossible whereas with temporary neuromuscular blockade, such a diagnosis may be made with certainty.

Technectium 99m cerebral perfusion scan or radionuclide angiogram. This test is also used in confirmation of brain death, specifically when the EEG is equivocal or barbiturates are present. A bolus of Tc-99m as sodium pertechnetate (200 μCi/kg, minimum of 5 mCi) is given intravenously in < 3 s. The patient is imaged in the anterior projection, using a gamma camera/computer system. Recording is begun at the time of injection at one frame per second for 60 s. Evaluation of the study is visual on the series of images and on time-activity curves obtained from the regions of interest over the cerebral hemispheres. In cerebral death, the radionuclide cerebral angiogram shows (1) bilateral absence of the arterial phase (anterior and middle cerebral artery territories), (2) lack of visualization of the sagittal sinus during the venous phase, (3) lack of arterial peak of cerebral time-activity curves, and (4) perfusion of the extracranial tissues only.

Brainstem evoked potentials. Indications are the same as for the EEG, to demonstrate that reactivity to incoming stimuli (auditory, somatosensory) is absent in brainstem nuclei, except in the first (cochlear) wave. A "flat" auditory brainstem response in the presence of viable peripheral conduction (cochlear wave) is considered unequivocal evidence of cerebral death because of the proximity of auditory nuclei to vital centers. If acoustic conduction is not demonstrable even at maximal stimulus intensity, auditory brainstem evoked potentials should be complemented by somatosensory brainstem evoked potentials to rule out the possibility that a flat response is caused by peripheral deafness. Brainstem sensory evoked potentials are not a substituted for the EEG, but do effectively complement the information derived from spontaneous electrical cortical activity when cerebral death is suspected on clinical grounds. Brainstem evoked potentials have the advantage of being free of artifact contamination and are less sensitive than the EEG to the effect of CNS depressants. The disappearance of activity from the EEG may precede the loss of brainstem responses because the cortical structures are more sensitive to the effect of severe...
insults than are the brainstem structures. Thus, at the time of an isoelectric EEG, brainstem responses may be abnormal though not completely absent. In this case, serial evoked potentials are indicated to provide evidence of deterioration in brainstem function. Brainstem evoked potentials are recommended when (1) the interpretation of the isoelectric EEG is controversial, (2) the EEG is not completely isoelectric, (agonal stage), or (3) the EEG is isoelectric but the effects of CNS depressants or hypothermia is present.

Transcranial Ultrasound / Doppler. These studies may be used when other standard studies are unavailable or if the patient cannot be readily transported. The Doppler studies are used to determine reversal of flow in the major cerebral arteries, indicating an absence of cerebral blood flow, specifically examining the internal carotid artery and the middle cerebral artery.