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# Understanding the Mystery of Brain Death

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Lack of understanding of brain death has contributed to recent controversial cases (Copnell, 2014). The cases of Jahi McMath and Marlise Munoz were the focus of national news, causing an intense debate among the public and healthcare professionals (Copnell, 2014). The concept of brain death is a mystery to the public (Powell, 2014). Copnell (2014) found that 60% of family members who had been told their loved one was brain dead believed that they were still alive. Health professionals not having a clear grasp of brain death's definition and assessment can lead to increased confusion in family members (Powell, 2014).

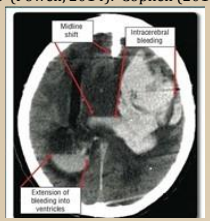


*Figure 1.* Computed Tomography scan showing hemorrhagic stroke, midline shift, and extension of bleeding into ventricles. Adapted from "Brain death: Assessment, Controversy, and Confounding Factors," by R.B. Arbour, 2012, *CriticalCareNurse, 33*(6), p.30. Copyright 2012 by American Association of Critical-Care Nurses.

## Causes of Brain Death

- **Stroke (hemorrhagic or ischemic)**
- **Intracranial tumors**
- **Infectious processes**
- **Head trauma**

## Implications for Nursing Care

- Nurses should explore misunderstandings, deliver consistent information, and make use educational videos and reading material to help family members understand their loved one's diagnosis (Copnell, 2014).
- The "Embrace Hope" structured multidisciplinary delineation of end-of-life interactions can be used to help educate family members and provide structured care to the dying or brain dead patient (Yeager, et al., 2010). "Embrace Hope" includes a care packet includes love locks, handprints, and grief information for the family members (Yeager, et al. 2010). "Embrace Hope" care guideline also includes a cultural/spiritual assessment tool and checklist for nursing interventions (Yeager, et al., 2010). The checklist includes interventions such as consulting palliative care and notifying Lifeline of Ohio Organ Procurement (LOOP) (Yeager, et al., 2010).
- Consulting palliative care offers families support and helps meets the needs of a patient's end-of-life care. When the patient has been officially declared brain dead the family is faced with questions regarding plan of care. Specialist such as palliative care and LOOP should be utilized to help approach the family members on whether they would like to consider organ donation (Siminoff, Agyemang, & Traino, 2013).

## Pathophysiological Process

Brain death is defined as "irreversible loss of all brain function, or "whole brain" death (Copnell, 2014, p.259)." Brain death can be caused by various factors, such as trauma, ischemia, meningitis, anoxic injury, or intracranial hemorrhages (Arbour, 2013). All of these factors have the potential to cause cerebral edema and brain stem herniation (Arbour, 2013). Diagnosis of brain death is determined by absence of brain reflexes, presence of apnea, and coma (Lugt, 2010). The diagnosis of brain death is considered to be equal to death (Thomas, 2012).

Signs and symptoms preceding to brain death include increased intracranial pressure (ICP), loss of consciousness, the Cushing response, and hypertension followed by hypotension with loss of vasomotor tone (Arbour, 2013). ICP rises when cerebral swelling, or other pathological processes occupy space in the fixed intracranial vault (McCance & Huether, 2014). The brain compensates by equally reducing the volume of other intracranial contents, such as cerebral spinal fluid (CSF) and blood volume (McCance & Huether, 2014). Sustained increases in ICP cause a reduction in cerebral blood flow, ischemia, and central herniation (Arbour, 2013; McCance & Huether, 2014).

In response to the pressure or ischemia on the pons, the brain activates the Cushing response to attempt to maintain adequate blood perfusion to the brain (Arbour, 2013). Clinical manifestations of the Cushing response are hypertension, bradycardia, and a widening pulse pressure (Arbour, 2013; McCance & Huether, 2014). As ICP rises and the brain becomes more hypoxic, the Cushing response eventually fails to maintain adequate blood flow to the brain (Arbour, 2013). With decreased blood perfusion to the brain, the patient may have progressive loss of consciousness, seizures, and posturing (Arbour, 2013).

As a last response to maintain cerebral blood flow, the sympathetic nervous system releases catecholamines (Arbour, 2013). The release of catecholamines causes tachycardia, vasoconstriction, and hypertension (Arbour, 2013). In the final stage of brain stem herniation, catecholamine stores are depleted, sympathetic nervous system regulation is lost, and the patient presents with hypotension and loss of vasomotor tone (Arbour, 2013).

Signs and symptoms of brain death include coma, absence of response to central pain stimulus, loss of cranial nerve reflexes, and apnea (Shutter, 2014). Consciousness can be assessed using the Glasgow Coma Scale (GCS) (Arbour, 2013). No motor response, eye opening, or verbal response (score of 3) indicates complete loss of consciousness (Arbour, 2013).

Another sign of brain death is loss of response to central pain (Shutter, 2014). Central pain can be assessed by applying noxious deep pressure to the supraorbital notch, upper trapezius, and sternum (Shutter, 2014). Noxious stimuli is applied to the core body structures to test for responsiveness of the corticospinal, rubrospinal, and vestibulospinal motor pathways (Shutter, 2014).

Dysfunction of cranial nerves 2 through 10 will also indicate brain death in a patient. "Cranial nerve dysfunction occurs later in the progression of severe intracranial hypertension because of increasing pressure on the brain stem, tissue distortion, and deformation of cranial nerve roots (Arbour, 2013, p.30). Pupillary reflex (assessment of cranial nerve 2) can be assessed by shining a flashing light or using a pupilometer (Shutter, 2014). In brain death the pupils are nonreactive, dilated, and in midposition (Arbour, 2013).

Cranial nerves 3 (oculomotor), 4 (trochlear), and 6 (abducens), can be evaluated by testing for an oculocephalic reflex (Doll's Eyes Phenomenon) (Shutter, 2014). In absence of brain death a patients eyes will turn conjugately towards the opposite side of the turned head, in brain death there is no eye movement (Shutter, 2014). Cranial nerve 8 can be tested by performing a cold caloric test (Arbour, 2013). In brain death there will be no eye movement after irrigating the ear canal with 60ml of ice water (Shutter, 2013). No eye movement when testing the oculocephalic and oculovestibular indicate an injury to the pons and midbrain (Arbour, 2013).

A patient does not have basic reflexes such as cough, cornea, and gag in brain death (Shutter, 2014). These reflexes are controlled by cranial nerves 5, 7, 9, and 10 (Shutter, 2014). A cough reflex can be assessed by using deep endotracheal suctioning and a corneal reflex can be assessed by lightly swiping a cotton swab over the corneal and watching for movement of the eyelid (Arbour, 2013; Shutter, 2014). Gag reflex can be assessed by stimulating deep posterior pharynx reflexes, using a suction device or tongue blade (Arbour, 2013; Shutter, 2014). Absence of cough, cornea, and gag reflexes indicate an injury to the pons, Medulla, and midbrain (Arbour, 2013).

The presence of apnea is one of the most confirmatory signs of brain death (Shutter, 2014). The brain's ability to cause breathing is tested by performing a CO2 challenge (Shutter, 2014). Specific criteria must be met prior to initiating apnea testing, if criteria is met close monitoring is needed to minimize risk during apnea testing (Arbour, 2013). Strict guidelines are set and must be followed in order to safely and adequately perform apnea testing (Shutter, 2014). Oxygen desaturation, unstable blood pressure, or unstable cardiac rhythms are indications for stopping apnea testing (Arbour, 2013). Findings consistent with brain death include no chest and abdominal movement for 8 to 10 minutes, and PaCO2 levels increasing 20mmHg or greater, or reaching 60mmHg (Shutter, 2014).

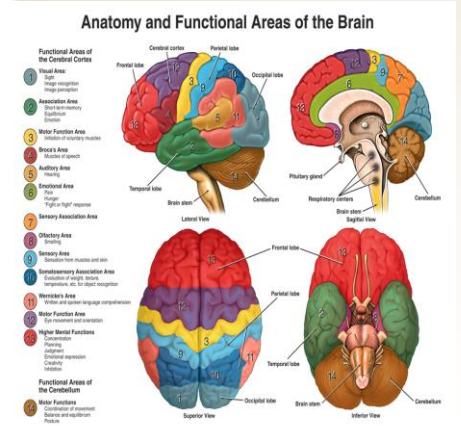


*Figure 2.* Anatomy and functional areas of the brain. Adapted from *The Dana Foundation, 2014, Retrieved from* http://www.dana.org/News/Details.aspx?id=43515. Copyright 2014 The Dana Foundation.

## Signs and Symptoms of Brain Stem Death

- **GCS score of less than three**
- **Absence of oculocephalic response to head turning (no eye movement)**
- **Fixed pupils**
- **Absent cough, corneal, and gag reflexes**
- **Absence of cold caloric responses to irritation of external auditory canals**
- **Absence of respiration (as confirmed by positive apnea test)**

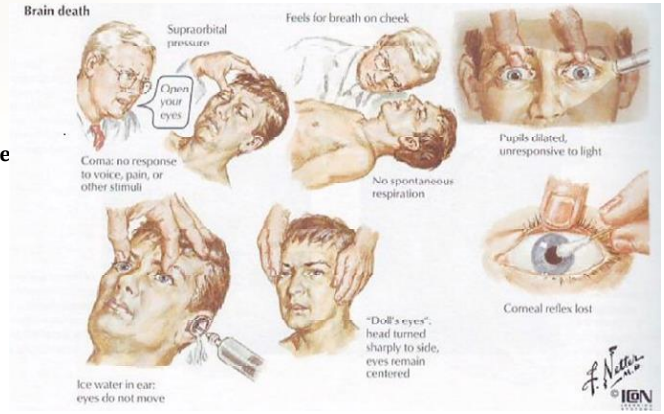


*Figure 3.* Assessment of Brain death. Adapted from *Hirntod-Diagnose*, by F. Netter, 2013. Retrieved from http://www.dwl.de/index.php?Hirntod%2BDiagnose. Copyright 2013 Compumedics Germany.

Consulting palliative care offers families support and helps meets the needs of a patient's end-of-life care. Lloyd-Williams, Morton, and Peters (2009) demonstrate that families dealing with brain death benefit greatly from palliative care approaches. When the patient has been officially declared brain dead the family is faced with questions regarding plan of care. Specialist such as palliative care and LOOP should be utilized to help approach the family members on whether they would like to consider organ donation (Siminoff, Agyemang, & Traino, 2013). Siminoff et al. (2013) states "effective communication that is delivered by trained, caring requesters at the appropriate time, in a supportive environment, and allows sufficient time for families to make an informed decision, optimizes the request process" (p.99)

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