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Brain Death Determination
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INTRODUCTION
Patient MR was a seventeen year old female involved in a motor vehicle collision. When the patient arrived at the emergency department, she was unresponsive and had severe brain injury leading to declaration of brain death. The pathophysiology behind the traumatic brain injury and the pathophysiology of brain death determination can be complex and unique for each patient. The case reflects the concept and use of a brain tissue monitoring device, and the tests used in the declaration of brain death will be explored. When brain death is determined, the patient has the opportunity to become an organ donor. Thus, nurses are vital in helping to guide patients or organs to become available. This is a huge healthcare concern that may be decreased if the general public is more educated regarding the process of brain death determination. By having a better understanding of brain death, the hope is that there will be more people willing to make the decision to become an organ donor. In order to address the enormous need for organ donors, there has to be an understanding of brain death testing at the cellular level.

CASE PRESENTATION CONTINUED
The severe global edema of the brain occurred due to acceleration-deceleration forces. The brain is composed of millions of cells that require adequate respiration to oxygenate the brain sufficiently. The brain accelerates at time of impact and quickly decelerates within the skull causing axonal shearing [8]. This global axonal shearing is seen when a patient is treated with a traumatic brain injury. Shearing alone can be a mechanism for brain edema but MR also had inadequate breathing and blood loss due to her other injuries which also contributed to the high intracranial pressure within the white matter of the brain. The combination of the excessive intracranial pressure coupled to function correctly and leads to an influx of intracranial edema which results in decreased cerebral perfusion pressure (CP), decreased cerebral perfusion pathway and then pyrrotena the kloa cycle and takes part in the synthesis ATP of molecules. Without lack of oxygen pyruvate is transformed to a lactate [6]. Lactate only further stimulates sodium influx into the cell by stimulating the sodium/potassium pump [8]. Once the edema occurs blood flow to the brain is inhibited. One mechanism to measure cerebral blood flow is to calculate cerebral perfusion pressure (CPP) [3]. CPP measures the pressure gradient between the mean arterial pressure (MAP) pushing blood through the cerebral circulation against the opposing intracranial pressure (ICP) [3]. For most traumatic brain injury patients, it is recommended to maintain the CPP between 50-70 mmHg [3]. CPP is maintained by increasing MAP and decreasing ICP. MAP is increased by use of vasoressors [6]. In the case of the MR, the vasopressor used was norepinephrine. The MAP was also increased by use of 0.9% normal saline boluses within the first twelve hours in the ICU. Mannitol and 3% saline were used in the attempt to decrease ICP below the desired 20 mmHg. Both of these solutions work to move fluid from the extracellular to the intracellular space which in turns decreases edema [8]. As earlier stated a Licox monitoring system was placed and measured brain tissue oxygenation (PbtO2). The Licox monitor uses a catheter which is inserted into the white matter of brain through a bare hole in the skull as seen in figure one. The catheter is placed in the white matter of the brain institution but generally a PbtO2 maintained greater than or equal to twenty is the desired goal and numbers below twenty are strongly correlated to a poor prognosis or outcome [6]. Maintaining MR's ICP below 20mmHg, CPP at seventy or above, and PbtO2 above twenty was a very difficult fight given the high ICP and difficulty in the brain's ability to move fluid to the I.C.U.

During a routine hourly neurological assessment, the bedside nurse noticed that bilateral pupils were fixed and dilated, she had no cough, gag, or corneal reflexes, was unresponsive to painful stimuli, and no response to pain in the lower extremities.

Brain Death Testing
Brain death is defined as the, “irreversible cessation of all functions of the entire brain, including the brain stem” [9]. In order to determine brain death there are multiple tests that physicians perform. The brainstem reflexes which include oculovestibular, oculovestibular (cold calorics), corneal, cough, gag, and pupillary light response are usually tested first [1]. The oculovestibular reflex tests for CN III, IV, and VIII and also tests the functionality of the pons and midbrain [1]. The oculovestibular reflex testing is performed by turning the patient’s head side to side and observing the movement of the eye [1]. The Oculovestibular reflex is interrupted and also tests the functionality of the pons and midbrain [1]. This test is completed by irrigating each ear can with 50ml of ice water and observing brainstem activity [1]. The corneal reflex tests for CN III, VII, and the functionality of the pons [1]. This test is completed by lightly brushing the eye and observing for corneal reflection of the pupil [1]. The cough and gag reflex tests for CN IX, X, and functionality of the medulla [1]. The gag test is achieved by stimulating the deep posterior pharynx with oral suction, and the cough test is used deep endotracheal suctioning [1]. The pupillary reflex tests for CN II, III, and the functionality of the midbrain [1]. This is completed by darkening the room and then shining a light into each eye and assessing for contraction of the pupil [1]. MR did not have any response to any of the above mentioned tests. A lack of response in these tests further validated the decision to apply the apnea test. This test involved obtaining a baseline arterial blood gas (ABG) and then removing the patient from the ventilator and placing a carotid into the endotracheal tube at 10 min [1]. The apnea test usually takes 8-10 minutes in duration and during this time the physician is assessing respiratory movement of the chest or abdomen [9]. After approximately 8 minutes another ABG is obtained and the physician is looking for either a Pco2 above 60 or an increase of the Pco2 by 20mmHg from the baseline ABG [9]. If there is no respiratory movement and an increase in the Pco2 then the apnea test is considered positive and this supports the clinical diagnosis of brain death [9]. The apnea test is completed by increasing the rate of CO2 to cause the respiratory centers in the medulla to initiate breathing in order to clear the CO2 [9]. Unfortunately MR was not a candidate for being able to perform the apnea test, the neurosurgery team decided to complete a nuclear flow study. This type of tests determines if there is blood flow to the brain. The patient is placed under a collimator with cameras that are anterior and parallel to the patient’s face [4]. A radiopharmaceutical bolus is administered to the patient via an IV and pictures are taken to capture the blood flow up to the brain [4]. The bolus is then observed on the computer and the health care provider can see the flow across the anterior and middle cerebral arteries [4]. When there is no blood flow to the brain, the bolus is seen through the carotids but stops at the base of the skull [4]. The bolus is not seen through the anterior and intracranial pressure exceeding CPP which is supplied by the internal carotids [4]. The bolus is transported to the external carotids which are also carried to the anterior and middle cerebral arteries [4]. If the patient was not a candidate for this test, the patient was later confirmed to be brain dead and her family made the decision to proceed with the organ donation process.

NURSING IMPLICATIONS
This case highlights the important role that nurses play in the care of a patient with a traumatic brain injury (TBI) that leads to brain death. It is imperative that nurses be aware of the mechanism of injury leading to the TBI and how this can affect the response of the brain. The bedside nurse needs to also be aware of the goals of therapy and how to measure these goals. Therefore, the nurses need to be educated not only on the monitoring equipment but also the meaning of the numbers produced by that equipment. Nurses are integral in the early recognition of the patient that is progressing to brain death and this early recognition leads to more stability of the patient and increased organ function during organ procurement [1]. The brain is very complex and often the signs and symptoms can be confusing, and it is imperative that the bedside nurse works in close conjunction with the neurosurgery team to determine what the clinical signs mean for the patient's care. The nurse needs to pay close attention to even the slightest changes in a patient's exam because it is important. The nurse needs to pay close attention to even the slightest changes in a patient's exam because it is important. The nurse needs to pay close attention to even the slightest changes in a patient's exam because it is important. The nurse needs to pay close attention to even the slightest changes in a patient's exam because it is important.

CONCLUSION
Patient MR was involved in a motor vehicle collision that resulted in a traumatic brain injury. By understanding the brain’s response to an acceleration-deceleration injury the patient’s care was more cognizant of the complications that could occur. Due to the patient’s exam and scans, a Licox monitoring system was placed and measured brain tissue oxygenation to have a real time picture of the status of the brain at a cellular level. With this vital information, intervention could be made first to stop any cerebral edema was worsening. Unfortunately for this patient, the brain injury was too severe and she progressed to brain death. With the declaration of brain death, the patient’s family authorized an organ donor and save the lives of numerous individuals on the national donor list.

REFERENCES
[7] ADDITIONAL RESOURCE

Figure 1: Illustration of Licox in place within the brain parenchyma [6].

Figure 2: Nuclear flow study that illustrates no cerebral blood flow and the classic “hot nose” sign [1].