Hypoxic- ischemic encephalopathy (HIE) is a major contributor to neonatal death and morbidity. As estimated 23% of the million neonatal deaths and 8% of all deaths at <5 years of age throughout the world each year are associated with signs of asphyxia at birth (19, 20). Perinatal asphyxia (PA) is a complex event resulting from a combination of factors that may lead to hypoxic ischemic injury (HII). The precise cause of this injury is not clearly defined (21, 22). The brain is the most susceptible organ to PA. The brain's metabolic rate is high, and its metabolic demands exceed its oxygen supply for long enough, the brain vascular auto regulation will fail. This, combined with low glycogen stores, will result in increased cerebral blood flow and potential ischemic brain injury (23, 24). The brain is also quite susceptible to hypoxia, but the combination of hypoxemia and perfusion as seen in most PA cases results in hypoxic ischemic injury (HII).

Brain assessment and monitoring of these infants is usually done by Electroencephalography (EEG), Infrared Spectroscopy (bSSD) and Magnetic Resonance Imaging (MRI) of the brain (15, 16). Cerebral and abdominal ultrasound (US) are performed in neonates, as part of the clinical care to save organs from irreversible damage. A recent study described the role of Color Doppler Imaging (CD) and Dynamic Color Flow Quantification in the assessment of cerebral and abdominal perfusion (17, 18). Our unpublished data regarding color Doppler perfusion measurements performed in 28 neonates with HIE, during hypothermia, provided important information.

Hypothermia has been used more frequently in infants with moderate to severe encephalopathy. It results in improved neurological outcomes (19). Moderate to severe HII (16-18).

As mentioned above, the brain in its entirety is affected by post asphyxial events. Its vulnerability to hypoxia and ischemia is due to its high metabolic activity. The brain consumes about 20% of the total oxygen supply and 25% of the total cardiac output. The brain has a limited ability to store energy in the form of glycogen and creatine phosphate. It is almost completely unable to store oxygen. As a result, any decrease in the availability of oxygen leads to a rapid decrease in cerebral metabolism. This is evident from the high cerebral metabolic rate. The brain is also highly dependent on the continuous supply of oxygen and glucose. It is particularly vulnerable to hypoxia because it has a high metabolic rate and relies on aerobic metabolism for its energy needs. Any decrease in the supply of oxygen or glucose can lead to a rapid decrease in cerebral metabolism, which can result in irreversible damage.

During the post-asphyxiative period a consistent observation has been a marked increase in cerebral blood flow which continues for several hours and may not decline toward baseline. Importantly, a delayed increase in cerebral blood flow may result with a significant delay (15, 16). After 24 hours of post-asphyxia, and with loss of metabolic function or death (25, 26). This loss of function is called necrosis and is responsible for secondary brain injury. Indeed, neuronal damage is established to a substantial degree by mechanisms occurring during this period. HIE-infants’ high levels of cerebral blood flow measured at 12 - 24% of the flow associated with normal neurological outcome (26, 27). It is well known in the literature that involvement of basal ganglia and thalami is associated with poor outcome (28, 29).

Our unpublished data showed that increased cerebral perfusion intensity to the basal ganglia in neonates with HIE, treated with hypothermia, was associated with improved neurological outcome (21, 22). CPI is expressed in cm/sec and is calculated by multiplying the mean velocity of flow pixels and flow velocity during the heart cycle. CPI is used to assess and quantify tissue perfusion in different organs and systems (30, 31).

CPI quantification with dynamic CD opens a window to better understand reperfusion injury in HII.

Therapeutic hypothermia has been used more frequently in infants with moderate to severe encephalopathy. It results in improved neurological outcomes (19). Moderate to severe HII (16-18).

Multiple organ failure occurs in 50-60% of neonates with severe HIE (13). Blood flow redistribution to the brain, heart and adrenal glands with decrease in flow to the kidney, bowel and skin plays an important role in the pathophysiology of HIE. Therefore, US is a non-invasive modality, by the bedside that can provide comprehensive imaging of multiple organs and systems. Our exhibit is a pictorial review with illustration and discussion of US and CDS perfusion intensity findings of the brain and abdomen in neonatal HII with pathologic correlation.