Andrology & Gynecology: Current Research

Extended Abstract

Color doppler in fetal hypoxia: An aid in diagnosing, managing and timely termination

Kuldeep Singh
Ultrasound and Color Doppler Clinic, India

The uteroplacental and fetoplacental circulations can be assessed by color Doppler a non-invasive method for understanding and studying fetal circulations. The uterine artery flow tells us the status of the uteroplacental circuit. The umbilical artery, middle cerebral artery, descending aorta, ductus venosus and umbilical vein study tells us the fetal adaptation to any hypoxic insult. With impaired placentation causing changes in the uterine artery one needs to be carefully surveying the fetal circulation for any adaptive changes. With hypoxic insult the blood flows preferentially to vital organs like the brain, heart and adrenals with compensatory shunting from the non-vital organs the abdominal viscera and lower limbs. The three ratio systolic/diastolic ratio, pulsatility index and resistive index are markers of resistance and thus are reflecting impedance values which are inversely proportional to the amount of blood flow in the respective vessel or organ. So brain sparing causes a high PI and brain edema would finally cause a rise in the Middle cerebral artery PI. This tool finally helps us to fine tune the administration of steroids in a premature fetus and timely termination of pregnancy to reduce the stay of the neonate in the ICU and reduce neonatal morbidity and mortality. Fetal hypoxia, oxygen deficiency in the tissues, of any cause leads to a conversion from aerobic to anaerobic metabolism, which produces less energy and more acid. If the oxygen supply is not restored, the fetus dies. Hypoxia may result from Reduced placental perfusion with maternal blood and consequent decrease in fetal arterial blood oxygen content due to low pO2 (hypoxemic hypoxia), Reduced arterial blood oxygen content due to low fetal hemoglobin concentration (anemic hypoxia), Reduced blood flow to the fetal tissues (ischemic hypoxia). Both respiratory and metabolic acidemia increase with hypoxemia. In umbilical venous blood, mild hypoxemia may be present in the absence of hypercapnia or acidemia. In severe uteroplacental insufficiency, the fetus cannot compensate hemodynamically and hypercapnia and academia increase exponentially. The carbon dioxide accumulation is presumably the result of reduced exchange between the uteroplacental and fetal circulations due to reduced blood flow. The association between hypoxemia and hyperlacticemia supports the concept of reduced oxidative metabolism of lactate being the cause of hyperlacticemia, and, under these circumstances, the fetus appears to be a net producer of lactate. Hypoxemic growth-restricted fetuses also demonstrate a whole range of hematological and metabolic abnormalities, including erythroblastemia thrombocytopenia, hypoglycemia, deficiency in essential amino acids, hypertriglyceridemia, hypoinsulinemia and hypothyroidism.