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Non-Immune Hydrops

Hydrops is the accumulation of extravascular fluid within two or more cavities of the fetus within the utero which may include ascites, pleural or pericardial effusions or skin or trunk edema, anasarca and placentomegaly appearing in advanced stages. Polyhydramnios develop due to pressure on the esophagus and decreased swallowing of amniotic fluid. Hydrops result from various underlying congenital anomalies that cause increased central vascular pressure, decrease lymph flow or decreased plasma oncotic pressure leading to a net imbalanced of fluid movement between the intravascular and interstitial compartments. Non-immune hydrops (NIH) is the most common cause of hydrops in the fetus since Rh alloimmunization or immune hydrops is rare due to routine immunization of Rh negative mothers. Congenital infections account for up to 8% of cases of NIH. NIH can occur in the fetus with chest occupying lesions or highly vascularized tumors that compress the mediastinum or increase cardiac demands respectively. Chest occupying lesions compress heart, SVC, IVC or ductus venosus leading to impaired atrial venous return and low output cardiac failure. Vascularized tumors causes high output cardiac failure from arteriovenous shunting and marked vascularity. Either way the perinatal mortality of NIH is very high. Prenatal ultrasound and echocardiography diagnose, predict outcome and dictate management of fetus with NIH. Patients with NIH who are able to have fetal intervention for their underlying condition benefits from improved survival especially when hydrops resolve ad there is no preterm delivery. Echocardiographic findings of NIH deterioration are the best predictors of the need of fetal surgical resection for fetuses with high-risk lung masses. Well-recognized causes of recurrent NIH are homozygous alpha-thalassemia and metabolic storage disorders such as mucopolysaccharidosis, Gaucher's, sialidosis and gangliosidosis. It is important to detect NIH early, diagnose the underlying cause and institute appropriate treatment. There is need for autopsy of all fetuses or neonates who die from NIH.

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Laparoscopic CO2 Embolism

Laparoscopic procedures using carbon dioxide as insufflating gas can alter circulatory, pulmonary, renal, splanchnic and endocrine function in some patients. Cardiac arrest has been reported in one of every 65,000 laparoscopic procedures with a mortality rate of 28%. Cardiac arrest is related to a vasovagal response after rapid peritoneal distension and CO₂ gas embolism as detected by transesophageal echocardiography (TEE). CO₂ embolism is a very rare but potential serious complication. It is caused by entrapment of carbon dioxide in an injured vein, artery or solid organ resulting in blockage of the right ventricle or pulmonary artery. Laparoscopic CO₂ embolism has been reported in various surgical procedures. Most serious cases occur during the beginning of the procedure due to Veress needle misplacement directly into a vein or solid organ. Late onset embolism is due to an injured vessel in the abdominal wall or operative site. Clinical manifestations depend on volume of CO₂ entering the circulation and that which is removed with ventilation. It causes a gas lock effect obstructing right ventricular ejection, right and left cardiac failure, paradoxical embolism with or without a patent foramen ovale, arrhythmia, pulmonary hypertension and cardiovascular collapse. CO₂ embolism does not cause the bronchoconstriction or changes in pulmonary compliance seen during air embolism. The patient develops hypotension, dyspnea, cyanosis, tachycardia, bradycardia, arrhythmia and asystole. TEE is the most sensitive method to diagnosed laparoscopic CO₂ embolism. Other less sensitive methods include transesophageal or precordial Doppler and ETCO₂. Preventive measures include correct position verification of Veress needle, use of low insufflating pressures, reverse Trendelenburg position and increasing the end-expiratory pressure. Management must be expeditious and include discontinuing CO₂ insufflation and releasing the pneumoperitoneum, hyperventilating the patient with 100% oxygen, volume expansion to elevated the CVP, vasopressors, inotropic agents to maintain cardiac output, and hyperbaric oxygen therapy specially for neurologic deficit caused by cerebral gas emboli.

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Vacuum Bell Device

Pectus excavatum is the most common chest wall deformity in children occurring in approximately one in 300 live births. Initially pectus excavatum in children was managed surgically using the open Ravitch technique until Nuss developed a minimal invasive technique using a bar in 1998 to repair satisfactorily the defect. The Nuss technique is operator dependent with a small but significant number of potential complications associated with a learning curve and number of cases performed. Conservative management of pectus excavatum can be carried out using a glass bell to elevate the sternum called the Vacuum bell (VB). The Vacuum bell is a suction cup that creates a vacuum on the anterior chest wall and is activated by a patient-controlled hand pump. There are three types of VB - 16, 19 and 26 cm in diameter and a model fitted for women. The device is used at home for a minimum of 30 minutes twice a day during 4-6 weeks afterward it can be used up to a maximum of several hours daily. VB therapy is indicated in patients with mild pectus excavatum or who wish to avoid a surgical procedure. It is also used in preparation for surgery, if a surgical implantable bar has to be removed earlier than scheduled, and as intraoperative vacuum device to lift the sternum away from the heart during the Nuss procedure and avoid the dreaded complication of heart rupture. Contraindications for VB include musculoskeletal disorders, vasculopathies, coagulopathies and cardiac disorders. Complications of VB therapy include subcutaneous hematoma, petechial bleeding, dorsalgia and transient paresthesia of the upper extremities during application of the device. For this therapy to have success the child must be motivated and be compliant with treatment. All children are encouraged to continue sporting activities and physiotherapy in order to improve body control. VB therapy is recommended to be started before the age of 10 years. Duration of treatment can be from 12 to 36 months.

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