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Atrophic Testis

Atrophic testes refer to a testis that has diminished in size and is accompanied by loss of function. An atrophic testis can be the result of perinatal torsion, cryptorchidism, trauma, previous surgical procedure, orchitis and steroid use. Most atrophic testes are abnormal due to a mechanical event shunting circulation rather than maldevelopment or iatrogenic. Anabolic steroids can cause testicular atrophy by reducing the amount of luteinizing hormone produced by the pituitary gland. Repair of inguinal hernia after incarceration can also cause testicular atrophy if vascular testicular occlusion occurred for en extended period of time. Testicular atrophy can occur in almost 50% of cases of non-palpable undescended testes. During perinatal descent the testis circulation is entrapped causing the atrophy or vanishing of the testis. They have sometimes been called vanishing testes since a remnant nubbin is found during inguinal or abdominal exploration. Very few of these remnants contains seminiferous tubules and even fewer shows viable germ cells. Contralateral testicular hypertrophy strongly indicates an atrophic testis in the other side. Ultrasonography can determine the significant smaller size of the affected testis and determine if is hypoechoic, a sonographic characteristic of the atrophic testis. The management of the atrophic testis is controversial. Removal of the remnant and placement of a prothesis is an alternative in children. With growth this prothesis will need replacement, hence placement during final adolescent growth years is more prudent. The risk of malignant degeneration of the testicular remnant is extremely low to justify surgical removal.

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Tension Gastrothorax

The term tension gastrothorax originally appeared in the literature as a complication of traumatic rupture of the diaphragm producing mediastinal shift due to a distended intrathoracic stomach. Congenital or acquired diaphragmatic defects can cause a tension pneumothorax. The two groups of children that can be affected by a tension gastrothorax include those with congenital diaphragmatic hernia with late presentation and traumatic diaphragmatic hernias the result of a previous accident. Most cases of tension gastrothorax occur around the five years of age in cases with an existing congenital defect. Vast majority are left-sided because the liver buttresses the right side. Increased intraabdominal pressure or negative intrathoracic pressure leads to herniation of the stomach into the chest. Respiratory symptoms initially followed by abdominal pain and vomiting develops. Other findings are tracheal deviation, reduced breaths sound, dullness or resonance and a displace cardiac apex. Tension gastrothorax can be erroneously interpreted as a tension pneumothorax leading to increase morbidity and mortality during treatment. The chest film can be diagnostic demonstrating a large air-filled structure with or without a fluid level within the left hemithorax causing apical collapse of the lung. Emergency management requires initial decompression with a large-bore nasogastric tube. If this fails transthoracic needle decompression of the stomach can be tried. Urgent definitive management requires surgical reduction of the intrathoracic stomach and repair of the diaphragmatic defect which can be accomplished preferably by laparotomy as it hasten quick reduction of stomach and repair of the diaphragmatic defect. Thoracotomy or thoracoscopy has also been utilized less frequently. Morbidity relates to pulmonary collapse, shock, bowel injury and sepsis due to gastric perforation in the thorax.

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Adrenalectomy in Wilms Tumor

Wilms tumor or nephroblastoma is the most common intraabdominal malignant solid tumor in children. It is managed with surgery, adjuvant chemotherapy and radiotherapy. Surgical management consists of radical nephrectomy when appropriate in many cases removing the adrenal gland concomitantly with the tumor. Adrenal involvement in patients with Wilms tumor is rare and difficult to predict. The decision to remove the

ipsilateral adrenal gland has been left to the judgement of the operating surgeon at the time of nephrectomy, and is likely based on the size and location of the primary tumor, ease of adrenalectomy, and suspicion for adrenal involvement Routine adrenalectomy does not confer a benefit for oncologic control (event free survival) when it is feasible to spare the adrenal gland. Intraoperative tumor spillage rates are higher in patients undergoing concomitant adrenalectomy. Patients in whom adrenalectomy was performed tended to have larger tumors than those in whom the gland was left in situ. The histopathologic status of the adrenal gland with tumor does not directly affect the oncologic outcome. Based on the low rate of adrenal involvement, and lack of apparent oncologic benefit to adrenalectomy concurrent with nephrectomy routine adrenalectomy does not appear to be mandatory. Preserving the adrenal gland was not associated with an increased risk of local recurrence. The above appears to hold truth to renal cell carcinoma management in adults.

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