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Bell=s Palsy

Bell's palsy is an acute idiopathic paralysis of the VII cranial nerve (facial nerve) resulting in inability to control facial muscles on the affected side. Bell-s palsy is a non life-threatening disorder that can cause significant impact in the life of the child. Other conditions that can cause fascial paralysis include brain tumor, head trauma, meningitis and Lyme-s disease. The mean age of involvement is eight years with a similar sex predilection. It is thought that an inflammatory condition leads to swelling of the facial nerve and compression in the narrow bone canal leading to nerve inhibition, damage or death. No readily identifiable cause for Bell's palsy has been found, though acute immune demyelination triggered by a viral infection may be responsible. The school nurse may be the first person to assess facial palsy and muscle weakness in children. Careful diagnosis and workup using head CT and MRI is warrant in all cases to exclude other serious causes of fascial paralysis. MRI enhancement of the distal intrameatal and labyrinthine segments of the nerve canal are specific for facial nerve palsy. Complete spontaneous recovery is seen in almost 70% of patients within six weeks of onset, but up to 30% have delayed or incomplete recovery. Use of steroid or antiviral therapy is controversial since most children recover without treatment. Physiotherapy, in the form of transcutaneous peripheral nerve stimulation, has an important role.

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Shoulder Dislocation

Shoulder dislocation in infants most commonly occurs in the newborn period, directly after delivery. Breech presentation, macrosomia, prolonged labor and assisted birth are risk factors associated with shoulder dystocia. Shoulder dystocia associated with brachial plexus palsy occurs in approximately 1.5% of all births. Associated with brachial plexus palsy, posterior shoulder dislocation and subluxation can develop directly after birth or even one year later due to the glenohumeral deformity that results

from the persistence muscle imbalance. Mean age at time of diagnosis is six months. Diagnosis of shoulder dislocation is based in physical examination, radiographs, ultrasound and MRI. Specific clinical signs include asymmetry of skin folds of the axilla or the proximal aspect of the arm, apparent shortening of the humeral segment, a palpable asymmetric fullness in the posterior region of the shoulder, or a palpable click during shoulder manipulation. All children with a brachial plexus birth lesion should be screened, above the assessment of neurological recovery, during the first year of life for posterior shoulder dislocation since such a condition may occur in 7% of children with a brachial plexus birth lesion. Management consists of prompt closed reduction of the shoulder by traction combined with adduction of the upper arm and countertraction with a gauze encircling the chest. The functional outcome is related to the severity of the neurological lesion, the duration of the dislocation and onset of deformity.

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Yolk Sac Tumor

Yolk sac tumor, also known as endodermal sinus tumor, is a common phenotype of testicular malignancy arising in descended testes. This tumor does not arise from the yolk sac, as the name implies, but from the germinal epithelium of the testis which has the ability to mimic the development pattern found in the yolk sac. Painless unilateral testicular enlargement is the most common presenting symptom in children. An associated hydrocele can delay the correct diagnosis. Ultrasound and CT scan will help suggest the diagnosis and extension of tumor. The definitive diagnostic step is surgical exposure of the testis through an inguinal incision, vascular exclusion and high cord ligation removal for histologic examination. Microscopy reveals an intermingle of epithelial and mesenchymal elements in a characteristic embryoid organoid pattern. Yolk sac tumor secretes alpha feto-protein, a marker helpful in the follow-up of these patients. The tumor spreads to lymph nodes and hematogenous (lung). After surgery the need of chemotherapy, radiotherapy or retroperitoneal lymph node dissection will depend on the presence of residual micro- or macroscopic disease. Patients with localized disease and normal postoperative serum alpha-fetoprotein levels do not benefit from retroperitoneal node dissection, postoperative abdominal irradiation or chemotherapy. Patients with retroperitoneal nodal involvement, widely metastatic disease or recurrent disease can be treated successfully with chemotherapy and in some cases with radiation therapy. For the few who do relapse, treatment at the time of relapse is curative for the majority.

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