Ovarian Tumors
Ovarian tumors are uncommon childhood malignancies (1%) characterized by recurrence and resistance to therapy. Aggressive surgery is limited to avoid compromising reproductive capacity and endocrine function. Low incidence and need of multinodal therapy encourages referral to centers dealing with effective cancer therapy. The most common histology is germ cell: dysgerminoma, teratoma, and endodermal sinus tumor. This is followed by the sex-cord stroma tumors with a low incidence of malignancy. They can cause feminization (granulosa-theca cell) and masculinization (androblastoma). Other types are: epithelial (older adolescent), lipid-cell, and gonadoblastoma. Ovarian tumors present with acute abdominal symptoms (pain) from impending rupture or torsion. They also cause painless abdominal enlargement, or hormonal changes. Preop work-up should include: human chorionic gonadotropin (HCG) and alpha-fetoprotein (AFP) levels. Imaging studies: Ultrasound and CT-Scan. The most important prognostic factor in malignant tumors is stage of disease at time of diagnosis. Objectives of surgery are: accurate staging (inspection of peritoneal surfaces and pelvic organs, lymph node evaluation), washing and cytology of peritoneal fluid, tumor removal, and contralateral ovarian biopsy if needed. Chemotherapy consists of: bleomycin, cis-platinum, and vinblastine. Radiotherapy is generally not effective, except in dysgerminoma. Elevation of tumor markers (AFP or HCG) after therapy signals recurrence.

Barrett’s
Barrett’s esophagus (BE) refers to replacement of the normal epithelium of the distal 2 to 3 cm of esophagus with metaplastic columnar epithelium containing globet cells. BE is rare (prevalence is increasing) in children. Evolves as a consequence of chronic GE reflux carrying an approximate 40-fold increase in development of malignancy in adult life. Three types of histological epithelium are described in BE: specialized columnar (intestinal metaplasia), junctional type (containing mucous glands), and gastric fundus type (containing chief and parietal cells) epithelium. Diagnosis of BE depends on screening endoscopic biopsies in children with reflux before and after treatment.
Consequence of reflux in BE are: development of a stricture (junction between metaplastic lining and squamous epithelium), a penetrating ulcer, bleeding, dysplasia or carcinoma-in-situ. Risk factors associated to the development of carcinoma are: length of disease, male sex, smoking history, and intestinal epithelium. Cohorts of children with an increased incidence of BE: mentally retarded, on chemotherapy, cystic fibrosis, after repair of esophageal atresia, and esophageal substitution. Asymptomatic BE children should be managed with acid suppressing medical therapy (omeprazole). Fundoplication should be offered to children with BE based on complications of GE reflux, failed medical therapy, or evidence of alkaline reflux induced BE. Successful antireflux surgery is not followed by regression of the metaplastic mucosa in BE, but can arrest the cephalad progression. Long-term endoscopic surveillance is needed to detect cases of dysplasia or carcinoma before transmural infiltration occurs.

**Juvenile Polyps**

Childhood polyps are usually juvenile (80%). Histology features a cluster of mucoid lobes surrounded by flattened mucussecreting glandular cells (mucous retention polyp), no malignant potential. Commonly seen in children age 3-10 with a peak at age 56. As a rule only one polyp is present, but occasionally there are two or three almost always confined to the rectal area (within the reach of the finger). Most common complaint is bright painless rectal bleeding. Occasionally the polyp may prolapse through the rectum. Diagnosis is by barium enema, rectal exam, or endoscopy. Removal by endoscopy is the treatment of choice. Rarely colotomy and excision are required.