General Pediatrics
Mattel Children’s Hospital Board Review Course
September 8, 2016

Lee Todd Miller, M.D.
Professor of Pediatrics
Associate Dean for Students
David Geffen School of Medicine at UCLA
Whirlwind Overview of Topics in...

1. Pediatric Ophthalmology
2. Pediatric Urology
3. Pediatric Dentistry
4. Lead Poisoning
Pediatric Ophthalmology

1. Normal Visual Development
2. Amblyopia
3. Strabismus and Pseudostrabismus
4. Congenital Glaucoma
5. Cataracts
6. Hordeolum vs Chalazion
7. The Red Eye!
8. Retinoblastoma
9. Uveitis
10. Eye Trauma
Normal Visual Development
At birth, our visual acuity is quite poor (in the range of 20/200 to 20/800 – i.e., legal blindness)
Visual acuity rapidly improves during the first 3 to 4 months of life.
Normal Visual Development

Normal Stimulation of each eye are required for

- Clear Retinal Images
- Cataract
- Strabismus

Proper Alignment
Normal visual development requires clear retinal images and proper eye alignment.
Amblyopia!
Normal Visual Development

Amblyopia

Normal Stimulation of each eye are required for Clear Retinal Images

cataract strabismus

Proper Alignment
Amblyopia occurs in approximately 2% of the general population, and is the most common cause of decreased vision in childhood.
Children are susceptible to developing amblyopia between birth and 7 years of age

(but are most susceptible during the critical period of visual development within the first few months of life)
The earlier the onset of abnormal stimulation, the greater the visual deficit.
The earlier the intervention, the better the prognosis for amblyopia!
Bruckner Test

• The single best vision screening examination for infants and young children

• It’s a simultaneous bilateral red reflex test and white reflex test
The Bruckner test gives us both a Red Reflex and a corneal light reflex simultaneously.
Forms of Strabismus

- Esotropia -- Eye turned in
- Exotropia -- Eye turned out
- Vertical Deviation -- Eye turned up and down
Phorias vs. Tropias
A **Phoria** is a tendency for the eyes to drift apart, but alignment is maintained by binocular fusion.
**Tropias** are larger angle deviations that are **fixed** (ie., they don’t drift).
Indications for Referral for Phorias

1. In any patient >2-4 months of age, if you cover one eye, remove the cover, and see that the covered eye drifted while covered, and then realigns when uncovered.

2. If the parents of any patient > 2-4 months of age report that they occasionally see eye drift.
Indications for Referral for Tropias

All patients with fixed deviations should be referred to the pediatric ophthalmologist.
What is the Clinical Significance of Ocular Misalignment?

• If it occurs early, before age 4-6 years, the child will cortically turn off, or suppress, the image from the deviated eye.
• This suppression prevents bothersome double vision.
• The deviated eye will become amblyopic and binocular fusion will be lost.
Strabismus vs. Pseudostrabismus
Congenital Glaucoma
Congenital Glaucoma:
Defined as increased intraocular pressure occurring at birth or shortly thereafter.
Congenital Glaucoma is very different than adult glaucoma.
Prior to age 3 years, **ocular enlargement** is a key clue to glaucoma.
Clinical Manifestations of Congenital Glaucoma

1. Ocular enlargement
2. Tearing
3. Photophobia
4. Corneal clouding
The increased tearing of glaucoma may be mistaken for nasolacrimal duct obstruction!
Association between Glaucoma and Sturge-Weber

Note increased risk of glaucoma in children with Sturge-Weber, especially when their port-wine stain is in the distribution of V1.
Cataracts
In contrast to adults, cataracts in children present special challenges, because early visual rehabilitation is critical to prevent irreversible amblyopia.
All newborns need a Red Reflex test before they leave the nursery!

A Red Reflex should be checked at all subsequent well child visits, as cataracts may be acquired!
Causes of Cataracts:

1. Familial (autosomal dominant)
2. Maternal infections during pregnancy (e.g., congenital rubella)
3. Idiopathic, sporadic cases
4. Associated with syndromes (Down Syndrome, Turner Syndrome)
5. Associated with metabolic problems (Galactosemia)
6. Trauma
Hordeolum vs. Chalazion
Hodeolum (Stye)

A hordeolum is a localized, purulent staph infection of one or more sebaceous glands of the eyelids (Zeis and Moll)
Chalazion

A chalazion is an inflammatory reaction in the meibomian glands secondary to breakdown of fatty secretions.
• Meibomian glands are sebaceous glands with orifices at the eyelid margins

• They secrete sterol esters and waxes that provide a covering to the tear film, thereby preventing evaporation
Pathology of Chalazion

Desquamated epithelial cells

Block the Meibomian Gland orifices

Results in stagnation of the lipids, and causes a secondary local inflammation
Hordeolum = a true infection of accessory glands of Zeis and Moll

Chalazion = a granulomatous inflammatory response within the meibomian gland
Pathology of a Chalazion
Treatment of Chalazions

1. **Hot Soaks** - to open up the clogged pores

2. **Baby Shampoo** - with a washcloth and warm water to open up the pores and dissolve the lipid plugs

3. **Erythromycin Ointment** -- treatment with antibiotics in case there’s some staph blepharitis which caused the orifice to clog up
When should we refer a patient with a chalazion?

1. If the pores don’t open up within 4 weeks of treatment
2. If the chalazion becomes secondarily infected
Prevention for Patients with Recurrent Chalazions

• May need baby shampoo eyewash, 1-2 times per day
The Red Eye
Differential Diagnosis of Red Eye

1. Trauma (corneal abrasion, subconjunctival hemorrhage)
2. Allergic diseases
3. Congenital Glaucoma
4. Infections
5. Retinoblastoma
6. Kawasaki Disease
7. Uveitis or iritis (associated with rheumatologic conditions)
8. Drug, toxin or chemical exposure
9. Trichiasis (inward turning of the eyelashes irritating the eye)
10. Foreign body
Differential Diagnosis of Red Eye

1. Trauma (corneal abrasion, subconjunctival hemorrhage)
2. Allergic diseases
3. Congenital Glaucoma
4. **Infections**
5. Retinoblastoma
6. Kawasaki Disease
7. Uveitis or iritis (associated with rheumatologic conditions)
8. Drug, toxin or chemical exposure
9. Trichiasis (inward turning of the eyelashes irritating the eye)
10. Foreign body
Infectious Causes of a Red Eye

1. Conjunctival infections (bacterial, viral, or parasitic)

2. Lacrimal duct infection or obstruction (dacrocystitis can produce tearing and ocular injection)

3. Eyelid infections: blepharitis and hordeolum

4. Periorbital or orbital cellulitis

5. Endophthalmitis
Bacterial Conjunctivitis - Presentation

1. Eyelids stuck together in the AM
2. Purulent discharge
3. Irritation
4. Concomitant otitis media
Bacterial Conjunctivitis - Microbiology

1. Non-typeable *H. flu*
2. *Strep pneumoniae*
3. *Moraxella*
4. *Staph aureus*
Viral Conjunctivitis Presentation

1. Very watery discharge
2. Irritation
3. May have foreign body sensation
4. Prominent Pre-auricular adenopathy
Viral Conjunctivitis
Microbiology

1. Adenovirus!!!
   #1 viral cause!!
2. Enterovirus
3. Herpes simplex
4. Paramyxovirus
Acute Allergic Conjunctivitis

Presentation

1. **Itching!**
2. Watery discharge with itching
3. Prominent papillae
4. May have signs and symptoms of allergic rhinitis
Allergic Conjunctivitis

1. Very common!
2. Type 1 hypersensitivity reaction
3. Scrapings show mast cells and eosinophils.
Ophthalmia Neonatorum (Neonatal Conjunctivitis)

Defined as conjunctivitis within the first 4 weeks after birth.
Infectious Causes of Neonatal Conjunctivitis

The most frequent causes of neonatal conjunctivitis in the United States are *S aureus*, *S epidermidis*, *S pneumoniae*, and *M catarrhalis*.

Ophthalmia neonatorum also can be caused by:

- *N gonorrhoeae* (DOL # 3-5)
- *C trachomatis* (DOL # 10-14)
- Herpes simplex virus (DOL # 2-14)
Pharyngoconjunctival Fever

1. The constellation of:
   a. Fever
   b. Pharyngitis
   c. Follicular conjunctivitis

2. Caused by **adenovirus**

3. Occurs in school aged children

4. Highly contagious, with community outbreaks common
Parinaud Oculoglandular Syndrome

• A rare condition characterized by unilateral granulomatous conjunctivitis, with visibly swollen ipsilateral preauricular or submandibular lymphadenopathy

• Caused by *Bartonella henselae* (cat-scratch disease).
Periorbital Cellulitis

A bacterial infection of the eyelids and surrounding soft tissues

Commonly associated with a break in the skin, but may also result from:

- Extension of a local infection, like a stye
- Commonly associated with sinusitis
- May be associated with a dental abscess
Periorbital Cellulitis
Microbiology:

The most common organisms are:

1. *Staphylococcus aureus*
2. *Streptococcus pyogenes*
3. *S. epidermidis*
4. *H. influenzae* (in the pre-vaccine era)
Orbital (Post-septal) Cellulitis

A bacterial infection that has extended into the retro-orbital space.

The most common predisposing factor is sinusitis (especially ethmoiditis).
Orbital (Post-septal) Cellulitis Microbiology

The most common pathogens are:

1. *Staphylococcus aureus* (with increasing incidence of MRSA)
2. *Streptococcus pyogenes*
3. *Streptococcus pneumoniae*
4. Anaerobes
Orbital (Post-septal) Cellulitis
Clinical Presentation

1. Ophthalmoplegia (pain with lateral eye movement, resulting in inability to move the eye or diplopia)
2. Proptosis
3. Blurred vision or change in visual acuity
4. Chemosis (conjunctival edema resulting from impaired venous drainage)
Orbital (Post-septal) Cellulitis Management

1. **Imaging Study:** CT with orbital cuts
2. **Antibiotics:** Initial treatment usually includes clindamycin + 3rd generation cephalosporin for a total of 10-14 days (inpt/outpatient)
3. **Surgical drainage** of abscess and sinusitis for severe cases or cases unresponsive to IV antibiotics
Orbital (Post-septal) Cellulitis
Complications

1. Cavernous sinus thrombosis
2. Intracranial abscess
3. Meningitis
Retinoblastoma

Retinoblastoma is a malignant tumor of the retina, and it’s the most common primary ocular malignancy in childhood.
Epidemiology of Retinoblastoma:

1. This malignant tumor of the developing retina usually occurs before 5 years of age.
2. As the most common pediatric eye tumor, it occurs in 1 in 20,000 children.
3. Average age of presentation is 13-18 months, with > 90% cases diagnosed before age 5 years.
Clinical Presentation of Retinoblastoma:

1. The most common finding is **leukocoria** (i.e., a white pupillary reflex on the RR test) (50-60%)

2. Strabismus is the 2\textsuperscript{nd} most common clinical presentation (20-25%)
Genetics of Retinoblastoma

1. Can occur *sporadically* with no family history
   - usually *unilateral*

2. Can be *autosomal dominant* with incomplete penetrance
   - usually *bilateral*

3. The gene for retinoblastomas (RB1) has been identified on the long arm of *Chromosome 13*
Prognosis and Treatment:

1. Critical to the treatment of retinoblastoma is early identification, as **cure rates are higher than 90% if the tumor is still localized within the eye.**

2. Delay in diagnosis can lead to CNS involvement and hematogenous metastasis.

3. Treatment may include:
   1. Radiation
   2. Chemotherapy
   3. Laser therapy
   4. Enucleation
Uveitis

Inflammation of any part of the uveal tract:

1. Iris
2. Ciliary body
3. Choroid
Uveitis: Signs and Symptoms:

1. Pain
2. Photophobia
3. Blurred vision
4. Conjunctivitis
5. Tearing
Definitive Diagnosis of Uveitis

Diagnosis requires a slit lamp examination.

Features on slit lamp include:

1. WBC’s circulating in the vitreous
2. Increased protein content secondary to inflammation
Conditions Associated with Uveitis

1. JIA (most especially girls with ANA⁺ pauciarticular JIA)
2. Inflammatory Bowel Disease
3. Reiter’s Disease (and other HLA-B27 haplotype ⊕ spondyloarthropathies)
4. Behcet syndrome
5. Sarcoidosis
Eye Trauma
Examination of the eye of a child who sustained eye trauma:

The first goal of the ocular examination is to determine the extent of trauma and to rule out a ruptured globe.
As a primary care practitioner, a great initial screen utilizes the mnemonic: “I-ARM.”

I = Inspection (symmetry, bleeding, squinting, sensation over cheek, etc)

A = Acuity (Snellen at 2-5 ft... or in infants, check if they fix and follow)

R = RR and Pupillary Response (check pupil size, shape, symmetry and reaction to light)

M = Mobility (EOMI... rule out entrapment from orbital blow out fracture)
Injuries that follow trauma to the eye:

1. Corneal abrasions
2. Ruptured globe
3. Laceration of the globe and surrounding structures (including the lacrimal system)
4. Hyphema
5. Foreign body
6. Dislocated lens
7. Retinal detachment
8. Orbital blow-out fracture
Intra-Ocular Foreign Body:

If there’s a penetrating injury with even the possibility of an ocular or orbital FB, a CT scan of head and orbits is indicated.
Ruptured globe:

- If rupture is suspected, apply eye shield on forehead and bony arch of forehead.
- Do not use an eye patch that will put pressure on the eyeball,
- In a jam, use a styrofoam or plastic cup!
Important Associations
Clues on Eye Exam and Eye Pathology:

- Tremulous iris.... Lens dislocation
- Peaked or teardrop pupil.... Corneal laceration (iris tissue tries to plug the corneal laceration)
- Decreased sensation over a cheek... Blow-out fracture
- Cloud moving across the visual field... Retinal detachment
Other Important Associations
Clues on Eye Exam and Eye Pathology:

• Bilateral periorbital ecchymoses..... basilar skull fracture

• 360 degree subconjunctival hemorrhage... posterior rupture of the globe

• Tearing, pain, and photophobia.... Corneal abrasion

• Abnormal pupil shape or abnormal reaction to light... consider CN 3 palsy or a ruptured globe
Definition = **Blood in the anterior chamber** (in the space between the iris and the cornea)
Causes of Hyphema

Most common cause of a hyphema is **blunt ocular trauma** = # 1 cause!

Other much less common causes include:

2. Diabetes mellitus
3. Intraocular tumors
4. Juvenile xanthogranulomatosis
Management of Hyphema:

1. **Important Pearl:** All patients suspected of having a hyphema should have an ophthalmic consultation!

2. Management usually includes bedrest, but may require surgery if increased intraocular pressure.

3. Cycloplegics may help prevent clot disruption

4. Aminocaproic acid may help prevent re-bleeding.
Complications of Hyphema

1. Late re-bleeding (3-5 days later) when the clot contracts.
2. May lead to increases intraocular pressure.
3. May also cause corneal blood staining.
Blow-out Orbital Fractures

Clues on exam include:

1. Step-off
2. Extraocular muscles not intact
3. Decreased sensation over a cheek
4. Enophthalmos (posterior displacement of globe)
Corneal Abrasion

1. Results from disruption of the corneal epithelium

2. Clinical presentation includes severe pain, tearing, photophobia and “foreign body sensation”

3. Diagnosis is by fluorescein staining – A scratch or abrasion of the corneal epithelium will result in + staining by fluorescein with a Wood’s light or a blue light
4. Management – Natural healing occurs as the existing epithelium fills in the areas of abraded epithelium. Patching now controversial and many say not indicated.

5. Referral - If the abrasion is associated with contact lens use, an ophthalmological consultation is indicated to rule out a bacterial corneal infection.
Differential diagnosis of a corneal abrasion includes:

1. A bacterial ulcer
2. Herpes keratitis
3. Retained corneal foreign body
Dislocation of the lens

Think of:
1. Marfan’s Syndrome
2. Homocystinuria
Pediatric Urology

1. Micropenis
2. Hypospadias
3. Phimosis
4. Paraphimosis
5. Cryptorchidism
6. Hydroceles and Inguinal Hernias
7. Testicular Torsion
8. Epididymitis
9. Testicular Tumors
Micropenis (microphallus)

1. **Definition** - A small, normally formed penis that is 2.5 standard deviations below the mean.

2. **Etiology** – Micropenis results from a hormonal abnormality that occurs after 14 weeks gestation.

3. The most common cause of micropenis is inadequate production of gonadotropin releasing hormone by the hypothalamus.
Micropenis (microphallus)

4. Associated Syndromes include:
   a. Kallman Syndrome
   b. Prader-Willi Syndrome

5. Management:
   Referral to pediatric endocrinology for work-up
**Definition:**
Hypospadias refers to a urethral opening that is on the ventral surface of the penile shaft, anywhere from the tip of the glans to the scrotum or perineum.

**Epidemiology:**
The incidence is about 1 in 250 male newborns.
Clinical Features of Hypospadias

1. Urethral meatus on ventral shaft of penis.

2. “Dorsal Hood” formed by abundance of preputial skin on dorsal surface and sides (and absent or thin skin on the ventral surface).

3. The more proximal the urethral opening, the more likely the penis will demonstrate ventral shortening and curvature (“chordee”).
Clinical Features of Hypospadias
Evaluation of Hypospadias

1. No imaging is usually required in the evaluation of hypospadias (as isolated upper urinary tract abnormalities are uncommon).

2. Consider additional work up for very proximal hypospadias, in association with cryptorchidism, to rule out:
   a. Mixed gonadal dysgenesis (46XY and 45XO mosaic patterns)
   b. Congenital adrenal hyperplasia
Management of Hypospadias

1. Avoid neonatal circumcision (since hypospadias repair may incorporate preputial skin into the urethroplasty)

2. Elective surgery at about 6 months of age
   a. To extend the urethra to the tip of the glans
   b. To correct any foreskin abnormality
   c. To straighten any curvature
Phimosis refers to the inability to retract the prepuce

(usually due to a somewhat fibrotic contraction of the preputial opening)
Complications include obstruction of urinary flow, with local inflammation and infection (including posthitis and balanitis).

Circumcision should be considered if there is ballooning of the foreskin with voiding, or if phimosis persists beyond 10 years.
**Definition:**
Painful constriction of the glans of the penis when a tight or phimotic foreskin is retracted behind the corona (creating a tourniquet effect)

**Management:**
Manual attempts to “reduce” the edematous prepuce may be successful, but some cases require surgical intervention (dorsal slit).
Cryptorchidism

1. A cryptorchid or undescended testis is common, with an overall occurrence rate of 4.5% of all newborns
   
   a. About 3% of all full term boys.
   
   b. About 30% of boys at 30 weeks gestation

2. Since testicular descent occurs late in gestation, it is not surprising that the incidence of cryptorchidism in premature infants is much higher.
Cryptorchidism

Undescended testes are usually in the inguinal canal, but they may also be located in ectopic locations (for example, in the abdomen, in the perineum, or in the superficial inguinal pouch).
Consequences of Cryptorchidism

The undescended testis is histologically normal at birth, but pathologic changes can be demonstrated as early at 6 to 12 months of life.

The consequences of cryptorchidism include:

a. Infertility
b. Increased risk of malignancy
c. Associated hernias
d. Torsion of the cryptorchid testes (torsion and infarction of the undescended testes occur because of excessive mobility of such testes)
e. Possible psychological effects of an empty scrotum
Please note that 15% of the tumors that occur in men with a history of cryptorchidism occur in the contralateral testis.
Management of Cryptorchidism

The great majority of undescended testes will descend within the first 3 months of life. If the testis does not descend by 6 months of life, it’s extremely unlikely that it will go on to descend spontaneously.

Laparoscopy is widely used as the initial diagnostic test in boys who have non-palpable testes.

Orchipexy is usually performed at about 1 year of age, and is the preferred treatment (rather than hormonal therapy).
Prune-Belly Syndrome (Eagle-Barrett Syndrome)

Classic triad includes:

A. Absence of the abdominal muscles
B. Abnormalities of the kidneys and urinary tract
C. Cryptorchisism
Differential diagnosis of a mass in the inguinal region

1. Hydrocele
2. Inguinal hernia
3. Trauma
4. Lymphadenopathy
5. Lymphadenitis
6. Undescended testes
7. Retracted testes
8. Tumor
Normal Anatomy and Embryology

- At 28 weeks gestation, the testis starts working its way through the internal inguinal ring. As it moves down, a diverticulum of the peritoneum, called the processus vaginalis, attaches itself to the testes.

- As the testes moves down the inguinal canal and through the external ring and into the scrotum, it pulls the processus vaginalis along with it. The portion of the processus vaginalis that surrounds the testes becomes the tunica vaginalis.
Normal Anatomy and Embryology

• The remainder of the processus vaginalis involutes by fusing together, thus obliterating the entrance of the peritoneal cavity into the inguinal canal. This involution probably occurs in the last few weeks of gestation.

• Failure of the obliteration to occur results in a variety of inguinal anomalies, including hernias and hydroceles.
Internal Inguinal Ring

External Inguinal Ring

Peritoneum

Tunica Vaginalis

Scrotum
Normal Anatomy
(in which the processus vaginalis has obliterated)
A hydrocele is a collection of fluid between the layers of the tunica vaginalis surrounding the testes.
Different Types of Hydroceles

- Normal Abdominal cavity
- Noncommunicating hydrocele
- Communicating hydrocele
- Hydrocele of the cord

Testicle
Scrotum
Clinical Presentation of Hydrocele

1. Swelling in the scrotum
2. A possible "bluish hue" to the overlying scrotal skin
3. Transillumination establishes the presence of fluid.
An **inguinal hernia** is a protrusion of abdominal content (often a loop of bowel) through the deep inguinal ring into the inguinal canal.
Clinical Presentation of Inguinal Hernias

1. Often a bulge in the groin, especially with crying or straining.

2. Note that the bulge may remain in the inguinal canal, or the hernia may extend down the canal and protrude into the scrotum.

3. On some occasions, you may be able to appreciate or feel the classic “silk sign” from the peritoneal sac in the canal.
Management of Inguinal Hernias

1. Referral to a pediatric surgeon or pediatric urologist.

2. Emergent referral for suspected incarceration or strangulation.
Hydroceles vs Inguinal hernias

The distinction is based upon the size of the opening at the sac neck.

If the opening is very small, and only peritoneal fluid egresses, the patient will have a communicating hydrocele.

If the opening is large enough, the intra-abdominal viscera may enter the inguinal canal, and then the patient will present with an inguinal hernia.
Differential diagnosis of *painless* scrotal mass:

1. Hydrocele
2. Inguinal hernia
3. Testicular tumor
4. Varicocele
5. Spermatocele
6. Testicular torsion in the prenatal period
Differential diagnosis of painful scrotal mass:

1. Trauma
2. Torsion of the spermatic cord
3. Torsion of the testicular appendix
4. Henoch-Schoenlein Purpura (HSP)
5. Incarcerated inguinal hernia
6. Epidydimitis
7. Orchitis
8. Kawasaki Disease
9. Acute bleeding into a testicular tumor
Testicular torsion vs. Testicular appendix torsion
Testicular torsion (Torsion of the Spermatic Cord)

1. Results from inadequate fixation of the testis in the scrotum, allowing excessive mobility in the testis.

2. The abnormal attachment is known as a “bell clapper deformity.”

3. The most common cause of testicular pain in boys 12+
Clinical Presentation of Testicular Torsion:

1. Acute pain and swelling of the scrotum
2. Absent cremasteric reflex.
3. Elevation of the testis may increase pain (negative Prehn sign).
Consequences of Testicular Torsion:

Spermatogenesis may be lost if >4-6 hours of diminished blood flow to the testis.

Loss of the testis (as seen to the right)
Diagnosis and Management of Testicular Torsion

Diagnosis
1. Clinical Presentation
2. High resolution doppler demonstrates absent flow to the affected testis.

Management:
Stat surgical exploration and bilateral orchipexy if the torsion cannot be manually reduced.
Torsion of the “appendix testis”

1. The “appendix testis” is a remnant of the Mullerian system on the upper pole of the testis

2. Torsion of the “appendix testis” is the most common cause of testicular pain between 2-11 years
Torsion of the “appendix testis”

1. Onset of pain may be more gradual than torsion of the spermatic cord.
2. “Blue dot sign” may provide a clue to the diagnosis: bluish hue appreciated under the scrotal skin.
Diagnosis and Management of Torsion of the “appendix testis”

Diagnosis:
1. Clinical Presentation
2. Nuclear medicine scans or high-resolution Doppler studies help distinguishtorsion of the testicular appendix from true torsion of the spermatic cord.

Management:
Treatment is supportive-- Rest, NSAID’s.
Testicular torsion vs. Testicular appendix torsion

Testicular torsion of the Spermatic Cord
The most common cause of testicular pain in boys 12+ (not common in younger boys)

Vs.

Torsion of the “appendix testis”
The most common cause of testicular pain between 2-11 years (and is rare in adolescents).
Epididymitis

1. Clinically, may present very similarly to testicular torsion in pubertal boys, but cremasteric reflex is usually present.

2. Elevation of the testis may relieve pain (+ Prehn sign).

3. Nuclear flow study shows increased flow (vs. decreased flow in testicular torsion).
Evaluation and Treatment of Epididymitis

1. Pubertal boys should be evaluated for *C. trachomatis* and *N. gonorrhoeae* and treated empirically for these organisms.

2. Antibiotics include IM ceftriaxone plus po doxycycline or azithromycin.
Note that epididymitis is rare in pre-pubertal boys, and if present, patients should be worked up for structural anomalies of the urinary tract after the acute infection is cleared.
Posterior Urethral Valves

1. Posterior urethral valve (PUV) disorder is a developmental anomaly of males in which congenital membranes obstruct the posterior urethra.

2. It is the most common cause of lower urinary tract obstruction in male neonates.
Presentation of Posterior Urethral Valves

1. PUV’s may be diagnosed on prenatal ultrasound, with a wide range of clinical presentations depending on the severity of disease.

2. If severe, newborns may have ascites, renal insufficiency, oliguria, pulmonary hypoplasia and renal failure.

3. Older infants may present with renal failure, and older children may present with minor voiding dysfunction, abnormal urinary stream, enuresis, etc.

4. Urinary tract infection is common at all ages.
Testicular tumors

1. Testicular cancer is the most common solid malignancy that affects males between the ages of 15 and 35.

2. The great majority of testicular cancers are germ cell tumors.
Clinical Presentation of Testicular Tumors

1. Painless swelling or a nodule within the testis that does not transilluminate.
2. In the minority of cases, pain may result from acute hemorrhage into the tumor.
Increased Risk of Testicular Tumors

1. Patients with a history of cryptorchidism and prior orchipexy are at an increased risk for testicular cancer in both testes (although greater in the undescended one).

2. Males who have gonadal dysgenesis and Klinefelter syndrome also have an increased risk for testicular cancer.
Pediatric Dentistry

1. Dental caries
2. Delayed dental eruption
3. Dental trauma
Dental Caries

The CDC reports that dental caries is perhaps the most prevalent of infectious diseases in our nation’s children.
Dietary Carbohydrates $\xrightarrow{Strep mutans}$ Acidic Metabolic End Products $\xrightarrow{\text{Demineralize the enamel}}$
Modification of the mother’s dental flora will impact a child’s rate of caries formation.
Nursing Bottle Caries
High Risk Groups for the Development of Caries

- Lower socioeconomic status
- Children with special health care needs
- Children who sleep with a bottle or who breastfeed throughout the night
- Children of mothers with high caries rates
Range of Eruption of Primary Teeth

While the primary teeth usually begin to erupt at 6 months of age, there is quite variability in the eruption pattern, which may range from 3 months to 16 months.
If parents are concerned about delayed dentition, inquire about the dental eruption history of family members.

Familial delayed eruption is the most likely etiology.
The differential diagnosis of delayed eruption includes:

- Hypothyroidism
- Hypopituitarism
- Rickets
- **Ectodermal dysplasia**, a disease in which patients have defects in both dental eruption and dental development, in association with alopecia and defects in sweating.
- Many syndrome are associated with delayed eruption of dentition.
Syndromes associated with delayed eruption of dentition

1. Apert syndrome
2. Cleidocranial dystososis
3. De Lange syndrome
4. Down syndrome
5. Hunter syndrome
6. Incontinentia pigmenti
7. Mucopolysaccharidosis
8. Osteogenesis imperfecta, type I
9. Progeria
Dental Trauma
Dental Avulsion

1. Definition = complete displacement of the tooth from the socket

2. Treatment

• **Treatment of Avulsion of primary tooth** – Do nothing!
• **Treatment of Avulsion of permanent tooth** – more complicated
Treatment of Avulsion of Permanent Tooth

1. Ideally, the tooth should be replaced in the socket by an adult within 30 minutes to prevent destruction of the root.

2. If the tooth can’t be repositioned, it can be transported in milk (any fat content) or saliva.

3. If an older child, tooth may be transported under the tongue.

4. If the tooth is dirty, it may be cleaned by holding it by the crown, and by running cool water or saline over it. Do not brush or scrub the root portion.
Lead Poisoning
Lead Poisoning

1. Risk Factors
2. Sources of Lead
3. Effects of Chronically Increased Blood Lead Levels
4. Laboratory Findings
5. Management of Increased Blood Lead Levels
Risk factors for Lead Poisoning

1. African American children
2. Children living in poverty
3. City dwellers
4. Children < 6 years of age
Traditional Sources of Lead

1. Lead based paints
2. Leaded gasoline
3. Lead pipes
4. Lead jewelry
5. Lead solder used to seal canned goods
Current Sources of Lead

1. Paint chips in the house
2. Paint dust in the house
3. Paint dust in the soil... brought into house... inhaled
4. Improperly glazed ceramics
5. Lead pipes if house built before 1920’s
6. Lead-contaminated soil from gasoline, if near highway
7. Nearby remodeling of older house
8. Folk medicines
Effects of Chronically Increased Blood Lead Levels

1. **Biochemical Effects**

2. **Clinical Effects**
   - A. Gastrointestinal
   - B. Central Nervous System

3. **Subclinical Effects**
   - A. Cognitive effects
   - B. Behavioral effects
• Cognitive Effects:
  Many longitudinal studies have confirmed the inverse relationship between BLL’s and cognitive function.

• Behavioral Effects:
  More inattentive, more hyperactive, and less able to follow directions
Laboratory Findings Associated with Increased BLL’s:

1. Microcytic anemia
2. Peripheral smear with basophilic stippling of RBC’s
3. Elevated serum iron levels
4. Elevated FEP
5. Elevated urinary coproporphyrin levels
Management of Increased BLL’s

1. Environmental assessment
2. Decrease hand-to-mouth transmission
3. Nutrition- Adequate Fe, Ca and Vitamin C
4. Chelation therapy if BLL > 45 mcg/dL
Pharmacologic therapy with chelating agents does not reverse neurocognitive defects in children who have lead neurotoxicity.
Chelating Agents for Increased BLL’s

1. Dimercaprol (BAL)
2. Calcium disodium methylenediaminetetraacetic acid (CaNa$_2$EDTA)
3. Succimer (DMSA, 2–3 mesodimercaptosuccinic acid)
4. D-penicillamine
THANK-YOU!