

THIRD EDITION

The American Academy of Pediatric Dentistry



THE Handbook OF PEDIATRIC DENTISTRY

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Chapter 1: INFANT ORAL HEALTH

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_Periodicity.pdf

http://www.aapd.org/media/Policies_InfantOralHealthCare.pdf Guidelines/G_

http://www.aapd.org/media/Policies_Guidelines/P_CariesRiskAssess.pdf

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I. DEFINITION

Professional intervention within six months after the eruption of the first primary tooth or no later than 12 months of age directed at factors affecting the oral cavity, counseling on oral disease risks, and delivery of anticipatory guidance

- Early intervention aimed at preventing or mitigating common pediatric oral diseases and conditions while initiating a relationship between infant, child, family and the pediatric dental caregiver
- Primary prevention of dental disease based on timely family education, instruction and motivation for behavioral changes, appropriate fluoride management, early identification of risks and tailored preventive programs
- Foundation upon which prevention of oral injuries, management of oral habits, assessment of oral development, and consideration of other individual and special needs enhance a child's opportunity for a lifetime free from preventable oral disease

II. RATIONALE

- Early oral exam, along with oral health risk assessment and anticipatory guidance are effective means of true primary prevention
- Early identification and intervention of oral health problems are cost effective and lead to satisfactory outcomes

III. GOALS

- Timely delivery of family education on caries etiology/process, appropriate oral hygiene and feeding/dietary habits for caries prevention with ultimate goal of avoiding future surgical intervention (if possible, initiate educational process during pregnancy)
- Timely consideration of fluoride management and preventive strategies as the primary dentition erupts based on individualized risk assessment
- Provide anticipatory guidance and identify high-risk children for Early Childhood Caries (ECC) at an early age (if possible, identify high-risk mothers during pregnancy)
- Establish a dental home by 12 months of age (Refer to "Policy on the Dental Home" at http://www.aapd.org/media/Policies_Guidelines/P_DentalHome.pdf)

IV. STEPS INVOLVED IN INFANT ORAL HEALTH CARE

- Record detailed medical and dental histories
- Clinical examination of oral structures in parent-assisted (knee-to-knee) position
- Counsel about caries risk factors and provide anticipatory guidance in the areas of dental and oral development, fluoride adequacy, teething, non-nutritive habits, injury prevention, dietary and oral hygiene instructions (Refer to Section V)
- Counsel about bacteria transmissibility and provide anticipatory guidance directed to the mother or other intimate caregiver in order to avoid or delay colonization

- Assess the infant's caries risk using AAPD Caries-Risk Assessment Tool (CAT) in order to address current problems, and determine individual preventive strategies and follow-up intervals (Refer to Section VII)
- Decide on supplemental procedures which may include caries risk testing, such as assay of salivary mutans streptococci (MS) levels by culture, selected radiographic examination, water fluoride analysis, consultation with other dental and medical providers and other interventions deemed necessary by a child's individual needs
- Follow-up procedures are those indicated in the “Guideline on Periodicity of Examination, Preventive Dental Services, Anticipatory Guidance, and Oral Treatment for Children”

V. ANTICIPATORY GUIDANCE

In dental anticipatory guidance, parents are given counseling in infant oral hygiene, home and office-based fluoride therapies, dietary counseling, and information relative to oral habits and dental injury prevention. Counseling of parents by providers about dental developmental changes expected to occur between their children's dental visits is an important part of preventive care. Like well-child medical visits, one of the cornerstones of the infant dental visit is to prepare parents and caregivers for future age-specific needs and dental milestones.

ANTICIPATORY GUIDANCE: SUGGESTED CONTENT GUIDE – BIRTH TO THREE YEARS			
Topic	6-12 months	12-24 months	24-36 months
Dental and oral development	<ul style="list-style-type: none"> • milestones • patterns of eruption • environmental and genetic influences • teething • infant oral cavity 	<ul style="list-style-type: none"> • occlusion • spacing issues • speech and teeth • tooth calcification 	<ul style="list-style-type: none"> • last primary tooth erupted • exfoliation • future orthodontic needs • radiographs
Fluoride supplementation	<ul style="list-style-type: none"> • F mechanisms • sources of F • choice of F vehicles • F and vitamins • toxicity issues/storage • formula and F 	<ul style="list-style-type: none"> • F dentifrice use • F in food sources • avoiding excessive ingestion 	<ul style="list-style-type: none"> • F use revisited at every interval • daily access
Non-nutritive habits	<ul style="list-style-type: none"> • pacifier use and types/safety • mouthing/oral stimulators 	<ul style="list-style-type: none"> • digit habit issues • effect on occlusion 	<ul style="list-style-type: none"> • revisit habit issues

Injury prevention	<ul style="list-style-type: none"> • signs of trauma • child abuse oral signs • emergency access instructions • implications for permanent teeth • car seats 	<ul style="list-style-type: none"> • daycare instructions • electric cord safety • replantation warning Re: primary teeth • child proofing 	<ul style="list-style-type: none"> • helmet safety • seat belts • safety network
Diet	<ul style="list-style-type: none"> • nutrition and dental health • bottle use and weaning • sippy-cup use and content • breast feeding • caries process 	<ul style="list-style-type: none"> • role of carbohydrates (juice) exposures • retention of food • review caries process • revisit sippy-cup issues 	<ul style="list-style-type: none"> • snacks • frequency issues • review caries process • role of carbohydrates (juice) exposures • revisit sippy-cup issues
Oral hygiene	<ul style="list-style-type: none"> • oral as part of general hygiene • acquisition of <i>S. mutans</i> • positioning baby for oral hygiene • special techniques 	<ul style="list-style-type: none"> • child participation • dentifrice use • Fl dentifrice for high risk 	<ul style="list-style-type: none"> • electric brushes/ toddler techniques • use of floss • continued parental participation

VI. ORAL HEALTH RISK ASSESSMENT

Systemic evaluation of the presence and intensity of etiologic and contributory caries risk factors designed to provide a disease estimation susceptibility and help in determining preventive and treatment strategies

What to address	What to ask
Medical history: pre-/perinatal history (hypoplasia), general health (healthy vs. special needs), medications (some high in sucrose)	Nutritional deficiencies in pregnancy Prematurity ($\sim < 36$ weeks gestational period) Birth weight ($\sim < 2.5$ kg) Medical problems/special health care needs (i.e. compromised salivary flow, compromised oral hygiene due to behavior problems, high-caloric diets, etc.) History of hospitalization and past/current medications
Oral hygiene: visible plaque on maxillary anterior teeth is one of the best predictors of future caries	Age brushing began? Are the child's teeth brushed daily, once in while or not yet? Who brushes the child's teeth? When are the child's teeth brushed: morning, before bedtime, morning and before bedtime and/or after meals? Any problems with positioning, child's cooperation, etc.?

<p>Infant Feeding: only formulas, breastmilk or water in infant bottles; milk is not cariogenic, but a vehicle for cariogenic substances (i.e. chocolate powder); breastmilk alone is not cariogenic, prolonged on-demand nighttime feeding associated with increased risk for caries; weaning from the bottle/sippy-cup at age 1 and from the breast as long as the mother and the child desires; breastfeeding in the 1st year of life found to be protective of future obesity</p>	<p>Breastfed/Bottle-fed? Breastfed/Bottle-fed to sleep and/or in the middle of the night? If yes, duration and frequency for each If bottle-fed, content of bottle: formula, milk, milk and sugary substances, juice/sugary drinks and/or water?</p>
<p>Dietary Habits: early introduction of unhealthy foods (i.e. sugary drinks and snacks) can alter taste preferences for foods and beverages and predispose to obesity; high frequency of sugary drinks and snacks between meals (≥ 3 times) increases caries risk; limit juice and sugary drinks daily intake to 4-6 oz and best given in open cups; best to limit sweet foods/drinks at mealtimes</p>	<p>Does the child regularly eat sweets more than 2x a day? What does the child like to snack on and how frequently? What type of container does the child usually use for drinks? Daily amount in oz during meals and/or throughout the day for the following drinks: 100% juice, juice drinks, regular/diet soda and sugary drinks (i.e. Kool-Aid)</p>
<p>Fluoride Adequacy: daily fluoride exposure through water or supplementation, and monitored use of fluoridated toothpaste (no more than a lateral smear) can be effective primary preventive procedures</p>	<p>Main water source from which the child is drinking: city water (unfiltered, Brita/Pur filter), city water (filtered, reverse osmosis), well water or bottle water? Fluoride level in the child's drinking water? Does the child take fluoride supplements? If yes, dosage and frequency Does the child use fluoridated toothpaste daily, once in a while or not yet? If yes, amount placed on toothbrush</p>
<p>Bacteria Transmission: Mutans streptococci (MS) transmission can be <i>direct</i> or <i>indirect, vertical</i> (usually from mother) or <i>horizontal</i> (within or outside of the family)</p>	<p>Does the child's mother (intimate caregiver) have any untreated decay? Does the child and mother (intimate caregiver) share the same utensils, foods and cups? Does the mother (intimate caregiver) pre-chew the child's food or kiss the child on the mouth?</p>
<p>Demographic data: low SES, low maternal educational level, and minority groups are at higher risk for ECC</p>	
<p>Teeth characteristics: white spot lesions considered severe ECC in children younger than 3 years of age; inspect for enamel hypoplasia, enamel defects, retentive pits/fissures; stained pits/fissures not common in primary dentition (possible higher risk for future cavitation?)</p>	
<p>Iatrogenic factors: use of braces or orthodontic/oral appliances provide hard, non-desquamating surfaces and serve as plaque traps</p>	

Salivary assays for MS: Ivoclar Vivadent CRT system (www.ivoclarviva.com), MSKB agar plates
Perceived risk by dental professional is reliable

VII. CARIES RISK ASSESSMENT

AMERICAN ACADEMY OF PEDIATRIC DENTISTRY CARIES-RISK ASSESSMENT*			
RISK FACTORS TO CONSIDER (For each item below, circle the most accurate response found to the right under "Risk Indicators".)	RISK INDICATORS		
	HIGH	MODERATE	LOW
Part 1 – History (determined by interviewing the parent/primary caregiver)			
Child has special health care needs	Yes		No
Child has condition that impairs salivary flow/composition	Yes		No
Child's use of dental home	None	Irregular	Regular
Time lapsed since child's last cavity	<12 months	12 to 24 months	>24 months
Child wears braces or orthodontic/oral appliances	Yes		No
Child's mother has active decay present	Yes		No
Socioeconomic status of child's caregiver	Low	Mid-level	High
Frequency of exposure to between meal sugars/cariogenic foods (include ad lib use of bottle/sippy cup containing juice or carbonated beverage)	>3	1 to 2	Mealtime only
Child's exposure to fluoride	Does not use fluoridated toothpaste; drinking water is not fluoridated; not taking fluoride supplement	Uses fluoridated toothpaste; usually does not drink fluoridated water and does not take fluoride supplement	Uses fluoridated toothpaste; drinks fluoridated water or takes fluoride supplement
Part 2 – Clinical evaluation (determined by examining the child's mouth)			
Visible plaque on anterior teeth	Present		Absent
Gingivitis		Present	Absent

Areas of demineralization (white spot lesions)	More than 1	1	None
Enamel characteristics; hypoplasia, defects, retentive pits/fissures	Present		Absent
Part 3 – Supplemental assessment (Optional)			
Radiographic enamel caries	Present		Absent
Levels of mutans streptococci	High	Moderate	Low

*Based on AAPD Policy on Use of Caries-risk Assessment Tool (CAT) for Infants, Children, and Adolescents. *Pediatr Dent* 2004;26(7) 25

Each child's overall assessed risk for developing decay is based on the highest level of risk indicator circled above (i.e. a single risk indicator in any area of the "high risk" category classifies a child as being "high risk").

VIII. RESPONSIBILITY OF NON-DENTAL PROFESSIONALS REGARDING INFANT ORAL HEALTH CARE

- Since health care professionals (i.e. physicians, nurses) are more likely to serve new mothers and children in their first three years of life compared to dental professionals, it is important they understand their role in providing parent/caregiver oral health education, and be aware of the infectious and transmissible nature of bacteria that cause ECC, associated ECC risk factors, methods of oral health risk assessment (CAT), anticipatory guidance, and appropriate decisions regarding timely and effective intervention, as well as appropriate referral

IX. ADDITIONAL READINGS

- O'Connor TM, Yang SJ and Nicklas TA. Beverage Intake Among Preschool Children and its effect on Weight Status. *Pediatrics* 118:e1010-e1018, 2006.
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Chapter 2: DENTAL DEVELOPMENT, MORPHOLOGY, ERUPTION AND RELATED PATHOLOGIES

AAPD GUIDELINE:

www.aapd.org/media/Policies_Guidelines/RS_DentGrowthandDev.pdf

- I. DENTAL DEVELOPMENTAL STAGES**
- II. DENTAL DEVELOPMENTAL ANOMALIES**
- III. ABNORMALITIES OF COLOR**
- IV. ERUPTION OF TEETH**
- V. ANOMALIES OF ERUPTION**
- VI. TABLES (T)**
- VII. ADDITIONAL READINGS**

I. DENTAL DEVELOPMENTAL STAGES

Embryology

- Neural crest cells
 - develop from ectoderm along the lateral margins of neural plate
 - undergo extensive migration
 - responsible for many skeletal and connective tissues: bone, cartilage, dentin, dermis, not enamel
- Dental lamina
 - begins development at 6 weeks of embryonic age
 - dental lamina differentiates from basal layer of oral epithelium
 - tooth buds arise from dental lamina
 - three phases initiation of primary dentition: 2nd month in utero, initiation of succedaneous dentition; 5 month in utero, to 10 months postnatal initiation of accessional dentition; 4 months in utero for permanent 1st molar, one year of age for permanent second molar, age 4-5 for third molar
- Components of tooth bud
 - enamel organ (from oral epithelium)
 - dental papilla
 - dental sac

Morphologic Developmental Stages

- Dental lamina - characterized by initiation
- Bud stage - initial swellings from dental lamina
 - characterized by proliferation and morphodifferentiation
- Cap stage
 - inner (concavity) and outer (convexity) enamel epithelium
 - stellate reticulum (center of epithelial enamel organ) - supports and protects ameloblasts
 - dental papilla (neural crest origin): formative organ of dentin and primordium of pulp
 - dental sac
 - characterized by proliferation, histodifferentiation, and morphodifferentiation
- Bell stage: invagination of epithelium deepens, margins continue to grow
 - stratum intermedium - essential for enamel production
 - primordia of permanent teeth bud off primary lamina
 - characterized by proliferation, histodifferentiation, and morphodifferentiation
- Advanced bell stage
 - future DEJ outlined
 - basal margin of enamel organ gives rise to Hertwig's epithelial root sheath
- Hertwig's epithelial root sheath
 - composed of inner and outer enamel epithelia without stratum intermedium and stellate reticulum
 - root sheath loses continuity once first layer of dentin laid down
 - remnants persist as rests of Malassez
- Enamel pearls
 - cells of epithelial root sheath may remain attached to dentin
 - may differentiate into ameloblasts and produce enamel

- Formation of enamel and dentin matrices
 - characterized by apposition

Histophysiology

- Initiation
 - dental lamina activity
 - problems lead to anomalies of tooth number
- Proliferation
 - encompasses bud, cap, early bell, late bell
 - problems lead to anomalies of size, proportion, number, twinning
- Histodifferentiation
 - encompasses cap, early bell, late bell
 - differentiation of odontoblasts precedes that of ameloblasts
 - problems lead to anomalies of enamel and dentin
- Morphodifferentiation
 - occurs in bud, cap, early bell, late bell
 - basic form and relative size established by differential growth
 - outline of DEJ established
 - occurs in bud, cap, early bell, late bell
 - problems result in anomalies of size and shape
 - regular and rhythmic deposition of matrix of hard dental structures
 - problems lead to anomalies of enamel, dentin and cementum
- Apposition
 - takes place in two stages
 - (1) immediate partial mineralization as matrix segments are formed
 - (2) maturation - gradual completion both processes occur simultaneously
 - takes place in waves from DEJ outward, from incisal to cervical
 - the term "maturation" is also used to describe post-eruption mineralization
- Calcification (Mineralization) and Maturation
 - problems lead to anomalies of mineralization of enamel and dentin

II. DENTAL DEVELOPMENTAL ANOMALIES

Development Defects of Teeth: <http://www.dent.unc.edu/research/defects/>

Anomalies of Number (Initiation) - Hyperdontia

- Incidence 0.3-3%; males 2:1 females
- Frequency - permanent dentition 5x as common as primary
- Location - 90% in maxilla
- Classification
 - supplemental > normal
 - rudimentary > conical, tuberculate, molariform (differentiate from odontoma)

Anomalies of Number (Initiation)- Hypodontia (Oligodontia)

- Incidence 1.5-10% excluding 3rd molars
- Frequency - third molars, mandibular 2nd premolar, maxillary lateral, maxillary 2nd premolar

- Significant correlation between missing primary and missing permanent successor
- May be inherited

Syndromes with supernumerary teeth

- Apert (acrocephalosyndactyly)
 - narrow, high palate
 - cleft of soft palate - 30%
 - delayed or ectopic eruption
 - shovel shaped incisors
 - hypoplastic midface
- Cleidocranial dysplasia
 - delayed development and eruption of permanent teeth
 - supernumerary teeth
 - delayed primary exfoliation
 - pseudoprognathism (mid-face hypoplasia)
 - enamel hypoplasia
- Gardner syndrome
 - delayed eruption
 - supernumerary teeth
 - osteomas of the jaw
- Crouzon syndrome (craniofacial dysostosis)
 - hypoplastic midface
 - inverted V-shaped palate
- Sturge-Weber syndrome
 - port-wine capillary malformation
 - overgrowth of bony maxilla
- Orofaciodigital syndrome I
 - multiple or hyperplastic frenuli
 - cleft tongue
 - cleft alveolus; hypodontia
- Hallermann-Streiff syndrome
 - mandibular hypoplasia
 - high palatal vault
 - premature teeth
 - delayed primary exfoliation
 - malar hypoplasia
- Others (cleft lip and palate, Down syndrome)

Conditions with hypodontia

- Ectodermal dysplasia
 - conical crowns
 - hypodontia to anodontia
 - deficient alveolar ridge
- Crouzon syndrome (craniofacial dysostosis)
 - maxillary hypoplasia
- Achondroplasia
 - midface hypoplasia
 - frontal bossing

- Chondroectodermal dysplasia (Ellis-van Creveld)
 - premature teeth - 25%
 - absent maxillary sulcus
 - conical crowns
 - partial anodontia
 - enamel hypoplasia
- Incontinentia pigmenti
 - conical crowns
 - delayed eruption
 - premature teeth
 - cleft lip/palate
- Orofaciodigital syndrome I
 - median pseudocleft of upper lip
 - cleft tongue
 - cleft palate
 - multiple hyperplastic frenum with clefts
- Hallerman-Strieff syndrome
 - mandibular hypoplasia
 - high palatal vault
 - premature teeth
 - delayed primary exfoliation
 - malar hypoplasia
- Rieger syndrome
 - midface hypoplasia
 - delayed eruption
 - hypodontia, usually upper incisors
- Seckel syndrome
 - microcephaly
 - facial hypoplasia
- Williams syndrome
 - partial anodontia
 - prominent lips
 - microdontia
 - enamel hypoplasia

Anomalies of Size (Proliferation)

- Microdontia/macrodontia
 - true generalized vs. relative generalized
 - single tooth macrodontia rare; fusion, gemination
- Microdontia
 - frequency: lateral incisors, 2nd premolars, 3rd molars

Conditions with microdontia

- Oligodontia
- Ectodermal dysplasia
- Chondroectodermal dysplasia (Ellis-van Creveld)
- Hemifacial microsomia
- Down syndrome
- Crouzon syndrome

Conditions with macrodontia

- Hemifacial hyperplasia
 - accelerated eruption on affected side submucous cleft
- Crouzon syndrome
- Otodental dysplasia
 - macrodontia affects posterior teeth globodontia (primary second molars) molar fusion

Twining/Conjoined Anomalies (Proliferation)

- Gemination
 - incidence: ~0.5% and more common in primary dentition
 - characteristics: abortive attempt by single tooth to divide bifid crown with single root and pulp chamber
 - familial inheritance
 - significance: crowding may retard eruption of permanent successor
 - clinical diagnosis: extra crown (assuming normal complement of other teeth)
- Twining
 - characteristics: complete cleavage of single bud results in supernumerary mirror image tooth (see hyperdontia)
- Fusion
 - incidence: ~0.5% and more common in primary dentition higher frequency in Japanese (5%)
 - characteristics: dentinal union of two embryologically developing teeth two separate pulp chambers; separate or fused canals many appear as large bifid crown with one chamber; dentin always confluent
 - significance: may retard eruption of permanent successor
 - clinical diagnosis: normal complement of crowns (unless fusion with supernumerary)
- Concrescence
 - characteristics: fusion that occurs after root formation is completed
 - etiology: trauma, crowding may occur pre- or post-eruption

Anomalies of Size and Shape (Morphodifferentiation)

- Dens in dente (dens invaginatus)
 - incidence: 1-7.7%; rare in African-Americans
 - frequency: maxillary lateral most affected; both dentitions
 - characteristics: invagination of inner enamel epithelium
 - significance: carious involvement via communication between oral environment and invaginated portion
- Dens evaginatus
 - incidence: 1-4.3%, higher in some racial groups (Chinese, Japanese)
 - characteristics: evagination of enamel epithelium focal hyperplasia of pulp mesenchyme
 - significance: pulp tissue within extra cusp which may fracture easily
 - syndromes: lobodontia - “wolf teeth,” fang-like cusps

- Taurodontism
 - failure of normal invagination of Hertwig's epithelial root sheath
 - incidence 0.54-5.6%; higher in mongoloid and capoid races
 - elongation of crown at the expense of the roots
 - significance: large pulps

Syndromes with taurodontism

- Klinefelter syndrome
 - small cranial dimension
 - bimaxillary prognathism
 - taurodontism in 30%
- Tricho-dento-osseous syndrome (TDO)
 - dolichocephalic with frontal bossing taurodents have periapical radiopacities and high pulp horns with likely microexposures; delayed eruption
- Mohr syndrome (orofaciocutaneous syndrome II)
 - lobed tongue upper lip midline cleft
 - oligodontia
- Ectodermal dysplasia
- Down syndrome
- Dilaceration
 - etiology: trauma to primary dentition, esp. intrusion
 - syndrome: lamellar congenital ichthyosis

Anomalies of Structure (Histodifferentiation)

- Amelogenesis imperfecta (AI)
 - heritable enamel defect
 - incidence variably reported as 1:14,000, 1:8000, 1:4000
 - multiple inheritance patterns
 - 14 subgroups under 4 major types
 - distinguished from other enamel defects: confinement to distinct patterns of inheritance; occurrence apart from syndromic, metabolic, or systemic condition
- AI Type I - Hypoplastic
 - insufficient quantity of enamel
 - both dentitions affected
 - most subgroup autosomal dominant
 - anterior openbite in 60%
 - subgroups (Witkop)
 - pitted - autosomal dominant (AD)
 - localized - AD
 - localized - autosomal recessive (AR)
 - smooth - AD
 - smooth - X-linked (X)
 - rough - AD
 - enamel agenesis - AR
- Dentinogenesis imperfecta (DI)
 - heritable defect of predentin matrix
 - normal mantle dentin
 - amorphous and atubular circumpulpal dentin
 - incidence 1:8000
 - 3 subtypes (Shields I, II and III)

- DI - Shields Type I
 - occurs with osteogenesis imperfecta (see below)
 - primary teeth more severely affected
 - permanent teeth most often affected are central incisors and 1st molars
 - amber translucence
 - periapical radiolucencies
 - autosomal dominant
 - rapid attrition
- DI - Shields Type II
 - occurs alone - no OI-hereditary opalescent dentin
 - both dentitions equally affected
 - same characteristics as DI-I
 - irregular or tubular pattern
 - rapid attrition
 - autosomal dominant
- DI - Shields Type III
 - rare; Brandywine population
 - bell-shaped crowns
 - shell teeth with short roots and enlarged pulp chambers
 - multiple pulp exposures
 - regular tubules
 - enamel pitting
 - different expression for the same DI-II gene
- Osteogenesis imperfecta
 - major types; OI Type I most common
 - bowing of legs
 - fragile bones - fractures
 - blue sclera
 - bitemporal bossing
 - defective collagen > loose ligaments
 - impaired hearing
 - macrocephaly
 - autosomal dominant

Anomalies of Structure (Apposition) - Enamel

- Amelogenesis imperfecta - hypoplastic/hypomaturation
 - normal thickness
 - low radiodensity, quite soft
 - brown color - porous surface
 - X-linked
 - defective or absent rod sheath
 - defective formation of apatite
 - sheath may be filled with debris
- Amelogenesis imperfecta - hypomaturation/hypoplastic with taurodontism
 - distinct from tricho-dento-osseous syndrome
 - mottled yellow-brown enamel with pits
 - molars are taurodont
 - autosomal dominant

- Acquired enamel hypoplasia - systemic causes:
 - nutrition: vitamins A, C, D, Ca, Phosphate
 - infection: rubella embryopathy, syphilis, cytomegalovirus
 - chromosome defects and syndromes:
 - Down syndrome
 - tuberous sclerosis
 - epidermolysis bullosa
 - Hurler syndrome
 - Hunter syndrome
 - Treacher-Collins syndrome
 - hypoparathyroidism
 - tricho-dento-osseous syndrome
 - Vitamin D-dependent rickets
 - Lesch-Nyhan syndrome
 - Fanconi syndrome
 - Sturge-Weber syndrome
 - Turner syndrome
- Neurologic defects: cerebral palsy
 - allergies/asthma
 - fluorosis
 - radiation
 - low birthweight
- Enamel hypoplasia - local causes
 - infection
 - trauma
 - iatrogenic surgery
 - retained primary teeth

Anomalies of Structure (Apposition) - Dentin

- Dentin dysplasia - 2 types (Shields)
- Shields type I dentin dysplasia - radicular dentin dysplasia
 - normal color of crown of primary and permanent teeth
 - short, blunted roots or rootless in both dentitions
 - obliterated pulp chambers
 - periapical radiolucencies
 - cascading of dentinal tubules in root
 - can be normal tubule orientation in coronal of normal dentin
 - root sheath problem
 - severe mobility and malalignment
 - autosomal dominant
- Shields type II dentin dysplasia - coronal dentin dysplasia
 - primary teeth affected
 - coronal dentin is involved as well as root dentin
 - amber colored primary teeth
 - permanent teeth look normal , but radiographically demonstrate thistle-tube shaped pulps, multiple pulp stones
 - autosomal dominant
- Regional odontodysplasia - “ghost teeth”
 - localized arrest in tooth development
 - atubular tracts, irregular tubules, interglobular calcification, no odontoblastic layer
 - cementum can be normal or aberrant

- thin enamel with diffuse shell appearance
- primary and permanent dentition affected
- 80% involve centrals
- no established etiology or inheritance pattern
- Other conditions with dentin abnormalities
 - Vitamin D-resistant rickets
 - x-linked dominant; autosomal recessive
 - failure of distal tubular reabsorption of phosphate in the kidneys
 - hypophosphatemic rickets
 - hypomineralized dentin
 - increased width to predentin
 - odontoblastic disorganization
 - enlarged pulp and pulp horns
 - enamel may be spared
- Hypoparathyroidism
 - permanent teeth affected predominantly short, wedge-shaped roots with delayed apical closure
 - interglobular calcification in dentin, especially at apices
 - enamel hypoplasia
- Pseudohypoparathyroidism
 - enlarged pulp chambers
 - irregular dentinal tubules
 - small crowns and short blunted roots
 - pitted enamel surfaces
- Albright's hereditary osteodystrophy
 - inadequate hydrogen ion clearance
 - hypocalcemia and hyperphosphatemia
 - ectopic calcifications
 - short stature, brachydactyly, blunted roots, small crowns
 - mental deficiency
 - X-linked dominant
 - irregular dentinal tubules
 - intrapulpal calcifications
- Ehlers-Danlos syndrome
 - hyperelastic, fragile skin and mucosa
 - skin hemorrhages and scars
 - joint hypermobility
 - X-linked
 - irregular dentin tubules with inclusions
 - intrapulpal calcifications

Anomalies of Structure (Apposition) - Cementum

- Hypophosphatasia
 - lack of serum alkaline phosphatase
 - urinary phosphoethanolamine
 - autosomal recessive
 - little cementum produced
 - early exfoliation of primary dentition

- Epidermolysis bullosa
 - fibrous acellular cementum
 - excess cellular cementum
- Cleidocranial dysplasia
 - deficient cellular cementum

Anomalies of Structure (Calcification) - Enamel

- Enamel hypocalcification
 - See causes for enamel hypoplasia
- Amelogenesis imperfecta type III - hypocalcified
 - deficit in calcification of matrix
 - normal thickness, soft enamel
 - anterior openbite in 60%
 - high calculus formation
 - delays in eruption
 - 2 subgroups
 - autosomal dominant
 - autosomal recessive
- Enamel fluorosis
 - greater than 2 ppm in water - 10% chance of fluorosis
 - greater than 6 ppm in water - 90% chance
 - Dean's index: normal, questionable, very mild, mild, moderate, severe
 - Tooth Surface Index of Fluorosis (TSIF) - Horowitz et al. JADA 1984;109:37
84.5% unaffected in optimally fluoridated areas
 - 78.1% had some degree of fluorosis when fluoride was 4x optimal
- Sclerotic dentin
 - deposition of Ca salts in tubules

III. ABNORMALITIES OF COLOR

Intrinsic Stains

- Blood-borne pigments
 - porphyria - porphyrin: purplish-brown
 - bile duct defects: green
 - neonatal hepatitis - bilirubin: black, gray
 - Rh incompatibility (erythroblastosis, fetalis) - bilirubin, biliverdin, blue-green, brown
 - anemias - hemosiderin: gray
 - dental trauma: red, gray, black
- Drug administration
 - tetracyclines
 - both dentitions affected
 - related to dose and duration
 - 21-26 mg/kg/day is threshold
 - primary teeth are more intense
 - tetracycline HCl most stain
 - oxytetracycline least stain
 - teeth darken with more exposure to UV light
- Cystic fibrosis
 - may be related to disease, tetracycline, or combination
 - color yellowish gray to dark brown

- Trauma
- Hypoplasia/hypocalcification disorders
 - amelogenesis imperfecta
 - dentinogenesis imperfecta
 - dental caries
 - enamel and dentin dysplasias
- Systemic fluoride

Extrinsic Stains

- Bacteria
 - green: *Bacillus pyocaneus*, *Aspergillus* most common
 - orange: chromogenic bacteria, poor OH, more easily removed than green
 - brown/black: much less common, difficult to remove, chromogenic bacteria
- Discoloring agents
 - foods
 - tobacco
 - restorative materials
 - medicaments
 - silver nitrate
 - iron sulfide
 - stannous fluoride
 - chlorhexidine

IV. ERUPTION OF TEETH

Theories of Eruption

- Root growth
- Vascular pressure
- Bone growth
- Periodontal ligament traction
- Connective tissue proliferation at the pulp apex

Eruption Sequences

- Most favorable eruption sequence in primary dentition ABDCE
- Most favorable eruption sequence in permanent dentition
 - maxilla: 61245378
 - mandible: 61234578
- Sequence more important than timing

Stages of Eruption

- Follicular growth
- Pre-emergent eruptive spurt
- Post-emergent eruptive spurt
- Juvenile occlusal
- Circumpubertal eruptive spurt
- Adult occlusal equilibrium

Variables That Influence Permanent Tooth Eruption

- Genetic - estimated at 78%
 - familial: high correlation based on twin studies
 - race: blacks slightly earlier than whites
 - sex: females ahead of males

- Environmental
 - low birth weight and prematurity: delayed eruption
 - nutrition: little or no effect
- Systemic
 - endocrine
 - high correlation with hypopituitarism and hypothyroidism
 - low correlation with altered growth
 - hormone production
- Clinical guides for use in assessing eruption stage/rate of permanent dentition
 - root development
 - overlying bone
 - infection
 - timing of primary tooth loss
 - before age 5 - delays premolar
 - after age 8 - accelerates premolar

V. ANOMALIES OF ERUPTION

Timing

- Premature teeth
 - erupt prior to 3 months of age
 - natal - present at birth
 - neonatal - present within first 30 days of life
 - natal 3:1 neonatal
 - incidence 1:2000-3500
 - 90% are true primary teeth
 - etiology unknown; superficially positioned bud?
 - most are poorly formed
 - associated finding: Riga-Fede disease
 - sublingual traumatic ulceration due to natal or neonatal teeth
 - syndromes: chondroectodermal dysplasia (Ellis-van Creveld)
 - 25% pachyonychia congenita
- Structures in the newborn often confused with premature teeth
 - Bohn nodules
 - buccal, lingual aspects of the maxillary alveolar ridge (away from midline raphe)
 - mucous gland tissue
 - Dental lamina cysts
 - found on the crest of the alveolar ridge
 - derived from remnants of the dental lamina
 - Epstein pearls
 - midpalatal raphe
 - trapped epithelial remnants
 - visible cysts in 80% of newborns

Teething

- Over half of babies have one or more problems during teething
 - otitis media
 - paroxysmal atrial tachycardia
 - gastroesophageal reflux

- Teething differential - R/O
 - febrile convulsions
 - URI
 - bronchitis
 - eczema
 - H. flu meningitis
 - fever $> 101^{\circ}\text{F}$ not attributed to teething - look for other causes

Cystic Development

- Eruption hematoma
 - dilation of follicular space
 - blood or tissue fluid
 - form of eruption cyst
- Primordial cyst: stellate reticulum
- Dentigerous cyst: reduced enamel epithelium
- Ameloblastoma
 - dentigerous cyst and odontogenic cyst
 - epithelial rests of Malassez
 - disturbed enamel organ

Delayed Primary Exfoliation and Permanent Eruption

- Local causes
 - trauma
 - impaction
 - ankylosis
 - supernumeraries
- Systemic conditions
 - cleidocranial dysplasia
 - chondroectodermal dysplasia (Ellis-van Creveld)
 - achondroplasia
 - osteogenesis imperfecta
 - Gardner syndrome
 - Down syndrome
 - deLange syndrome
 - Apert syndrome
 - hypothyroidism
 - hypopituitarism
 - ichthyosis (also associated with ankylosis)
 - Albright's hereditary osteodystrophy
 - Hunter syndrome
 - incontinentia pigmenti
 - fibromatosis gingivae
 - low birth weight

Accelerated Eruption of Primary and Permanent Teeth

- Local causes
 - early loss of primary tooth (closer to normal time of permanent tooth eruption)
- Systemic conditions
 - hemifacial hypertrophy
 - precocious puberty
 - hyperthyroidism
 - Sturge-Weber syndrome
 - chondroectodermal dysplasia (Ellis-van Creveld)

- osteogenesis imperfecta
- pachyonychia congenita
- Soto syndrome (cerebral gigantism)

Premature Exfoliation of Primary Teeth

- Diseases of bone
 - fibrous dysplasia
- Diseases of periodontium
 - prepubertal periodontitis
 - Papillon-LeFevre
- Diseases of metabolism
 - hypophosphatasia
- Deviations in growth and development
 - hemihypertrophy
 - premature teeth
- Diseases of blood
 - leukemia
 - Chediak-Higashi
 - cyclic neutropenia
- Physical and chemical injuries
 - acrodynia
 - facial burns
- Benign and malignant tumors
 - histiocytosis/Langerhans cell group (non-lipid reticuloendothelioses)
 - Letterer-Siwe (quickly fatal)
 - Hand-Schuller-Christian (better prognosis)
 - eosinophilic granuloma (excellent prognosis)
- Dental anomalies
 - dentin dysplasia
 - odontodysplasia

Ectopic Eruption (Permanent Molars)

- Incidence of permanent first molars: 2-3% (25% in CLP)
- Etiology for permanent maxillary first molars
 - larger mean sizes of all maxillary permanent and primary teeth
 - larger affected Es and 6s
 - smaller maxilla
 - posterior position of maxilla related to cranial base (smaller SNA)
 - abnormal angulation of erupting 6
 - delayed calcification of some affected 6s
- Self correction
 - 66% (only 22% in CLP)

Ankylosis (Infraocclusion)

- Anatomical fusion of cementum with alveolar bone occurring at any time during course of eruption (histological diagnosis)
- May occur prior to emergence or occlusal contact
- Clinically diagnosed as “submerged” tooth - area of ankylosis often not detected by x-ray; dull noise to percussion (controversial)
- Etiology: unknown

- Possible extrinsic factors
 - trauma
 - disturbed local metabolism
 - localized infection
 - tooth replantation
- Possible intrinsic factors
 - gap in PDL
 - aberrant deposition of cementum or bone
- Prevalence—1.3%-38.5% (depending on diagnostic criteria, sample characteristics)
 - Primary mandibular second molar most often affected
 - Associated with agenesis of succedaneous teeth
 - Multiple teeth seen as frequently as single
- Sequelae
 - deflected eruption paths
 - impacted premolars
 - loss of arch length and alveolar bone
 - supraeruption of opposing teeth (esp. maxilla)
- Treatment - empirical
 - observe (esp. for primary first molars, esp. for mandibular teeth)
 - extract
 - restore to occlusion
 - luxate (permanent teeth)
- Timing
 - primary mandibular first molars when permanent first molars erupt often exfoliate on schedule
 - do not infraocclude dramatically can be restored to occlusion
 - primary mandibular second molars later onset than lower Ds likely to be bilateral usually more severe infraocclusion than lower Ds
 - primary maxillary first and second molars relatively rapid progression occurs close to or ahead of eruption of 6s usually must extract

Maxillary Central Diastema

- Prevalence
 - 44-97% in 6-year-olds (ugly duckling stage)
 - 33-46% in 9-year-olds
 - 7-20% in 14-year-olds
- Racial distribution
 - higher in African-Americans, Mediterranean whites
 - higher in females at younger ages (?)
- Etiologies
 - normal
 - development of mixed dentition
 - familial/racial - associated with bimaxillary protrusion
 - excessive skeletal growth: acromegaly
 - pernicious habit: lip biting, digit sucking
 - deficiency of tooth material in arch due to:
 - spaced dentition
 - missing/peg laterals
 - extractions
 - excessive overjet
 - excessive overbite
 - ectopic laterals/crowded to lingual

- physical impediment to normal closure
mesiodens
retained primary teeth
midline pathology
enlarged labial frenum (may be effect rather than cause)
interruption of transseptal fibers
- artificial
rapid palatal expansion
Milwaukee brace
- Treatment - usually done after eruption of permanent canines
 - based on diagnosis of cause - Bolton analysis helpful
 - eliminate habit if present
 - mesial tipping of central incisors
 - bodily movement of central incisors
 - reduction of excess overjet
 - surgical intervention - transseptal fibers/frenum
 - enlargement of incisors

VI. TABLES

Developmental Stages and Associated Anomalies

Developmental Stage	Deficient Development	Excessive Development
Initiation	• Anodontia	• Supernumerary teeth
Proliferation	<ul style="list-style-type: none"> • Hypodontia • Congenital Absence • Fusion 	<ul style="list-style-type: none"> • Natal teeth • Epithelial Rests • Gemination
Histodifferentiation	<ul style="list-style-type: none"> • Amelogenesis Imperfecta -Hypoplastic type 	• Dentinogenesis Imperfecta
Morphodifferentiation	<ul style="list-style-type: none"> • Peg lateral • Mulberry molars • Hutchinson incisors • Microdontia 	<ul style="list-style-type: none"> • Tuberculated cusps • Carabelli Cusp • Macrodontia • Taurodontia • Dens in dente
Apposition	<ul style="list-style-type: none"> • Enamel Hypoplasia <ul style="list-style-type: none"> - systemic - local - congenital • Dentinal dysplasia 	<ul style="list-style-type: none"> • Enamel Pearls • Hypercementosis
Calcification	<ul style="list-style-type: none"> • Amelogenesis Imperfecta -Hypocalcified type • Fluorosis • Interglobular Dentin 	<ul style="list-style-type: none"> • Odontoma • Sclerotic Dentin
Eruption	<ul style="list-style-type: none"> • Ankylosis, Impaction • Transposition • Delayed Eruption 	<ul style="list-style-type: none"> • Neonatal Teeth • Precocious Eruption

Chronology of the Human Dentition

AAPD Guideline: www.aapd.org/media/Policies_Guidelines/RS_DentGrowthandDev.pdf

Tooth Eruption Charts: http://www.ada.org/public/topics/tooth_eruption.asp

	Initiation (Intra-uterine) weeks	Calcification (weeks)	Primary Dentition	
			Maxillary	Mandibular
Central incisor A	6	14	7½ mo	6 mo
Lateral incisor B	6	16	9 mo	7 mo
Canine C	7	17	18 mo	16 mo
First molars D	6	15	14 mo	12 mo
Second molar E	8	19	24 mo	20 mo

Sequence of calcification of primary teeth

- a. Calcification: A D B C E
- b. Cusps of posterior teeth: MB, ML, DB, DL
- c. One calcification center for anterior teeth

Permanent Dentition

	Initiation	Calcification	Eruption		
			Maxillary	Mandibular	Maxillary
Central incisor	5 mo (IU) ¹	3-4 mo	3-4 mo	7-8 yr	6-7 yr
Lateral incisor	5 mo (IU)	10-12 mo	3-4 mo	8-9 yr	7-8 yr
Canine	5 mo (IU)	4-5 mo	4-5 mo	11-12 yr	9-10 yr
First premolar	5 mo (IU)	1.5-1.7 yr	1.7-2 yr	10-11 yr	10-12 yr
Second premolar	10 mo (PP) ²	2-2.2 yr	2.2-2.5 yr	10-12 yr	11-12 yr
First molar	20 wks (IU)	Birth	Birth	6-7 yr	6-7 yr
Second molar	12 mo (PP)	2.5-3 yr	2.5-3 yr	11-13 yr	11-13 yr
Third molar	5 yr (PP)	8 yr	9 yr		

¹Intra uterine

²Post partum

- Length of time for root completion of primary and permanent teeth
 - a. Primary teeth - 18 months post eruption
 - b. Permanent teeth - 3 years post eruption
- Time interval between crown completion and eruption to full occlusion in permanent teeth - 5 years (Shumaker and El Hadary, JADA 61:535, 1960)

VII. ADDITIONAL READINGS AND WEB SITES

1. Jones KL, Smith's Recognizable Patterns of Human Malformation. Elsevier Saunders. Philadelphia, 2006.
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Chapter 3: ORAL PATHOLOGY/ORAL MEDICINE/SYNDROMES

- I. INFANT SOFT TISSUE LESIONS
- II. WHITE LESIONS—DIFFERENTIAL DX
- III. LOCALIZED GINGIVAL LESION
- IV. GENERALIZED GINGIVAL ENLARGEMENT
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- XXV. DWARFISM**
- XXVI. SELF-MUTILATION**
- XXVII. ADDITIONAL READINGS AND WEB SITES**

I. INFANT SOFT TISSUE LESIONS— DIFFERENTIAL DX

Common

- Epstein Pearls/Bohn Nodules/Dental Lamina Cyst
 - Epstein Pearls—palatal midline, epithelial inclusion cyst
 - Bohn Nodules—buccal and lingual surface of alveolus, ectopic mucous glands
 - dental lamina cyst—crest of alveolus; remnants of dental lamina

Uncommon

- Vascular malformations/tumors (see macroglossia)
 - hemangioma—may involve major salivary glands, usually parotid
 - diffuse enlargement of gland
 - normal or reddish-blue skin coloration
 - regresses with age
 - lymphangioma—cystic hygroma poorly circumscribed swelling of cervical region of neck
 - tx: may include surgery

Rare

- Congenital epulis of newborn
 - firm pedunculated mass arising from alveolus at birth
 - maxillary lateral and canine region most common site
 - females > males
 - maxilla > mandibular
 - tx: excision
- Neuroectodermal tumor of infancy
 - smooth surfaced expansile lesion of alveolus
 - premaxilla most common site
 - may be pigmented
 - usually occurs in infants under 6 months
 - maxilla > mandible
 - displacement of teeth
 - X-ray: poorly circumscribed radiolucency with floating teeth
 - tx: excision
- Hemifacial hypertrophy
 - unilateral oral and facial enlargement, usually evident at birth
 - involves soft tissues, bone, tongue, palate, teeth
 - teeth may exfoliate and erupt prematurely
 - 25% MR
 - increased incidence of embryonal tumors
(Wilms tumor, hepatoblastoma)
 - tx: cosmetic surgery
- Hemifacial microsomia (Goldenhar syndrome)
 - unilateral microtia, macrostomia and failure of formation of mandibular ramus and condyle
 - unknown etiology
 - frequent eye and skeletal involvement
 - 50% have cardiac pathology—VSD, PDA
 - tx: ortho, functional appliances, cosmetic surgery

II. WHITE LESIONS—DIFFERENTIAL DX

Common

- Candidiasis—common oral organism, but usually does not cause infection unless host immunocompromised
 - newborn may acquire infection from mother with untreated vulvovaginitis
 - increased susceptibility with long-term antibiotics, cortico-steroids, debilitating disease, oral lesions
 - oral lesions raised white plaques
 - tx: nystatin, ketoconazole, amphotericin B
- Leukoedema
 - most commonly seen in blacks
 - usually bilateral
 - stretching of mucosa causes lesion to disappear
 - increase thickness of mucosa, intracellular edema of spinous layer
- Trauma
- Geographic tongue—multiple areas of desquamation of filiform papillae
 - lesions similar to psoriasis
 - Monro's abscess

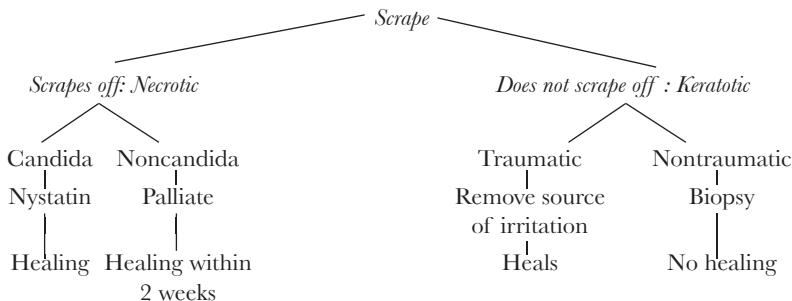
Uncommon

- White sponge nevus
 - autosomal dominant
 - diffuse, white, thickened, folded appearance of oral mucosa (may involve tongue)
 - present at birth, may involve other mucosa
- Allergy
 - combination of contactant with epithelial proteins forms antigen
 - burning sensation
 - vesicle formation with subsequent rupture
- Leukoplakia—white patch
 - chewing tobacco
 - lip biting
 - 10-15% demonstrate dysplasia or malignancy

Rare

- Heck's disease—Focal epithelial hyperplasia
 - Indians and Eskimos most commonly affected
 - multiple nodular lesions usually with sessile base
 - lower lip, tongue, buccal mucosa common sites
 - occurs in children between 3–18 years
 - possible viral etiology
- Hereditary Benign Intraepithelial Dyskeratosis (HBID)
 - affects individuals of mixed Caucasian, Indian, and Black ancestry living in North Carolina
 - appears similar to white sponge nevus

White Lesion



III. LOCALIZED GINGIVAL LESION

—DIFFERENTIAL DX

Common

- Abscess
- Papilloma
- Fibroma

Uncommon

- Pyogenic granuloma
 - sessile, erythematous nodule
 - surface ulceration
 - soft to palpation
 - tx: surgical excision, removal of irritant
- Peripheral giant cell tumor
 - well circumscribed, sessile nodules
 - “liver” coloration
 - often ulcerated and hemorrhagic
 - tx: surgical excision
- Peripheral ossifying fibroma
 - well circumscribed, sessile or pedunculated lesion
 - usually non-ulcerated and pink, firm
 - X-ray may show calcification
 - underlying bone uninvolved
 - tx: surgical excision

Rare

- Langerhans Histiocytosis X
- Osteogenic sarcoma
- Ewing sarcoma
- Hemangioma

IV. GENERALIZED GINGIVAL ENLARGEMENT—DIFFERENTIAL DX

Common

- Gingivitis
- Mouth-breathing
- Drug Induced
 - Dilantin
 - Cyclosporin
 - Calcium Blockers

Uncommon

- HIV gingivitis
 - fiery red edema of attached gingiva beyond mucogingival junction
- Allergy
 - flavoring agents
 - tartar control toothpastes

Rare

- Familial gingivofibromatosis
 - diffuse, multinodular overgrowth of fibrous tissue of gingiva
 - AD; syndrome with hirsutism also has been identified
 - clinically identical to dilantin hyperplasia, but involves loose alveolar tissue
 - tx: surgical excision
- Leukemia
 - Gingivitis secondary to neutropenia
 - Infiltrate in AML, ANLL

V. PIGMENTATION—DIFFERENTIAL DX

Common

- Localized pigmented lesions
 - amalgam/graphite tattoo
 - hematoma
 - hemangioma
 - nevus - intradermal, junctional, compound, juvenile
 - ephelis - freckle

Uncommon

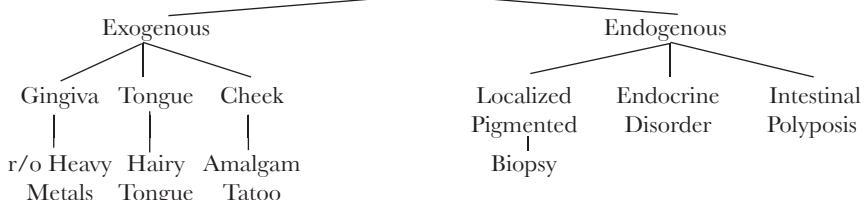
- Endocrine disease
 - Addison's disease—adrenal insufficiency
 - weakness, nausea, vomiting, low PP, pigmentation
 - oral: blotches resembling bluish-purple ink
- Peutz-Jeghers syndrome—AD
 - melanin hyperpigmentation of lips
 - benign polyposis of small intestine
 - (3% become malignant)
 - buccal lesions less likely to fade than lip lesions
- Carotenemia
 - excessive ingestion of carotene containing foods

- Liver Disease/Hemolytic Anemias
 - jaundice due to excess bilirubin

Rare

- Malignant melanoma
- Neuroectodermal
- Medications
 - antimalarial drugs, produce grey coloration of mucosa
 - patient must take drug at least 4 m to develop pigmentation
 - pigmentation of hard palate sharply delineated from soft palate
- Heavy metal ingestion
 - Bismuth
 - gingivostomatitis similar to ANUG
 - blue-black pigmentation of interdental papillae
 - Lead
 - rare in children
 - salivary gland swelling and dysphagia
 - grey pigmentation of marginal gingiva
 - Mercury
 - ropy, viscous saliva
 - faint grey alveolar gingival pigmentation
 - perio similar to ANUG
 - Silver (Argyria)
 - skin slate grey
 - diffuse pigmentation
 - Copper
 - blue-green gingiva and teeth
 - Zinc
 - blue-grey line on gingiva
 - periodontal involvement
 - no pain
- Hemochromatosis—iron storage disease
 - First degree
 - associated with increased absorption of iron form GI tract
 - hepatic cirrhosis
 - bronzing of skin (generalized)
 - diabetes mellitus
 - Second degree
 - excessive iron intake
 - blue-gray pigmentation of hard palate
- Neurofibromatosis

PIGMENTED LESIONS



VI. HEMORRHAGE AND/OR HEMORRHAGIC LESIONS—DIFFERENTIAL DX

Common

- Acute gingivitis
- Trauma

Uncommon

- Acquired coagulation disturbance
- Thrombocytopenia
- Blood dyscrasia
 - Factor VIII
 - Factor IX
 - von Willebrand disease
- Vitamin K deficiency
- Liver disease
 - diminished absorption of fat soluble Vitamins A, D, E, K
(Vitamin K needed for production of prothrombin, Factors VII, IX, X)
 - liver produces all coagulation factors except VIII and possibly XIII
- Vascular malformations

Rare

- Hereditary hemorrhagic telangiectasia
 - autosomal dominant
 - multiple capillary and venous dilations of skin and mucous membranes
 - lesions blanch with pressure
 - bleeding from mouth secondary only to epistaxis
 - all dental manipulation must be atraumatic as possible
- Sturge-Weber syndrome
 - venous angiomas of leptomeninges
 - ipsilateral facial angiomas
 - ipsilateral gyriform calcifications of cerebral cortex
 - mental retardation
 - seizures
 - hemiplegia
 - ocular defects
- A-V fistula
 - may be acquired or congenital
 - acquired usually secondary to penetrating wound
 - congenital are extremely variable
 - may manifest:
 - swelling
 - cutaneous hemangioma
 - varicose veins in atypical locations
 - increase in temperature of part
 - thrills and murmurs

VII. LIP SWELLING/MASS—DIFFERENTIAL DX

Common

- Mucocelle
 - arise due to severance of duct or possible partial obstruction
 - most frequent on lower lip, but may occur on palate or tongue, floor of mouth
 - tx: surgical excision
- Trauma

Uncommon

- Nasolabial cyst
 - results from entrapment of epithelium along junction of maxillary, lateral nasal, and globular process
 - females > males
 - tx: surgical excision/curettage

Rare

- Neoplasm—Neuroma, Hemangioma
 - Lymphangioma
 - 95% arise before 10 years
 - superficial or deep
 - tx: excision
 - Traumatic neuroma—nodule
 - Hemangioma
 - blood vessel proliferation (hamartoma)
 - deep red or blue coloration
- Sipple syndrome
 - Multiple endocrine neoplasia, Type IIB
 - mucosal neuromas
 - medullary carcinoma of the thyroid
 - pheochromocytoma

VIII. MACROGLOSSIA—DIFFERENTIAL DX

Common

- Trauma
- Foreign body
- Down syndrome
 - relative macroglossia (50%)
 - microdontia (55%)
 - oligodontia (50%)
 - Class III
 - open-mouth posture
 - fissured tongue
 - decreased caries
 - increased periodontal disease
 - delayed eruption and over-retained teeth
 - tooth morphological abnormality
 - abnormal palate shape (70%)
 - enamel hypocalcification (32%)
- Congenital

Uncommon

- Vascular malformations
 - present at birth
 - become clinically evident in late infancy or childhood
 - may increase in size following trauma, infection, or endocrine changes
 - 35% associated with skeletal changes
- Lymphangioma
 - diffuse vs. cystic
 - tongue most common site
 - surface often papillary and vesicular
 - usually occur early in life
 - Tx: surgical excision
- Capillary malformations
 - port-wine stain
 - present at birth
 - does not regress
- Sturge-Weber syndrome
 - venous angiomas of the leptomeninges
 - ipsilateral facial angiomas
 - ipsilateral gyriform calcifications of cerebral cortex
 - mental retardation
 - seizures
 - hemiplegia
 - ocular defects
 - telangiectasias
- Venous
 - “cavernous hemangioma”
 - does not involute
 - involves skin, subcutaneous tissue, possibly bone
- Arterial
 - ectasias
 - aneurysms
 - stenoses
- Combined
 - arteriovenous fistula
 - arteriovenous malformation
- Hemangioma
 - vascular tumor of infancy
 - flat or raised blue-red lesion
 - usually develop first year of life
 - blanch on pressure
 - usually involute by adolescence
- Neoplasm
 - granular cell myoblastoma—tongue most common site
 - smooth or slightly papillary surface
 - probably derived from nerve tissue
 - tx: surgical excision

Rare

- Cretinism
 - congenital hypothyroidism (myxedema in adults)
 - MR, retarded somatic growth, generalized edema
 - shortening of cranial base—retraction of nose with flaring
 - mandible underdeveloped, maxilla overdeveloped
 - tongue enlargement secondary to edema delayed eruption, exfoliation
 - progressive infiltration of skin and mucous membranes by mucopolysaccharides and mucoproteins
- Neurofibromatosis
 - café au lait spots
 - autosomal dominant (AD)
 - neurofibromas
- Various storage diseases—Mucopolysaccharidosis
 - Hurler syndrome* (prototype) autosomal recessive (AR)
 - coarse facies, large head
 - premature closure of sagittal and metopic sutures frequently causes scaphocephaly
 - nasal bridge depressed
 - enlarged lips
 - open-mouth and protruding tongue after 5 years
 - widely spaced teeth
 - localized areas of bone destruction
- Amyloidosis
 - mixture of protein, glycoprotein, polysaccharides, and lipids
 - may be deposited in any body site
 - primary—genetic or hereditary form
 - secondary—sequel of prolonged inflammatory or infectious disease (i.e., rheumatoid arthritis, ankylosing spondylitis)
- Beckwith-Wiedemann syndrome
 - macroglossia
 - omphalocele or umbilical hernia
 - cytomegaly of adrenal cortex
 - hyperplasia of gonadal interstitial cells
 - renal medullary dysplasia
 - hyperplastic visceromegaly
 - postnatal somatic gigantism
 - mild microcephaly
 - severe hypoglycemia

IX. SUBLINGUAL SWELLING/MASS—DIFFERENTIAL DX

Common

- Ranula
 - mucous retention
 - dome shaped, soft swelling of normal or blue color
 - involves submaxillary or sublingual gland
 - tx: excision or marsupialization

- Trauma

Uncommon

- Abscess
- Hemorrhage
- Salivary gland infection

Rare

- Dermoid cyst
- Salivary gland tumor
- Abscess/Ludwig's angina
- Lymphoepithelial cyst

X. SOFT TISSUE NECK MASS—DIFFERENTIAL DX

Common

- Viral upper respiratory infection

Uncommon

- Bacterial infection
- Cat-Scratch Fever
- Hodgkin's disease
- Leukemia
- Infectious mononucleosis
- Thyroglossal duct cyst
 - remnant of thyroglossal duct
 - occurs midline anywhere along path of thyroglossal duct
 - usually below hyoid

Rare

- Mumps
 - usually involves parotid
 - paramyxovirus (cytomegalic virus or staph in immunocompromised patient)
 - incubation 2–3 weeks
 - pain, fever, malaise, headache, vomiting may precede swelling
 - xerostomia
 - tx: symptomatic
- Kawasaki disease
 - Mucocutaneous Lymph Node Syndrome
 - bilateral conjunctivitis
 - fissured lips
 - infected pharynx
 - strawberry tongue
 - erythema of palms and soles
 - rash
 - cervical adenopathy
- Salivary gland tumor
 - pleomorphic adenoma most common benign lesion
 - parotid most common site

- mucoepidermoid carcinoma most common malignant lesion
- papillary Cystadenoma Lymphomatosum
- hemangioma
- Branchial cleft cyst
 - area of anterior border of sternocleidomastoid muscle
 - soft, movable, poorly delineated mass
 - Theories:
 - (1) origin from remnant of branchial clefts;
 - (2) remnant of salivary gland
- Cystic hygroma
 - lymphangioma
 - may be present at birth
 - slowly enlarges, may cause respiratory distress

XI. PALATAL SWELLING—DIFFERENTIAL DX

Common

- Abscess
 - usually anaerobic bacteria
 - average infection involves 6 anaerobes and 1 aerobe

Uncommon

- Salivary gland tumor
 - pleomorphic adenoma
 - mucoepidermoid carcinoma
 - adenocystic carcinoma
 - adenocarcinoma

Rare

- Nerve tumor
 - benign tumors
 - slow growing, well circumscribed
 - smooth surfaced nodule
 - most commonly found on tongue, palate
 - tx: surgical excision
- Kaposi sarcoma

XII. PALATAL RADIOLUCENCY—DIFFERENTIAL DX

Common

- Median palatal cyst—arises from epithelium entrapped in fusion of palatal processes
 - Midline radiolucency opposite premolars may cause fluctuant swelling on palate
 - tx: surgical excision/curettage
- Nasopalatine duct cyst—arises from remnants of nasopalatine duct
 - located in midline between roots of maxillary incisors
 - may cause root divergence
 - may cause fluctuant swelling
 - tx: surgical excision/curettage

Uncommon

- Trauma

Rare

- Midline lethal granuloma
- Wegener granulomatosis
- Infectious disease
- Neoplasm

XIII. MAXILLARY AND/OR MANDIBULAR ENLARGEMENT—DIFFERENTIAL DX**Common**

- Sickle cell anemia
 - autosomal recessive 1/400 blacks in US
 - defective hemoglobin S (substitution valine for glutamic acid on beta chain)
 - sickling occurs under low O₂
 - rapid destruction of sickled RBC's
 - stagnation—thrombus and/or infarct
 - collection of affected RBC's in vital organs
 - X-ray: stepladder trabeculation, hair on end
 - painful crises; may have had splenectomy; may need ABs for dental treatment

Uncommon

- Albright syndrome
 - Polyostotic fibrous dysplasia
 - Abnormal skin pigmentation “coast of Maine”
 - Endocrine dysfunction—precocious deformity
 - X-ray—“Ground Glass” appearance of lesions
- Fibrous dysplasia—monostotic form
 - benign fibro-osseous lesion of jaw
 - begins early in life with gradual painless enlargement, then stabilizes in adulthood
 - may obliterate mucobuccal fold
 - x-ray: ground glass appearance
- Cherubism
 - autosomal dominant
 - bilateral fullness of cheeks
 - hypertelorism
 - irregularly spaced dentition
 - lesions similar to central giant cell tumor
 - multilocular radiolucencies
- Neoplasm
 - osteosarcoma
 - rhabdosarcoma
 - osteoma

Rare

- Gigantism
 - excess growth hormone
 - underlying lesion usually eosinophilic or mixed cell adenoma of the anterior lobe of pituitary
 - may be seen radiographically
- Hemihypertrophy
 - nonspecific, may occur in a variety of disorders
 - may involve single digit, limb, face, or half, or body
 - usually evident at birth
 - right > left
 - males > females
 - Embryonic tumors may be associated with this disorder
- Thalassemia
 - defect in rate of hemoglobin synthesis
 - persistent fetal hemoglobin
 - most commonly involves beta chain
 - severe hypochromic, microcytic anemia
 - homozygous: major
 - heterozygous: minor
 - hair on end radiographic appearance
 - tx: transfusions

XIV. INTRA-ORAL ULCERS/STOMATITIS— DIFFERENTIAL DX

Common

- Herpes gingivostomatitis—Herpes Simplex Type I
 - fever, lymphadenopathy, headache, malaise, intense gingival erythema, and oral vesicles throughout mouth
 - vesicles rupture leaving painful ulcers
 - fissuring and erupting of lips common
 - cytology: multinucleated giant cells, inclusion bodies, ballooning degeneration
 - tx: palliative and supportive
- Herpangina—Coxsackie A
 - multiple small vesicular lesions involving tonsillar pillars, uvula and soft palate
 - vesicles rupture leaving ulcers with erythematous borders
 - malaise, fever
 - most common in young children during summer months
- Varicella zoster
 - Chickenpox—crops of pruritic vesicles on skin and mucous membrane
 - vesicles may precede fever
 - begins on trunk—spreads to limbs and face
 - infectious 24 hours before to 6–7 days after vesicles appear
 - resolves in 7–10 days
 - tx: palliative and supportive
- Aphthous ulcers
 - central necrosis and ulceration with erythematous halo
 - involves “unbound” mucosa

- tx: steroids, antibiotic rinses, topical anesthetics
- Chemotherapy
- ANUG
 - fusospirochetes
 - necrosis, ulceration, punched out papillae
 - predisposing factors: vitamin deficiencies, compromised immune function, stress, poor oral hygiene
 - rare in young children
- Trauma
- Impetigo
 - most commonly caused by *staphylococcus aureus*, or in combination with Group A B-hemolytic streptococcus
 - localized disease treated with topical antibiotics
 - widespread disease treated with systemic antibiotics

Uncommon

- Hand, foot, and mouth disease
 - Coxsackie A16
 - epidemic
 - fever, malaise, lymphadenopathy
 - vesicles and ulcerations intraorally and on hands, arms, feet, and legs
 - tx: palliative and supportive resolves in 7–10 days
- Erythema multiforme
 - erythematous macules, papules, bullae, and erosions involving skin and mucous membranes
 - possible allergic etiology (drug reaction)
 - target lesions
 - may have ocular and genital involvement (Stevens-Johnson syndrome)
 - tx: steroids

Rare

- Behcet syndrome
 - oral aphthae
 - genital ulceration
 - ocular lesions
 - tx: steroids
- Epidermolysis bullosa—hereditary vesiculobullous disease of skin and mucous membranes
 - EB simplex
 - most common form: AD, cutaneous involvement ranges from occasional blisters of hands and feet to life threatening blistering, onset at birth
- Junctional EB (several subtypes)
 - autosomal recessive
 - ranges from relatively mild blistering to life-threatening
 - may have generalized enamel hypoplasia
- Dystrophic EB

Dominant Form

- generalized blistering with onset in infancy
- dystrophic or absent nails

- may have oral involvement
- affected individuals have normal life span

Recessive Form

- may have excessive blistering leading to scar formation (mitten hand deformities)
- may have oral involvement
- Systemic lupus erythematosus
 - chronic multisystem progressive disorder
 - oral ulcerations/eruptive lip lesions
 - fever and arthralgias

XV. RAISED INTRA-ORAL SOFT TISSUE LESIONS—DIFFERENTIAL DX

Common

- Fibroma
 - most common intraoral soft tissue neoplasm
 - elevated lesion of normal color with smooth surface
 - may be firm or soft
 - surface may be secondarily ulcerated
 - commonly seen on lips, buccal mucosa, palate
 - tx: surgical excision
- Papilloma
 - viral etiology
 - sessile or pedunculated lesion with papillary surface
 - mucosa may be hyperkeratotic or normal in color
 - common on palate, tongue, buccal mucosa, lip, gingiva
 - tx: surgical excision

Uncommon

- Lingual thyroid nodule
 - midline on tongue near foramen cecum
 - smooth and vascular surface
 - 70% of patients with lingual thyroid lack normal thyroid tissue

Rare

- Lipoma
 - well circumscribed submucosal mass less than 1 cm in diameter
 - soft, freely movable
 - yellow color
 - common on tongue, buccal mucosa, gingiva, floor of mouth
 - tx: surgical excision

XVI. MULTILOCULAR RADIOLUCENCIES—DIFFERENTIAL DX

Common

- Ameloblastoma
 - may occur at any age, although most common between 20–40 years
 - commonly involves posterior mandible
 - arises from remnants of dental lamina

- multilocular, may cause root resorption
- tx: surgical excision

Uncommon

- Odontogenic keratocyst
 - unilocular or multilocular with thin sclerotic border
 - most common in posterior mandible—ascending ramus area
 - may be locally aggressive with expansion of bone and root resorption
 - often painful
 - basal cell nevus syndrome—AD
 - tx: surgical excision
- Ameloblastic fibroma
 - derived from epithelial and mesenchymal components of tooth apparatus
 - commonly found in posterior mandible, often associated with unerupted tooth
 - generally seen in patients under 20 years

Rare

- Odontogenic myxoma
 - uncommon, arises from mesenchyme of tooth germ
 - slow enlargement
 - more commonly involves posterior portion of jaws
 - slow progressive swellings, may cause facial deformity
 - unilocular or multilocular radiolucency which may have fine trabeculations, margins usually well-defined
 - may displace unerupted teeth, most commonly associated with missing or unerupted tooth
 - tx: surgical excision
- Central giant cell tumor
 - commonly involves mandible, may cross midline
 - locally invasive
 - multilocular with smooth or ragged border
 - frequently causes tooth displacement
 - tx: surgical excision
- Aneurysmal bone cyst
 - under 20 years peak incidence
 - tender, painful
 - swelling of involved area
 - expansile, cystic, honeycombed, or soap bubble radiolucency
 - tx: curettage
- Central hemangioma—hamartoma
 - listen for bruit, palpate for thrill

XVII. SOLITARY OR MULTIPLE RADIOLUCENCY WITH INDISTINCT OR RAGGED BORDERS—DIFFERENTIAL DX

Common

- Histiocytosis-X group (Langerhans cell histiocytoses):
 - variety of disorders of mononuclear phagocytes
 - Letterer-Siwe
 - infants

- prominent skin and visceral involvement
- Hand-Schuller-Christian
 - skull lesions
 - diabetes insipidus
 - exophthalmos
 - Eosinophilic granuloma
 - localized to bone

Uncommon

- Osteomyelitis
 - usually arises from odontogenic infection
 - necrosis of bone with pain, fever
 - Garre osteomyelitis
 - osteomyelitis of maxilla in infants
- Osteosarcoma
 - 2nd-3rd decade
 - mandible
 - males
 - most common initial symptom is jaw enlargement
 - x-ray: poorly delineated radiolucency, may be root resorption or sun-ray appearance with widening of periodontal ligament space
- Ewing sarcoma
 - mesenchymal tumor of bone usually seen in long bones
 - jaw involvement rare
 - 1st - 2nd decade, males more common than females
 - rapid swelling, pain, ulceration
 - metastasis to lungs and bone common
 - x-ray—moth eaten radiolucency
 - cortical infiltration may produce onion-skin appearance
 - may see sunburst like osteosarcoma

Rare

- Burkitt lymphoma
 - initially described in African children
 - mainly affects jaw and abdomen
 - strong association with EB virus
 - disease multifocal and may affect abdomen, pelvic viscera, retroperitoneal soft tissues facial bones, CNS
 - jaw involvement common initial finding
 - rapid expansion of jaws with loosening of teeth
 - x-ray: early loss of lamina dura, may have enlargement of follicular spaces, ill-defined radiolucencies
- Metastatic lesions
 - neuroblastoma

XVIII. PERIAPICAL RADIOPACITY-DIFFERENTIAL DIAGNOSIS

Common

- Idiopathic osteosclerosis
- Condensing osteitis

- commonly involves mandibular first molar
- reactive process of bone at apex in association with carious lesion
- symptoms related to pulpal status
- tx: pulpal therapy

Uncommon

- Cementoma
 - periapical cemental dysplasia
 - teeth vital
 - lesion occurs near and in periodontal ligament
 - lesions usually multiple
 - tx: none

Rare

- Tumor of cementoblasts
 - mandibular molar-premolar area
 - obliterates root outline
 - expansion of overlying bone may occur
 - tx: extraction
- Central cemento-ossifying fibroma
 - young adults
 - either jaw, mandible greater than maxilla
 - slow growing
 - displacement of teeth common
 - tx: excision

XIX. PERICORONAL RADIOLUCENCY— DIFFERENTIAL DX

Common

- Normal follicular space

Uncommon

- Dentigerous cyst
 - forms around crown of impacted tooth
 - may be expansive, destructive to surrounding bone
 - unilocular radiolucency
 - tx: marsupialization or excision

Rare

- Ameloblastic fibroma—derived from epithelial and mesenchymal components of tooth apparatus (odontogenic tumor)
 - posterior mandible, often associated with unerupted teeth
 - unilocular or multilocular radiolucency with sclerotic borders
 - slow enlargement
 - generally seen in patients under 20 years
- Ameloblastoma
 - usually seen in 20–40 years age group
 - mandibular-ramus area
 - may show expansion with obliteration of mucobuccal fold

XX. PERICORONAL RADIOLUCENCY CONTAINING —RADIOPAQUE FLECKS—DIFFERENTIAL DX

Common

- Adenomatoid odontogenic tumor

Uncommon

- Gorlin cyst - keratinizing and calcifying odontogenic cyst
 - most common in mandible, usually central within bone
 - may appear peripherally as gingival lesion
 - well circumscribed radiolucency with radiopaque flecks
 - may be associated with odontoma or other odontogenic tumors

Rare

- Ameloblastic fibro-odontoma—mixed tumor
 - may involve either jaw, usually in the molar region
 - involved with impacted tooth
 - well circumscribed expansile radiolucency with solitary or multiple opacities representing odontomas
 - consider ameloblastic odontoma-ameloblastoma and odontoma (extremely rare)
- Odontogenic adenomatoid tumor
 - common in anterior maxilla
 - often around impacted canines
 - may cause separation and displacement of teeth
 - tx: surgical excision
- Pindborg tumor - calcifying epithelial odontogenic tumor
 - mandible, premolar-molar region most commonly involved
 - painless swelling of jaw
 - well circumscribed radiolucency containing large and small radiopacities

XXI. RADIOLUCENCIES WITH DISTINCT BORDERS— DIFFERENTIAL DX

Common

- Primordial cyst
 - found in place of tooth
 - may arise from normal complement of teeth, or supernumerary, odontoma
 - unilocular, well-circumscribed
 - may be associated with apex of teeth, or crown of impacted tooth
 - often found in close proximity to mandibular premolar roots
 - tx: surgical excision and curettage
- Residual cyst
 - cyst that remains in place after removal of tooth
 - tx: surgical excision and curettage
- Stafne bone defect
 - Submandibular fossa

Uncommon

- Nasopalatine duct cyst—develops from remnants of nasopalatine duct

- midline radiolucency between maxillary incisors
- may cause divergence of roots
- may cause fluctuant swelling
- teeth are vital
- Median palatal cyst—arises from epithelium entrapped along fusion line of two palatal processes
 - underlying, radiolucency, opposite premolar area
 - may cause fluctuant swelling
- Globulomaxillary cyst—possibly a fissural cyst
 - inverted pear-shaped radiolucency between maxillary lateral and canine
 - may cause root divergence
 - teeth are vital
- Traumatic bone cyst (Solitary bone cyst)
 - usually in mandible
 - may cause expansion
 - teeth vital
 - tx: surgical exploration

Rare

- Lateral periodontal cyst
 - commonly found in mandibular canine-premolar area
 - teeth vital
 - etiology not clear
 - tx: surgical excision and curettage

XXII. SINGLE OR MULTIPLE RADIOPACITIES— DIFFERENTIAL DX

Common

- Foreign body
- Root fragments
- Odontoma
 - compound toothlets
 - complex - haphazard
 - may be associated with other odontogenic neoplasms

Uncommon

- Osteoma
 - benign tumor of mature bone
 - almost exclusively in face
 - mandible > maxilla
 - seen at lower mandibular border angle, lingual of ramus
- Gardner syndrome
 - autosomal dominant
 - multiple osteomas
 - epidermoid and dermoid cysts (50-60%)
 - multiple polyposis of large intestines with high malignant potential
- Dental findings
 - supernumerary teeth
 - delayed eruption

- Garre osteomyelitis
 - asymptomatic osteomyelitis
 - periosteal bone deposition
 - X-ray: onion skin appearance

Rare

- Albers-Schonberg disease—osteosclerosis
 - a severe form—AR
 - a mild—AD
 - obliteration of marrow spaces
 - brittle bones, pathologic fractures
 - extramedullary hematopoiesis
 - tx: bone marrow transplant
- Osteosarcoma—facial bones 2nd-3rd decade
 - mandible > maxilla
 - male > female
 - initial symptoms - jaw enlargement
 - gingiva may be ulcerated with pain, paresthesia, anesthesia
 - x-ray findings
 - two forms - one poorly delineated, destructive, root resorption
 - other—bone production predominates (sunburst) early finding—periodontal ligament space widening
- Ameloblastic odontoma
 - ameloblastoma and composite odontoma
 - mandible/maxilla
 - slowly expansile which may produce facial deformity
 - X-ray: central destruction with expansion of cortical plates with radiopaque mass within lesion
 - tx: wide excision similar to ameloblastoma
- Teratoma—heterotopic collection of tissue
 - cystic appearing lesion containing hair, teeth, etc

XXIII. CLEFT/LIP PALATE—DIFFERENTIAL DX

Common

- Cleft Lip/Palate
 - CL = 1/1000 L > R incidence greater in Asians
 - CL/P M > F
 - Isolated Cleft Palate: 1/2000

Uncommon

- Pierre Robin syndrome
 - glossptosis
 - micrognathia
 - cleft palate
 - 15–25% have heart disease
 - mandibular growth usually progresses normally
- Mandibulofacial dysostosis—Treacher Collins syndrome
 - 1st branchial arch, pouch, groove
 - downsloping palpebral fissures, depressed cheekbones, deformed pinnae,

- receding chin, large fish-like mouth
- hypoplastic mandible
- 30% cleft palate
- Cleidocranial dysostosis
 - brachycephalic
 - frontal & parietal bossing
 - depressed nasal bridge
 - delayed closure of sutures and fontanelles (wormian bones)
 - supernumerary teeth
 - clavicular defect
 - delayed or failure of exfoliation of 1° teeth
 - delayed eruption of 2° teeth
 - palate highly arched often with submucous cleft or complete cleft
 - roots lack layer of cellular cementum

Rare

- Oral-facial-digital syndrome
 - hypoplastic alar cartilages
 - hypotrichosis
 - brachycephaly
 - mental retardation
 - syndactyly, clinodactyly
 - median pseudo-cleft upper lip
 - cleft tongue, cleft palate
 - multiple hyperplastic frena with clefts
 - hypodontia: mandibular lateral incisors
 - hyperdontia: maxillary canines

XXIV. CRANIOSYNOSTOSIS—DIFFERENTIAL DX

Common

- Apert syndrome
 - premature closure of cranial sutures
 - syndactyly
 - Turribrachycephaly
 - High steep flat frontal bones
 - shallow orbits, ocular hypertelorism
 - parrot nose
 - 30% cleft palate
 - MR
 - crowded dentition
 - V-shaped maxilla
 - Class III with anterior openbite
 - retarded eruption
- Crouzon syndrome
 - premature closure of cranial sutures
 - brachycephalic
 - Maxillary hypoplasia
 - ocular hypertelorism
 - parrot nose
 - crowded dentition

- V-shaped arch
- Exophthalmus

Uncommon

- Pfeiffer syndrome
 - usually normal intelligence
 - broad thumbs and great toes
- Carpenter syndrome
 - acrocephaly
 - soft tissue syndactyly
 - congenital heart disease
 - MR
 - hypogenitalism
 - mild obesity

Rare

- Saethre-Chotzen syndrome
- Jackson-Weiss syndrome
- Antley-Bixler syndrome
- Baller-Gerold syndrome
- Cloverleaf skull syndrome

XXV. DWARFISM—DIFFERENTIAL DX

Common

- Achondroplasia – 80% sporadic, mutations, AD
 - 1/20,000 live births
 - short limbed dwarfism
 - enlarged head
 - depressed nasal bridge
 - short, stubby trident hands
 - lordotic lumbar spine
 - prominent buttocks
 - protuberant abdomen
- Hypopituitarism
 - well proportioned body, fine silky hair, wrinkled atrophic skin
 - hypogonadism
 - eruption and exfoliation delayed
 - malocclusion common due to small dental arch
 - panhypopituitarism may lead to other systemic problems

Uncommon

- Mucopolysaccharidosis (Hurler, Hunter, San Filippo, Morquio, Maroteaux Lamy)
 - All involve some degree of dwarfism
- Nutritional

Rare

- Chondroectodermal dysplasia; Ellis-van Creveld syndrome
 - bilateral manual postaxial polydactyly 40–50% have cardiac defects
 - chondrodysplasia
 - hidrotic ectodermal dysplasia

- occasionally congenital heart malformations
- fusion of middle of upper lip to maxillary gingival margin
- 25% natal teeth
- Hallerman-Streiff syndrome
 - dyscephaly
 - thin beaked nose
 - mandibular hypoplasia
 - hypotrichosis
 - small palpebral fissures
 - bilateral congenital cataracts
 - diminished body growth
 - oral findings:
 - high palatal vault
 - hypodontia
 - natal teeth
 - over-retained primary teeth
- Hypothyroidism
- Turner syndrome
 - 45 X karyotype
 - females only
 - near normal IQ
 - sterile
 - coarctation of aorta most common cardiac defect
 - webbed neck
 - enamel hypoplasia
- Osteogenesis imperfecta
 - tarda form—autosomal dominant (90%)
 - blue sclera
 - brittle bones
 - impaired hearing
 - dentinogenesis imperfecta
 - loose ligaments
 - congenital lethal form—autosomal recessive
- Cretinism—see macroglossia

XXVI. SELF-MUTILATION—DIFFERENTIAL DX

Common

- Mental retardation
- Autism
 - profound withdrawal
 - obsessive desire for preservation of sameness
 - skillful relation to inanimate objects
 - retention of an intelligent, pensive physiognomy
 - language development not understandable
 - often self-abusive, self-stimulating

Uncommon

- Congenital indifference to pain
 - autosomal recessive

- frequent scarring of face with mutilation of lips, arms, and legs, as well as phalangeal amputation due to self-mutilation
- tongue and lips especially subject to injury
- extensive decay not associated with pain

Rare

- Lesch-Nyhan syndrome
 - X-linked
 - MR
 - spastic CP
 - choreoathetosis
 - bizarre, self-mutilating behavior – including lip destruction with teeth
 - absence of hypoxanthine - guanine
 - phosphoribosyltransferase (enzyme involved in purine metabolism)

XXVII. ADDITIONAL READINGS AND WEB SITES

<http://www.cochrane-oral.man.ac.uk/>

The Cochrane Oral Health Review Group comprises an international network of health care professionals, researchers and consumers preparing, maintaining, and disseminating systematic reviews of randomized controlled trials in oral health. Oral health is broadly conceived to include the prevention, treatment and rehabilitation of oral, dental and craniofacial diseases and disorders.

<http://medgen.genetics.utah.edu/photographs.htm>

A nice atlas of a number of syndromes and oral conditions.

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=OMIM>

This database is a catalog of human genes and genetic disorders authored and edited by Dr. Victor A. McKusick and his colleagues at Johns Hopkins and elsewhere, and developed for the World Wide Web by NCBI, the National Center for Biotechnology Information. The database contains textual information and references. It also contains copious links to MEDLINE and sequence records in the Entrez system, and links to additional related resources at NCBI and elsewhere.

<http://www.uiowa.edu/~oprm/AtlasWIN/AtlasFrame.html>

An atlas of a variety of oral pathology including clinical, histological, and radiographic findings.

<http://www.usc.edu/hsc/dental/opath/Diseases/index.html>

An atlas of oral pathology divided by disease type, disease name, location, and appearance.

<http://www.oralpath.com/OralPathOLD2.htm>

An oral pathology website with links to a number of other related sites.

1. Pinto A. Pediatric soft tissue lesions. Dent Clin NA. 49:241-258, 2005.
2. Ellies M, Schaffranietz F et al. Tumors of the salivary glands in childhood and adolescence. J Oral Maxillofac Surg 64:104901058, 2006.

Chapter 4: FLUORIDE

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_FluorideTherapy.pdf

- I. MECHANISM OF ACTION
- II. FLUORIDE DENTIFRICES
- III. FLUORIDE RINSES
- IV. SELF-APPLIED GELS AND CREAMS
- V. FLUORIDE VARNISH
- VI. PROFESSIONALLY APPLIED GELS AND FOAM
- VII. FLUORIDATED WATER
- VIII. DIETARY FLUORIDE
- IX. FLUORIDE SUPPLEMENTS (T)
- X. FLUOROSIS ISSUE
- XI. ACUTE FLUORIDE TOXICITY
- XII. FLUORIDE CONCENTRATION OF COMMERCIAL PRODUCTS (T)
- XIII. FLUORIDE CONTENT OF INFANT FORMULAS (T)

- XIV. FLUORIDE COMPOUND/ION CONCENTRATION CONVERSIONS (T)**
- XV. PRESCRIPTION EXAMPLES (T)**
- XVI. ADDITIONAL READINGS**

I. MECHANISMS OF ACTION

- **Topical**
 - inhibits demineralization
 - promotes remineralization
- **Antibacterial**
 - concentrates in plaque
 - disrupts enzyme systems
- **Systemic** (controversial)
 - improves enamel crystallinity
 - reduces acid solubility
 - improves tooth morphology

II. FLUORIDE DENTIFRICES

- Best topical application for compliance
- F ion 0.1% = 1000 ppm
- Ingestion (0.2-0.3 mg can be swallowed by pre-school aged children when brushing twice a day)
- Recommendations/instructions for use
 - fluoride dentifrice >90% use in U.S.)
 - begin use with eruption of first tooth (minimal, smear amount)
 - very small, pea-sized amount in pre-school aged children
 - parents must supervise small children
 - rinse and expectorate following brushing

III. FLUORIDE RINSES

- OTC products: 0.05% NaF = 0.022% F ion = 220 ppm ~ 1 mg/5 mL (daily use)
- Rx products: NaF 0.2% (weekly use)
- Indications
 - orthodontics appliances
 - radiation therapy to head, face or neck
 - prosthetic appliances
 - high sucrose diet - either liquid or solids
 - high risk patients with history of caries, poor oral hygiene
- Risks: F ingestion and alcohol (some products)

IV. SELF-APPLIED GELS & CREAMES

- Indications
 - early childhood caries (ECC)
 - orthodontic appliances
 - radiation therapy to head, face or neck
 - high sucrose diet - either liquid or solids
 - high risk patients with history of caries and poor oral hygiene
 - reduced salivary flow
- Available preparations
 - 1.1% NaF = 5000 ppm (brush on, tray)

- 0.4% SnF₂=1000 ppm (brush on)
- 1.1% APF=5000 ppm (brush on, tray)

V. FLUORIDE VARNISH

- **Formulations**
 - 5% NaF varnish -22,500 ppm=22.5 mg/cc
- Recommendations/instructions for use of varnish:
 - very small amount used, so appropriate for pre-school aged children
 - advise child and parents of colored film on teeth
 - after application instruct parents/child not to brush until the next day

VI. PROFESSIONALLY APPLIED GELS/FOAM

- **Formulations**
 - 1.23% APF (gel and foam)=12,300 ppm=12.3 mg/cc
 - 2% NaF gel=9040 ppm=9 mg/cc
- Pellicle does not inhibit F uptake; no need for prophylaxis except for plaque/stain removal
- Indicated for school-aged children; use with caution in pre-school children due to ingestion concerns
- Acidulated F can etch porcelain restorations

VII. FLUORIDATED WATER

- **Issues**
 - controlled/natural water fluoridation
 - optimal range is 0.7 to 1.2 mg/L (0.7 to 1.2ppm)
 - diet: “halo” or “diffusion” effect
 - bottled water (variable F- content but usually low F- concentrations; may vary seasonally and with manufacturer)
 - filtration systems: reverse osmosis and distillation reduce F- to very low levels

VIII. DIETARY FLUORIDE

- **Sources of dietary fluoride**
 - casual ingestion of dentifrice/rinses
 - foods/beverages made with water including infant formula
 - formula (0.1-0.3 ppm on average for both soy/milk-based)
 - some juices, particularly grape juices have high F- (2-4ppm)
 - some processed chicken and seafood products have high F-
 - great variability in F- content of foods and beverages (varies by type, flavor, manufacturing site)

IX. FLUORIDE SUPPLEMENTS

Current Supplementation Regimen (1994)

Age	Water Fluoride Concentration (ppm)		
	<0.3	0.3-0.6	>0.6
Birth < 6 months	0.0*	0.0	0.0
6 months < 3 years	0.25	0.0	0.0
3 years < 6 years	0.50	0.25	0.0
6 to at least 16 years	1.00	0.50	0.0

*Dose in mg F ion

- Prescribing fluoride supplements
 - select doses: age, water F status (water analysis)
 - select supplement: drops, tablets, lozenge, rinse
 - write prescription: specific directions; maximum 120 mg per Rx
 - educate parent and patient
 - compliance often low
 - prenatal use (not shown effective)

X. THE FLUOROSIS ISSUE

- Fluorosis is a permanent intrinsic white-to-brown discoloration of enamel
- Occurs during tooth formation during the first few years of life
- Increase in prevalence due to increased ambient fluoride
- Sources of ingested fluoride
 - diet/“halo” effect from foods, beverages (including infant formula)
 - dentifrice consumption
 - previous supplementation schedules based on presumed lower fluoride intake
 - inappropriate Rx's for children already receiving adequate fluoride
- Measured by Dean's Index (very mild to severe), Tooth Surface Index of Fluorosis (TSIF), Fluorosis Risk Index (FRI) and Thylstrup- Fejerskov Index (TF)

XI. ACUTE FLUORIDE TOXICITY

- Symptoms of overdose—GI, CNS; death in 4 hr
- Probably toxic dose 5 mg F/kg
- Certainly lethal dose 16-32 mg F/kg (Hodge and Smith) 15 mg F/kg Whitford)
- Treatment – Induce vomiting or bind F
 - <8 mg F/kg: milk, observe >6 hr, refer if symptoms develop
 - ≥8 mg F/kg: syrup of ipecac, followed by milk; refer immediately
 - unknown dose: asymptomatic: treat as <8 mg F/kg symptomatic: give milk, refer immediately
 - poison control center: gastric lavage, IV calcium gluconate

XII. FLUORIDE CONCENTRATIONS OF COMMERCIAL PRODUCTS

<i>Office Topical Products</i>	<i>Fluoride concentration and compound</i>	<i>Concentration of fluoride ion</i>
APF	2.7% NaF	1.23% F-
NaF	4.4% NaF	2% F-
SnF ₂	10% SnF ₂	2.5% F-
NaF Varnish	5% NaF	2.2% F-
<i>Tray/ Brush On Products</i>		
Prevident™	1.1% NaF	0.5% F-
Gel Kam™	0.4% SnF ₂	0.1% F-
Dentifrices	1,000 ppm NaF or MFP	0.1% F-
Weekly Rinses	0.2% NaF	0.09% F-
Daily Rinses	0.05% NaF	0.02% F-

XIII. FLUORIDE CONTENT OF COMMERCIAL INFANT FORMULAS

<i>Formula</i>	<i>Fluoride Content</i>
Enfamil™	0.1-0.2 ppm
Prosobee™	0.2-0.3 ppm
Similac™	0.3 ppm
Isomil™	0.3 ppm
SMA™	0.21 ppm
Nursoy™	0.29 ppm

Source: Mead Johnson Inc., Ross Inc., Wyeth Inc.

XIV. FLUORIDE COMPOUND/ION CONCENTRATION CONVERSIONS

$X\% \text{ NaF} = 0.45 (X) \% \text{ F} = (0.45X) (103) \text{ ppm F}$ $0.5\% \text{ NaF} = 0.225\% \text{ F} = 2250 \text{ ppm F} X\%$ $\text{SnF}_2 = 0.25 (X) \% \text{ F}$ $\text{ppm F} = \text{mg F/L} = \text{mg F/mL}$

XV. PRESCRIPTION EXAMPLES

School-age High Caries Risk Patient

- Rx: NaF Creme (0.5%F-) or SnF2 Gel (0.4% SnF2)
- Disp: (varies depending on product)
- Sig: Brush on fluoride crème once/twice daily instead of toothpaste

6-Year-Old Residing in a Fluoride-Deficient Area

- Rx: Sodium Fluoride Tabs (1 mg F-/tablet)
- Disp: 120 tabs
- Sig: Before bed time after a thorough brushing, chew one tablet, swish and swallow

XVI. ADDITIONAL READINGS AND WEB SITES

<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5014a1.htm>

1. Shulman J, Willis L. Acute Fluoride Toxicity from Ingesting Home Use Dental Products in Children from birth to 6 Years of age, *J Public Health Dent*, 57: 150-158, 1997(summer).
2. Leverett D, Adair S, et al. Randomized Trial of the Effect of Prenatal Fluoride Supplements in Preventing Caries. *Caries Res.* 1:174-179, 1997.
3. Levy SM. Review of Fluoride Exposures and Ingestion. *Community Dent Oral Epidemiol* 22:173-180, 1994.
4. Kirisby MC, Levy SM, et al. Assessing Fluoride Concentrations of Juices and Juice Flavored Drinks. *J Am Dent Assoc* 127:895-902, 1996.
5. Burnell K. Systemic and Topical Fluorides. In, ADA Guide to Dental Therapeutics, ADA Publishing Co., Inc., pages 214-225, 1998.
6. Weintraub JA, Ramos-Gomez F et al. Fluoride varnish efficacy in preventing early childhood caries. *J Dent Res* 85:172-176, 2006.
7. Valeria CC, Marinho BDS, Higgains JPT et al. Systematic review of controlled trials on the effectiveness of fluoride gels for the prevention of dental caries in children. *J Dent Ed* 67:448-454, 2004.

Chapter 5: RADIOLOGY

AAPD ENDORSEMENT:

http://www.aapd.org/media/Policies_Guidelines/E_Radiographs.pdf

- I. RADIOGRAPHIC PRINCIPLES
- II. RADIATION HYGIENE
- III. TECHNOLOGICAL ADVANCES
- IV. RISKS AND EFFECTS
- V. TECHNIQUES/INDICATIONS
- VI. RECORDKEEPING/ADMINISTRATIVE MANAGEMENT
- VII. ADDITIONAL READING (T)

I. RADIOGRAPHIC PRINCIPLES

- General considerations
 - ionizing radiation regulations: federal government establishes performance standards for x-ray-generating equipment, state/local agencies govern users (eg, facility design, shielding, use and maintenance of equipment)
 - dental radiographic exposures contribute approximately 1% of total health care x-ray dosage
 - used to diagnose oral diseases, as well as to monitor dentofacial development and progress of therapy
 - health benefit will outweigh risk from radiation exposure if:
 - examination is clinically indicated and justified
 - technique is optimized to ensure high quality diagnostic images
 - principles followed to minimize exposure to patient, staff, and public
 - current guidelines require review of medical/dental history and clinical examination to determine need for x-ray exposure
 - obtain any available prior radiographs (from other offices if necessary)
 - order radiographs only if diagnostic yield expected to affect patient care
 - review all radiographs for evidence of caries, bone loss from periodontal disease, developmental anomalies and occult disease
 - rate of caries progression determines frequency of bitewings for low caries risk patient:
 - primary teeth ^ approximately one year for caries to progress through outer half of enamel and another year through inner half; 12 to 24 month interval is appropriate
 - permanent teeth ^ averages 3 years to progress through enamel although immature permanent teeth susceptible to faster progression; every 18-36 months is recommended for the adolescent.
 - use largest image receptor possible to obtain maximum information (eg, #2 films for bitewings to visualize erupting permanent teeth)
 - have parent (not occupationally-exposed personnel) hold film/immobilize child during x-ray exam if necessary; provide shielding (ie, leaded aprons, gloves)
 - if radiograph of diagnostic quality is unobtainable, confer with parent to determine appropriate management techniques (eg, advanced behavior guidance modalities, deferral, referral), considering relative risks and benefits of options
 - new imaging technology offers possibility of 3-D visualization of skeletal and other structures
 - maximize interpretive yield:
 - review in environment free from distraction
 - reduce room illumination to level of displayed images
 - eliminate glare
 - use magnification
 - utilize systematic approach
 - for conventional films: use opaque mount; view with variable luminance
 - for digital images: use software that permits adjustments of contrast, brightness, and negative-positive viewing

II. RADIATION HYGIENE

- Radiation safety
 - be familiar with state/local regulations as they may exceed these recommendations
 - ALARA (as low as reasonably attainable) Principle: do all that is possible to minimize exposure
 - use fastest image receptor available
 - intraoral: changing from D- to F-speed film or to modern digital image receptors reduces dose by factors of at least 2
 - extraoral: high-speed (400 or greater), rare earth screen-film systems or digital-imaging systems or equivalent
 - rectangular collimation: reduces radiation dose by factor of 4 to 5 without adverse influence on image quality; may be component of positioning devices or accessory devices
 - beam-receptor alignment devices (eg, XCP) for routine periapical radiography (only marginally effective for bitewings)
 - use 70 kVp or higher intraoral x-ray techniques
 - leaded apron/thyroid collar during exposures whenever possible
 - position operator behind barrier or at least 2 m from tube head during exposure, at location of minimum exposure (45° from primary beam as it exits patient; maximum exposure generally is in primary beam exiting from patient; maximum scatter 90 to 180° from primary beam as enters patient)
 - require personal dosimeters for pregnant clinical staff members and consider for others (some regulatory agencies may require for all occupationally-exposed personnel):
 - helps identify undesirable practices and unsuspected sources of high exposure
 - demonstrates compliance with individual dose limits and documents annual and lifetime dose
 - develop technique charts to assure staff uses proper exposures
 - implement quality assurance program for equipment (eg, machines, cassettes, digital image receptors, processors, leaded aprons/collars, view boxes), film, darkroom integrity and especially processor chemistry
 - use time/temperature for processing as recommended by manufacturer (sight developing is a major cause of poor quality films)
 - post warning signs
 - incorporate shielding design in new office construction

III. TECHNOLOGICAL ADVANCES

- Digital imaging
 - available as
 - direct digital radiography (DR): solid-state detectors [eg, charged-coupled devices (CCD), complementary metal oxide semiconductors (CMOS), charge injection devices (CID), flat panel]
 - computed radiography (CR): phosphor imaging plates [storage phosphors (SP) or photostimulable phosphors (PSP)]
 - indirect digital radiography: image initially recorded on conventional film and later digitally process to produce electronic image
 - advantages of DR/CR over conventional film:
 - image obtained may be immediately available (eg, with CCD receptors)
 - no darkroom requirement, elimination of processing chemistry

- image may be electronically manipulated (may compensate for exposure errors but no increase in diagnostic information content)
- easily stored and transmitted in digital form
- radiation dose may be less than with conventional intraoral films (receptors are available with speeds similar to or faster than E)
- disadvantages of DR/CR over conventional film:
 - smaller receptor for DR than conventional film (may require more exposures)
 - thicker CCD, CMOS, CID receptors make positioning difficult (may result in more remakes)
 - young children may not tolerate wired sensors/may chew on cables
 - SP imaging plates cannot be bent without permanent artifact
 - image quality: resolution is less but approaches diagnostic quality of conventional film in detecting occlusal and proximal caries, periodontal bone lesions, and root canal systems; diminished when printed
 - need for additional equipment (eg, computer)
 - limited standards, apparatus, and software to evaluate performance of dental digital-imaging systems

IV. RISKS AND EFFECTS

- Radiation biology
 - major biological risks are carcinogenesis, fetal effects and mutations
 - risks from low doses have been estimated by extrapolation from high-dose data (eg, Japanese atomic bomb survivors, individuals exposed by Chernobyl accident) with considerable disagreement concerning the model used for extrapolation
 - as risk of carcinogenesis is very small but significant, radiographs should be obtained only when there is expectation that diagnostic yield (including the absence of pathology) will influence patient care
 - tissues of growing children are more sensitive to exposure than adult tissues
 - juvenile thyroid is among the most sensitive organs to radiation-induced tumors, both benign and malign; risk decreases significantly with age at exposure, essentially disappearing after age 20
 - critical organs: eye (cataract), hematopoietic (leukemia), gonads (genetic disorders), skin (cancer), thyroid (cancer), pregnancy (birth defects), breast (cancer)
 - gonadal absorbed dose from a typical dental x-ray procedure is equivalent to about 1 hour of natural background radiation
 - with pregnancy:
 - radiation exposure to thyroid during pregnancy is associated with low birth weight
 - common dental projections rarely, if ever, deliver measurable absorbed dose to the embryo or fetus.
 - use protective thyroid collar and apron if x-ray exposure required
 - if dental care to be delayed until after delivery, also delay exposure

V. TECHNIQUES/INDICATIONS

- routine exposure of anterior periapical, occlusal, or panoramic films during the primary dentition is not indicated.
- Bitewings
 - may be oriented vertically or horizontally

- reveals approximately coronal halves of maxillary and mandibular teeth, interproximal contacts, and portions of interdental alveolar septa
- used primarily for detecting/monitoring occlusal and interproximal caries, crestal alveolar bone level; secondarily for eruption patterns, caries/restoration proximity to pulp spaces, primary molar furcation pathology, developmental anomalies
- primary/mixed dentition: 1 film/side to include distal of canine and erupted posterior dentition; adult dentition: 1-2 films/side depending on tooth size and alignment
- insert horizontally and rotate; wet packet/roll corners to improve acceptance; tab held against occlusal surfaces as patient closes in centric occlusion; vertical angle is + 8 to 10 degrees
- frequency of radiographic examination based on caries risk assessment (risk status may change over time, thus radiographic recall interval may change)
- Periapical/occlusal radiography
 - indicated for identified or suspected pathosis; to evaluate dental development, dentoalveolar trauma, or deep carious lesions; with oral involvement in known or suspected systemic disease
 - occlusal radiographs also indicated for:
 - unsatisfactory panoramic image due to abnormal incisor relationship
 - localizing tooth position
 - pathology (eg, assessing for sialoliths, buccolingual extent of pathological lesions, traumatic injury to bone)
 - palatal expansion
 - techniques for periapical images
 - paralleling: long axis of tooth parallels the image receptor
 - bisecting angle: central ray directed perpendicular to a plane that bisects the angle created by the long axis of the tooth and the film
- Panoramic
 - advantages compared to intraoral series:
 - rapid acquisition of a single image encompassing entire dental arches and their supporting structures
 - useful for craniofacial trauma
 - without discomfort of intraoral image receptor placement
 - minimal problems of infection control
 - effective dose is approximately equal to 4 intraoral images, both using state of art technique
 - reduced cost
 - disadvantages
 - image magnification/distortion
 - due to positioning difficulties, serial images show different distortions
 - resolution inadequate for definitive diagnosis of dentoalveolar trauma, incipient caries, pulpal pathology/beginning periapical lesions, root shape or resorption, or marginal periodontal disease
 - longer exposure time may prove difficult for/allow movement by uneasy patients
 - few manufacturers/models allow adjustment for patients' varying dimensions
- Lateral jaw radiography
 - lateral indicated when cooperation is substantially limited: bring head back and to side, place film (occlusal films/rigid cassette) against cheek, expose from opposite side below the angle of the mandible

- Devices/aids/other considerations
 - use positioning devices (eg, Snap-a-Ray®, XCP) for periapical films
 - OR/GA films: to stabilize, use Snap-a-Ray®, hemostat, Velcro® head strap, tape
 - gagging: topical lidocaine, cyclone rinse, distraction, nitrous oxide
- Clinical indicators for radiographic exposure
 - dentoalveolar trauma: multiple intraoral views as required to evaluate extent of injury or fracture of teeth/supporting structures
 - trauma extending beyond the dentoalveolar complex may dictate extraoral imaging
 - soft tissue trauma: use 1/4 normal exposure time for foreign objects
 - localization techniques:
 1. buccal object rule: 2 projections with tube shift, object is lingual if moves same direction as tube, buccal if moves opposite direction relative to reference
 2. true (ie, cross-sectional) occlusal
 - pulp therapy: post-op evaluation requires periodic clinical and radiographic assessment of treated tooth and supporting structures; pulpectomy, working length from existing films
 - extraction: pre-op needed; post-op if root retained
 - trauma: new film in 3-4 wks for periapical lesion, 6-8 weeks for root resorption
 - orthodontics: lateral/PA cephalometric, panoramic, intraoral films as required
 - periodontal conditions: selected PA's to assess level of supporting alveolar bone, condition of the interproximal bony crest, length and shape of roots, bone loss in furcations, and calculus deposits
 - temporomandibular joint (TMJ): definitive films indicated on a selected basis for joint sounds in the absence of other signs and symptoms of dysfunction
 - swallowed restoration or appliance: obtain anteroposterior abdomen film to assure object is in GI tract and not aspirated

VI. RECORDKEEPING/ADMINISTRATIVE MANAGEMENT

- record films exposed, including remakes, and diagnosis
- mount and label films with name and date
- consent should cover routine films
- do not mark or write on films
- infection control policy includes performance of oral radiographic procedures
- waste management policy includes film processing solutions and lead foil

VII. ADDITIONAL READINGS AND WEB SITES

1. American Dental Association, USDHHS. *The selection of patients for dental radiographic examinations - 2004*. Available at <http://www.ada.org/prof/resources/topics/radiography.asp>.
2. AAPD. *Clinical Guideline on Prescribing Dental Radiographs for Infants, Children, Adolescents, and Persons with Special Health Care Needs*. http://www.aapd.org/media/Policies_Guidelines/E_Radiographs.pdf.

3. National Council on Radiation Protection and Measurements. *Radiation protection in dentistry*. Report No. 145. NCRP Publications, Bethesda, Md. 2003, revised October 2004. <http://www.ncrponline.org>
4. Kantor ML. Longitudinal trends in the use of individualized radiographic examinations at dental school in the United States and Canada. *J Dent Educ.* 70:160-168, 2006.

TYPE OF ENCOUNTER	PATIENT AGE AND DENTAL DEVELOPMENTAL STAGE				
	Child with Primary Dentition (prior to eruption of first permanent tooth)	Child with Transitional Dentition (after eruption of first permanent tooth)	Adolescent with Permanent Dentition (prior to eruption of third molars)	Adult Dentate or Partially Edentulous	Adult Edentulous
New patient* being evaluated for dental diseases and dental development	Individualized radiographic exam consisting of selected periapical/occlusal views and/or posterior bitewings if proximal surfaces cannot be visualized or probed. Patients without evidence of disease and with open proximal contacts may not require a radiographic exam at this time.	Individualized radiographic exam consisting of posterior bitewings with panoramic exam or posterior bitewings and selected periapical images.	Individualized radiographic exam consisting of posterior bitewings with panoramic exam or posterior bitewings and selected periapical images. A full mouth intraoral radiographic exam is preferred when the patient has clinical evidence of generalized dental disease or a history of extensive dental treatment.	Individualized radiographic exam, based on clinical signs and symptoms.	
Recall patient* with clinical caries or increased risk for caries**	Posterior bitewing exam at 6-12 month intervals if proximal surfaces cannot be examined visually or with a probe		Posterior bitewing exam at 6-18 month intervals	Not applicable	
Recall patient* with no clinical caries and no increased risk for caries**	Posterior bitewing exam at 12-24 month intervals if proximal surfaces cannot be examined visually or with a probe	Posterior bitewing exam at 18-36 month intervals	Posterior bitewing exam at 24-36 month intervals	Not applicable	
Recall patient* with periodontal disease	Clinical judgment as to the need for and type of radiographic images for the evaluation of periodontal disease. Imaging may consist of, but is not limited to, selected bitewing and/or periapical images of areas where periodontal disease (other than nonspecific gingivitis) can be identified clinically.			Not applicable	
Patient for monitoring of growth and development	Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring of dentofacial growth and development	Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring of dentofacial growth and development. Panoramic or periapical exam to assess developing third molars		Usually not indicated	
Patient with other circumstances including, but not limited to, proposed or existing implants, pathology, restorative/endodontic needs, treated periodontal disease and caries remineralization	Clinical judgment as to need for and type of radiographic images for evaluation and/or monitoring in these conditions				

*Clinical situations for which radiographs may be indicated include but are not limited to:

A. Positive Historical Findings

1. Previous periodontal or endodontic treatment
2. History of pain or trauma
3. Familial history of dental anomalies
4. Postoperative evaluation of healing
5. Remineralization monitoring
6. Presence of implants or evaluation for implant placement

B. Positive Clinical Signs/Symptoms

1. Clinical evidence of periodontal disease
2. Large or deep restorations
3. Deep carious lesions
4. Malposed or clinically impacted teeth
5. Swelling
6. Evidence of dental/facial trauma
7. Mobility of teeth
8. Sinus tract ("fistula")

9. Clinically suspected sinus pathology

10. Growth abnormalities
11. Oral involvement in known or suspected systemic disease
12. Positive neurologic findings in the head and neck
13. Evidence of foreign objects
14. Pain and/or dysfunction of the temporomandibular joint
15. Facial asymmetry
16. Abutment teeth for fixed or removable partial prosthesis
17. Unexplained bleeding
18. Unexplained sensitivity of teeth
19. Unusual eruption, spacing or migration of teeth
20. Unusual tooth morphology, calcification or color
21. Unexplained absence of teeth
22. Clinical erosion

**Factors increasing risk for caries may include but are not limited to:

1. High level of caries experience or demineralization
2. History of recurrent caries
3. High titers of cariogenic bacteria
4. Existing restoration(s) of poor quality
5. Poor oral hygiene
6. Inadequate fluoride exposure
7. Prolonged nursing (bottle or breast)
8. Frequent high sucrose content in diet
9. Poor family dental health
10. Developmental or acquired enamel defects
11. Developmental or acquired disability
12. Xerostomia
13. Genetic abnormality of teeth
14. Many multisurface restorations
15. Chemo/radiation therapy
16. Eating disorders
17. Drug/alcohol abuse
18. Irregular dental care

Chapter 6: PERIODONTAL DISEASES AND CONDITIONS

AAPD ENDORSEMENT:

http://www.aapd.org/media/Policies_Guidelines/E_Plaque.pdf

http://www.aapd.org/media/Policies_Guidelines/E_PerioTherapy.pdf

http://www.aapd.org/media/Policies_Guidelines/E_PeriodontalDisease.pdf

- I. GINGIVAL DISEASE**
- II. CHRONIC PERIODONTITIS**
- III. AGGRESSIVE PERIODONTITIS**
- IV. PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASE**
- V. DEVELOPMENTAL OR ACQUIRED DEFORMITIES OR CONDITIONS**
- VI. CLINICAL PERIODONTAL EXAMINATION**
- VII. ADDITIONAL READINGS**

I. GINGIVAL DISEASE

Plaque-Induced Gingivitis

- Features
 - gingival inflammation
 - no loss of attachment or bone
 - reversible
- Prevalence
 - low in early childhood
 - peaks during puberty
 - 60% of teenagers have gingival bleeding on probing
- Etiology
 - plaque dependent
 - individual susceptibility variable
 - reactivity gradually increases with age
 - may be related to steroid hormones
 - puberty
 - pregnancy
 - menstruation
 - oral contraceptives
 - local factors may contribute
 - crowding
 - orthodontic appliances
 - mouthbreathing
 - eruption
 - calculus (about 10% of children and one third of teenagers have calculus)

Plaque-Induced Gingival Enlargement

- Clinical features
 - enlargement of interdental papilla and/or marginal gingiva
 - ranges from pale and fibrotic to red and friable
 - may be generalized or localized
- Caused by prolonged exposure to plaque
- Common local contributory factors
 - mouth breathing
 - orthodontic appliances

Dental Management

- Rigorous plaque control
- Gingivectomy or gingivoplasty may be required

Drug Influenced Gingival Enlargement

- phenytoin (dilantin, anti-epileptic)
- cyclosporin (immunosuppressant)
- calcium channel blockers (diltiazem, nifedipine, amlodipine)
- Clinical features
 - starts as painless enlargement of interdental papillae and marginal gingiva
 - fibro-epithelial growth
 - may progress to cover crowns
 - related to plaque control
 - does not occur in edentulous areas
 - regresses and may disappear after cessation of drug therapy

- Treatment
 - replace with alternate drug if possible
 - professional prophylaxis and rigorous home care
 - daily use of chlorhexidine may be beneficial
 - gingivectomy or gingivoplasty

Pyogenic Granuloma (Pregnancy Tumor)

- painless localized gingival enlargement
- blue-red color
- occurs in pregnancy

Gingival Abscess

- Localized, painful lesion of marginal gingiva or interdental papilla
- Sudden onset
- Caused by embedded foreign object (popcorn hull, fingernail fragment, etc.)

Dental Management

- Debridement and establish drainage
- Chlorhexidine irrigation

Pericoronitis

- Inflammation of gingiva covering partially erupted tooth
- Most common around erupting third molars
- Food trap, environment conducive to bacterial growth
- Pericoronal flap becomes inflamed and swollen
- Enlarged flap traumatized by occlusion, very painful

Dental Management

- Treated by debridement, antibiotic therapy for systemic involvement
- Chlorhexidine irrigation

Vitamin C-Associated Gingivitis

- Features
 - edematous, spongy gingiva, clinical appearance of non-specific gingivitis
 - spontaneous bleeding
 - impaired wound healing

Dental Management

- Treatment of underlying deficiency
- Plaque control

Acute Necrotizing Ulcerative Gingivitis (ANUG)

- Features
 - rapid onset of very painful gingivitis
 - interproximal and marginal necrosis and ulceration
 - may progress to necrotizing ulcerative periodontitis in immuno-compromised individuals
 - peak incidence in late teens and 20s in developed countries
 - common in young children in less developed countries
 - predisposing factors: malnutrition, stress, lack of sleep

Dental Management

- Local debridement (ultrasonic scaling)

- NSAIDs for pain
- Penicillin or metronidazole for systemic involvement

II. CHRONIC PERIODONTITIS

- Features
 - loss of periodontal attachment and bone
 - can be arrested
- Prevalence
 - about one-half the adult population significantly affected
 - 20% of 14-17 year-olds have attachment loss of at least 2mm in one or more sites
- Pathogenesis
 - bacterial plaque etiologic agent
 - polymicrobial infection, *Porphyromonas gingivalis* strongly associated, uncultivated species may be important
 - neutrophil primary host defense mechanism
 - host inflammatory response contributes to disease process
- Treatment of incipient attachment loss
 - oral hygiene instructions
 - scaling and root planing
 - correction of local contributory factors (overhanging restorations, calculus)
- Major risk factors
 - smoking
 - diabetes

Insulin-Dependent Diabetes Mellitus (Type I) and Chronic Periodontitis

- Increased incidence of gingivitis
- Increased risk and earlier onset of periodontitis (10-15% of teenagers)
- Poor metabolic control appears to increase risk of periodontitis
- Periodontal health associated with better blood glucose control
- Plaque and calculus levels comparable to normal controls

Dental Management

- Plaque control and scaling and root planing

III. AGGRESSIVE PERIODONTITIS (FORMERLY "EARLY ONSET PERIODONTITIS")

Localized Aggressive Periodontitis (LAP) in the primary dentition (formerly prepubertal periodontitis [PPP])

- Features
 - attachment loss and bone loss around primary teeth
 - affects only some of the deciduous teeth
 - most commonly affects primary molars
 - inflammation not a prominent feature
 - children otherwise systemically healthy
 - prevalence less than 1%
 - may subsequently progress to LAP in the permanent dentition

- Suggested etiologic factors
 - leukocyte chemotactic defect
 - cementum defect
 - usually but not always associated with *Actinobacillus actinomycetemcomitans*

Dental Management

- Little data
- Scaling and root planing or extraction of primary teeth
- Antibiotic therapy: amoxicillin and metronidazole for 7 to 10 days

Localized Aggressive Periodontitis (LAP) in the permanent dentition

- Features
 - localized bone loss around permanent incisors and first permanent molars and no more than two other teeth
 - patient otherwise systemically healthy
 - attachment loss rapid (3 times rate of adult onset disease)
 - may have minimal plaque and inflammation
 - often first detected at age 10 to 15 years
 - 50% of cases preceded by bone loss in primary dentition
 - probably the same disease as LAP in the primary dentition
- Prevalence
 - 0.2% in whites and 2.6% in blacks
- Etiology
 - Actinobacillus actinomycetemcomitans* in most but not all cases
- Suggested etiologic factors
 - defects in neutrophil chemotaxis and phagocytosis, over-reactive monocyte response, genetic defects in gene encoding IgG2

Dental Management

- Combination of scaling and root planing and systemic antibiotics
 - amoxicillin and metronidazole for 7 to 10 days
 - antibiotic therapy should be repeated until *A. actinomycetemcomitans* is eradicated
 - microbiologic monitoring needed to ascertain eradication of *A. actinomycetemcomitans*
 - local antibiotic therapy not effective
- Surgical correction of defects
- Regenerative therapy

Generalized Aggressive Periodontitis

- Features
 - generalized attachment loss in the permanent dentition in persons under 30 years of age
 - probably the result of progression of LAP
- Treatment same as for LAP

IV. PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASES

Hypophosphatasia

- Features
 - genetic disorder

- 4 levels of severity: perinatal (lethal), infantile, childhood, and adult
- phenotypes range from premature loss of deciduous teeth to severe bone abnormalities leading to neonatal death
- the earlier the presentation of symptoms, the more severe the disease
- in mild forms early loss of primary teeth may be first clinical sign, usually lower incisors
- abnormal cementum
- pulp chambers may be abnormally large
- Etiology
 - deficient or defective alkaline phosphatase
- Diagnosis
 - low serum alkaline phosphatase (normal values are higher in children, so normal control values must be adjusted for age)
 - increased phosphoethanolamine in urine
 - radiographic examination of bones
- Dental prognosis
 - permanent teeth often not affected

Dental Management

- Supportive therapy

Leukocyte Adhesion Defect (LAD)

- Autosomal recessive defect
- Leukocyte surface glycoprotein defect resulting in poor leukocyte adherence
- Oral manifestation of LAD is generalized periodontitis in the primary and young permanent dentition
- Severe generalized periodontitis refractory to treatment
- Frequent respiratory, skin, ear and other soft tissue bacterial infections

Dental Management

- rigorous oral hygiene, antibiotic therapy and extraction of affected teeth

Papillon-LeFèvre Syndrome

- Genetic disorder
- Features
 - palmar and plantar hyperkeratosis
 - attachment and bone loss resulting in premature loss of primary and permanent teeth
 - inflammation can be severe
 - may be *Actinobacillus actinomycetemcomitans* infection

Dental Management

- Rigorous oral hygiene
- Antibiotic therapy and extraction of affected teeth have been used with variable success

Down Syndrome

- Prevalence of periodontal disease 60-100% in those under 30 years of age
- May be noted in deciduous dentition
- Mandibular incisors often affected
- Suggested etiology of periodontitis

- poor vascularization of gingival tissues
- T-cell maturation defect or PMN chemotactic defect

Chediak-Higashi Syndrome

- Rare, autosomal recessive
- Oculocutaneous albinism, photophobia, nystagmus, peripheral neuropathy
- Severe gingivitis and periodontitis

Neutropenia

- Decreased circulating PMNs
- Several forms
 - cyclic neutropenia
 - chronic benign neutropenia of childhood
 - chronic idiopathic neutropenia
 - familial benign neutropenia
- Periodontal symptoms
 - severe gingivitis with ulcerations
 - attachment loss and alveolar bone loss
 - early loss of deciduous teeth
 - severe periodontal disease in permanent dentition
- History of other recurrent soft tissue infections
- Diagnosed by white blood cell differential count

Dental Management

- rigorous oral hygiene, antibiotic therapy and extraction of affected teeth

Langerhans Cell Histiocytosis (formerly “histiocytosis X”)

- Group of disorders with variable symptoms resulting from abnormal proliferation and dissemination of histiocytic cells of the Langerhans system
- About 10% show oral involvement
- Bone lesions may produce “floating teeth”
- Gingival swelling
- Diagnosis by biopsy

Acute Leukemia

- Gingival enlargement due to infiltration with leukemic cells may be presenting symptom, particularly of acute monocytic leukemia (AML): gingiva appears hyperplastic, edematous, bluish red
- Petechiae or mucosal ulcerations may be present with any form of leukemia
- Initial diagnosis by CBC

V. DEVELOPMENTAL OR ACQUIRED DEFORMITIES OR CONDITIONS

- Mucogingival defects
 - pocket depth exceeds width of attached keratinized gingiva
 - lower incisor most common location
 - in children defect may result from labial positioning of tooth erupting through band of attached gingiva

- Localized areas of gingival recession (“stripping”)
 - usually due to labial malposition of tooth
 - most common in lower incisors
 - may be difficult to clean
 - may produce mucogingival defect
- High labial frenum attachment
 - may exacerbate stripping and mucogingival defects in mandible
 - may be cosmetically objectionable in maxilla
- Treatment
 - narrow band of attached gingiva can be maintained with good plaque control
 - gingival graft may be needed to create anatomic contours conducive to good plaque control
 - frenectomy may be indicated to relocate frenal attachment
 - if orthodontic treatment is indicated, surgery is usually postponed until after lingual repositioning of lower incisors or closing of maxillary midline diastema

VI. CLINICAL PERIODONTAL EXAMINATION

- Gingival inflammation
 - signs
 - color change
 - edema
 - bleeding on gentle probing
 - gingival crevicular exudate
 - Gingival Index (GI) widely used to assess gingival health
- Plaque accumulation
 - several indices have been used to quantitate plaque levels
 - most include assessment of portion of surface covered by plaque
- Bone loss
 - the height of the interproximal crest should be 1-2 mm below the CEJ on bitewing radiograph
- Attachment loss
 - attachment level determined by subtracting the distance from the CEJ to the free gingival margin from the probing pocket depth (PPD) (distance from the free gingival margin to the bottom of the pocket)
 - measured at 6 sites per tooth
 - more difficult to determine in younger patients because CEJ is usually below free gingival margin
 - due to inaccuracies in measurement, loss or gain of 2 mm or more clinically meaningful
- Mucogingival problems
 - to determine width of attached gingiva locate mucogingival junction and measure to free gingival margin
 - note sites with less than 1 mm of attached gingiva
- Periodontal Screening & Recording((PSR)
 - system designed for easier and faster screening of periodontal health for adults
 - uses probe with colored band from 3.5 to 5.5 mm
 - if band is even partially submerged, a complete periodontal exam is indicated
 - can be used in children and adolescents, but erupting teeth give false positives

VII. ADDITIONAL READINGS

- Albandar JM, Rams TE. Risk factors for periodontitis in children and young persons. *Periodontol* 2000. 2002;29:207-222.
- Califano JV. Position paper: periodontal diseases of children and adolescents. *J Periodontol*. 2003;74:1696-1704.
- Oh TJ, Eber R, Wang HL. Periodontal diseases in the child and adolescent. *J Clin Periodontol*. 2002;29:400-410.
- Sollecito TP, Sullivan KE, Pinto A, Stewart J, Korostoff J. Systemic conditions associated with periodontitis in childhood and adolescence. A review of diagnostic possibilities. *Med Oral Patol Oral Cir Bucal*. 2005;10:142-150.

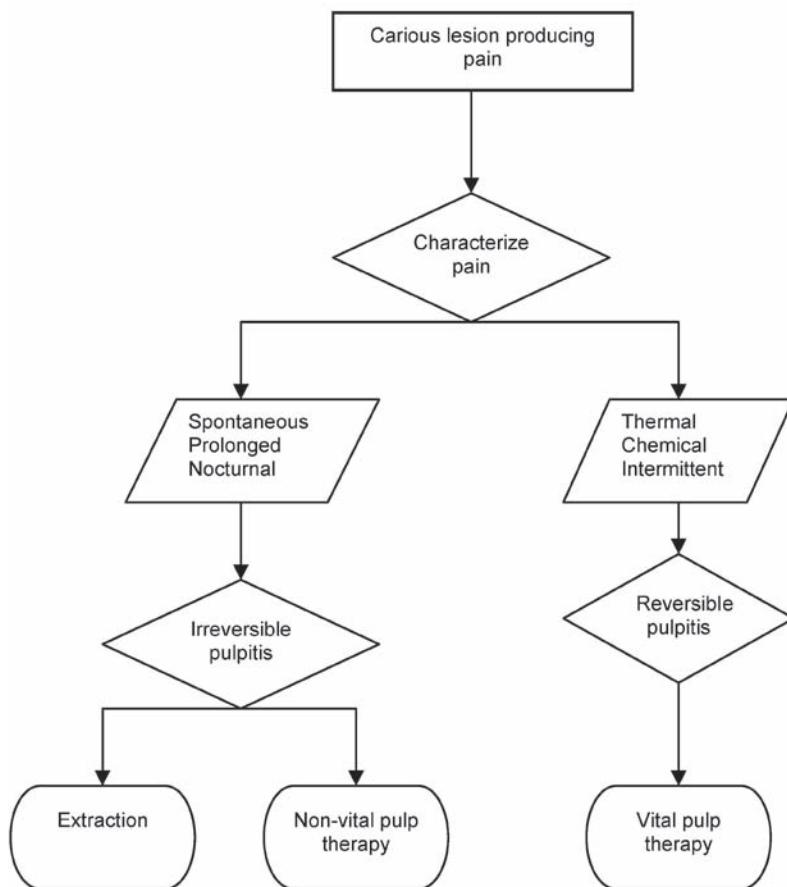
Chapter 7: PULP THERAPY IN PRIMARY AND YOUNG PERMANENT TEETH

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_Pulp.pdf

- I. CLINICAL AND RADIOGRAPHIC ASSESSMENT OF PULP STATUS (T)**
- II. VITAL PULP THERAPY FOR PRIMARY TEETH (T)**
- III. NON-VITAL PULP THERAPY FOR PRIMARY TEETH (RX)**
- IV. VITAL PULP THREATMENT IN YOUNG PERMANENT TEETH**
- V. NON-VITAL PULP THERAPY FOR YOUNG PERMANENT TEETH**
- VI. ADDITIONAL READINGS**

I. CLINICAL AND RADIOGRAPHIC ASSESSMENT OF PULP STATUS



Candidate teeth for vital pulp therapy:

- tooth with deep caries without pulp exposure
- carious or traumatic pulp exposure
- transitory thermal and/or chemical stimulated pain
- physiologic mobility
- normal soft tissues
- no percussion sensitivity (except in cases of food impaction)
- intact continuous ligament space
- intact periapical and/or furcation bone
- Less than 1/3 physiologic root resorption
- tooth is restorable

Candidate teeth for non-vital pulp therapy or extraction:

- carious or traumatic pulp exposure

- spontaneous pain
- persistent thermal and/or chemical stimulated pain
- pathologic mobility
- inflamed soft tissues, parulis
- percussion sensitivity
- widened and/or discontinuous ligament space
- furcation and/or periapical radiolucencies
- external and/or progressive internal resorption
- dystrophic intrapulpal calcifications

Confounding factors in diagnosis of pulp status

- lymphadenopathy - rule out URTI, otitis media
- color of pulpal bleeding not reliable indicator of pulp histologic status
- excessive bleeding strongly correlated with degenerative changes
- 1/3 of teeth with carious pulp exposures have 'normal' pulps
- 1/3 of teeth with deep caries with no pulp exposures have 'abnormal' pulps

Reliability of clinical assessments

	Primary tooth	Immature permanent tooth	Mature permanent tooth
electrical	—	+	+
thermal	+	+	++
percussion	+	+	++

II. VITAL PRIMARY TOOTH PULP THERAPY

Treatment objectives

- eradicate potential for infection
- maintain tooth in a quiescent state
- preserve space for underlying permanent tooth
- retain 1° tooth, if 2° tooth is congenitally absent

Protective base

- Indications: tooth with exposed dentin after caries removal.
- Objectives: preservation of vitality, minimize injury to the pulp, minimize postoperative sensitivity

Indirect Pulp Treatment (IPT)

- Indications: tooth with deep carious lesion, vital pulp
- Objectives: preservation of vitality, arrest of caries advance, formation of tertiary dentin.
- Technique: excavation of most affected dentin, application of medicament over thin layer of sound or carious dentin with no clinically evident pulpal exposure, place $\text{Ca}(\text{OH})_2$ or glass ionomer cement, restoration should seal completely, e.g. stainless steel crown
- re-entry for completion of caries removal is not necessary.

Direct Pulp Capping (DPC)

- Indications: small mechanical or traumatic exposures in primary teeth, tooth is restorable - doubtful prognosis
- Contraindications: carious exposure in primary tooth, persistent inflammation, internal resorption, calcific metamorphosis
- Objectives: preserve pulp vitality under tertiary dentin bridge
- Ca(OH)_2 may produce internal resorption
- pulpotomy is preferred due to predictable outcomes

Pulpotomy

- Indications: carious/iatrogenic pulp exposure, coronal pulp affected/infected, radicular tissue vital (or affected but vital) as judged by clinical and radiographic means, tooth is restorable
- Objectives: to maintain tooth in symptomless state until tooth is not strategic, healthy supporting tissues, no harm to succedaneous tooth
- Technique: excavate caries, amputate coronal pulp, achieve hemostasis, treat radicular pulp with medicament/technique, restore with permanent restoration

Technique	Method of action	Acceptable outcomes	Comments
Formocresol (FC) Full-strength or Buckley's	Tissue fixation	62-97%	Gold standard, safety concerns, distribution to viscera with multiple pulpotomies, 3-5 minutes application
Dilute FC (1/5 concentration)	Tissue fixation	Equivalent to full-strength FC	5 minute application, most in-office dilutions inaccurate
Ferric sulfate	Hemostatic	Equivalent to FC	Most 'A' level evidence of all vital 1° techniques
MTA	Mineralization	Equivalent to FC	Expensive
Vital primary tooth root canal therapy	Obturation of pulp space	Superior to FS	Technically challenging (single randomized control study)
Laser (Er:YAG)	Hemostatic	Equivalent to FC	Single randomized control trial
Calcium hydroxide (Ca(OH)_2)	Mineralization	31-87%	Demonstrated worse outcome than FC in randomized control trial
Electrosurgical	Surgical	Poorer than FC	Lateral heat production results in damage to supporting tissues
Zinc oxide eugenol (ZOE) pulpotomy	Palliative	57%	Internal resorption common outcome

Formocresol Pulpotomy (FC)

- Full-strength or Buckley's FC (19% formaldehyde, 35% cresol in 15% glycerin & water): 3-5 minutes application
- Dilute FC (1/5 concentration: 1 part FC: 4 parts vehicle (3 parts glycerin: 1 part water): 5 minute application
- dilution mixture settles out, re-mixing indicated
- histologic zones in FC treated radicular pulp
 - acidophilic zone: fixation (coronal)
 - pale staining zone: atrophy (middle)
 - broad zone of inflammatory cells (apical)
- bactericidal
- no dentinal bridging, but calcific changes evident
- persistent chronic inflammation
- succedaneous tooth damage a small risk
- exfoliation accelerated
- cellular toxicity
- immune sensitization risk
- humoral and cell-mediated responses - controversial
- mutagenic and carcinogenic potential - controversial

Ferric sulfate (FS)

- 15.5% in aqueous base, pH = 1
- denatures protein & forms ferric ion complex that occludes cut blood vessels
- shorter application time than FC (10-15 seconds)
- self-limiting internal resorption reported

Mineral trioxide aggregate (MTA)

- dental cement with discrete crystals and amorphous structure, pH = 12.5
- pulp canal obliteration common
- promising clinical results

Vital primary tooth root canal therapy

- survival superior to FS pulpotomy (one study with limited numbers)
- resorption of ZOE canal filling a concern
- incisors with carious exposure have better outcomes than traumatized incisors
- overfilling associated with poorer outcomes

III. NON-VITAL PULP THERAPY FOR PRIMARY TEETH

Treatment options

- pulpectomy: necrotic or abscessed
- extraction

Pulpectomy

- Indications
 - strategic tooth for arch development

- restorable
- adequate root remaining
- Objectives
 - resolution of infective process
 - no signs or symptoms of pathosis
 - radiographic success: adequate fill, healthy supporting tissues, no external resorption, normal exfoliation
- Technique
 - eradicate radicular pulp remnants
 - irrigate with sodium hypochlorite
 - obturate with resorbable cement
 - use lentulo spiral, pressure syringe, cone technique to fill
- Outcomes
 - ~85% acceptable outcomes for molars or incisors

Criteria for ideal root filling

- antiseptic
- resorbable
- harmless to adjacent tooth germ
- radiopaque
- easily inserted
- easily removed
- biocompatible

Zinc oxide and eugenol

- most common material in North America
- biocompatibility is questionable
- resorbability is questionable

Calcium hydroxide ($\text{Ca}(\text{OH})_2$)

- biocompatible
- longevity of material & action are questionable

Alternative root filling materials

Kri 1 Paste(Pharmachemie,Zurich,Switzerland)

- formula
 - p-chlorophenol 2.025%
 - camphor 4.860%
 - menthol 1.215%
 - iodoform 80.80%
- action
 - highly resorbable
 - bacteriocidal
 - healthy tissue ingrowth at apex
- Kri 1 vs. ZOE
 - Acceptable outcomes: Kri 1 84% vs. ZOE 65%
 - Kri 1 overfill more favorable outcomes than ZOE overfill

Vitapex (Neo dental Chemical Products, Tokyo, Japan)

- iodoform
- Ca(OH)_2

Endoflas (Sanlor Laboratories, Colombia, South America)

- iodoform
- Ca(OH)_2
- zinc oxide

Maisto's paste

- iodoform
- parachlorophenol
- camphor - menthol

Ledermix (Lederle Laboratories, Wolfatshausen, Germany)

- dimethylchlorotetracycline
- triamcinolone

Empiric therapy for acute infection:

Penicillin: 25-50 mg/kg/day in 3-4 divided doses for 7 days (max dose: 3g/day)

Amoxicillin: 20-40mg/kg/day in 3 divided doses for 7 days (max dose: 3g/day)

If penicillin allergic: Clindamycin: 10-25 mg/kg/day 3-4 divided doses for 7 days (max dose: 1.8g/day)

IV. VITAL PULP TREATMENT FOR YOUNG PERMANENT TEETH

Protective base

- see Protective base above

IPT

- see IPT above
- place Ca(OH)_2 or glass ionomer cement; leakage-free restoration indicated
- place permanent restoration
- re-entry for completion of caries removal is controversial

DPC

- see DPC above

Pulpotomy

Objectives of pulpotomy (Ideal)

- Maintain vitality of radicular pulp

Objectives vary with treatment choice

- root-end closure: apexogenesis
- eliminate need for surgery
- facilitate gutta percha fill with apical stop

Ca(OH)_2

- superficial necrosis
- deeply staining zone: basophilic Ca(OH)_2 elements

- coarse fibrous tissue: fibroblasts odontoblasts orient to periphery
- calcified dentin bridge at 4-8 weeks
- vital pulp tissue
- antibacterial
- good results reported with MTA

Apexogenesis

- remove coronal portion of vital pulp
- place agent to preserve radicular vitality
- encourage continued root development
- emergency procedure for future RCT
- promote tertiary dentin formation
- no evidence of inflammatory resorption
- no evidence of root and periradicular pathosis

Partial pulpotomy (Cvek)

- preservation of cell rich coronal pulp
- increased healing potential due to preserved pulp
- physiologic apposition of cervical dentin
- obviate need for RCT
- natural color and translucency preserved
- maintenance of pulp test responses

Partial pulpectomy

- partial extirpation of radicular pulp
- indicated for persistent hemorrhage from pulp stumps
- $\text{Ca}(\text{OH})_2$ is medicament of choice
- good results reported with MTA

V. NON-VITAL PULP THERAPY FOR YOUNG PERMANENT TEETH

Objectives

- promote continued apical root development
- achieve apical closure

Apexification ($\text{Ca}(\text{OH})_2$; Frank technique)

- necrotic tissue removal short of the apex
- place agent ($\text{Ca}(\text{OH})_2$) in canals
- may be necessary to replace $\text{Ca}(\text{OH})_2$ q3-6 months
- action of $\text{Ca}(\text{OH})_2$
bactericidal
low grade irritation induces hard tissue formation
dissolves necrotic debris

Apexification (single appointment MTA)

- MTA placed in apical 1/3 of canal
- bonded core to fill canal
- permanent restoration placed

Evaluation of success

- asymptomatic
- radiographic absence of pathology
- continued root development
- hard tissue barrier at apex

VI. ADDITIONAL READINGS

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Chapter 8: RESTORATIVE DENTISTRY

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_Restorative.pdf

http://www.aapd.org/media/Policies_Guidelines/RS_SedationRecord.pdf

- I. AMALGAM (T)
- II. CAVITY LINERS
- III. CAVITY VARNISHES
- IV. STAINLESS STEEL CROWNS (T)
- V. RESIN-BASED COMPOSITES AND BONDING AGENTS
- VI. GLASS IONOMER CEMENTS
- VII. CAVITY PREPARATION IN PRIMARY TEETH
- VIII. MANAGING OCCLUSAL SURFACES OF YOUNG PERMANENT TEETH (T)
- IX. ADDITIONAL READINGS

I. AMALGAM

Advantages

- Economics
- Time efficiency
- Less sensitive to operator variables
- Historical longevity
- Wide application potential
- Reasonable clinical serviceability
- Physical properties relative to posterior restorations

Disadvantages

- Esthetics
- No bonding to tooth structure
- Initial microleakage
- Necessity for mechanical retention
- Environmental concerns associated with proper disposal
- AAPD Objectives are to restore form and function in primary and permanent teeth and maintain vitality

The dental literature supports the safety and efficacy of dental amalgam in all segments of the population. Furthermore, the dental literature supports the use of dental amalgam in the following situations:

1. Class I restorations in primary and permanent teeth
2. Two-surface Class II restorations in primary molars where the preparation does not extend beyond the proximal line angles
3. Class II restorations in permanent molars and premolars
4. Class V restorations in primary and permanent posterior teeth

Bonded Amalgam

- Amalgam bonding agents may reduce need for some mechanical retention
- Superior to cavity varnish
- Dentin bonding agents 4-META hybrid layer, metal ionomer cement

Mercury Issue

- More research is needed on the health effects of low-level mercury exposure and the contribution that dental amalgam makes to these low mercury levels

Reducing Occupational Exposure to Mercury

- Be aware of hazards
- Know potential sources of mercury spills
- Provide proper ventilation
- Periodically monitor mercury vapor levels in office and urinary levels in staff
- Use precapsulated alloys
- Avoid skin contact
- Use HVE when finishing or removing amalgams; change face mask afterwards
- Store amalgam scrap under used fixer

- Dispose of scrap appropriately
- Clean mercury spills with commercial kits
- Wear professional clothing only in the dental operatory

Durability in Primary Teeth

Study	Survival Rate (%)	Time Period
Holland et al.	46	3 years
Ovist et al.	50	2 years
Roberts et al.	66	5 years

Kilpatrick, *J Dent* 21:57, 1993.

Summary of Mean Survival Time (MST) Prediction

Study	Patient Age at Placement	MST
Mitchell/Walls	3 years	11 months
Mitchell/Walls	7-8 years	44 months
Levering/Messer	4 years	5 years (51%)
Levering/Messer	>4 years	5 years (70%)
Roberts/Sherriff	4-5 years	5 years (44%)
Welbury et al.	5-11 years	41.4 months

II. CAVITY LINERS

- Calcium hydroxide: antimicrobial effects
- Advantages of light-cured calcium hydroxide
 - less soluble
 - no mixing needed
 - controlled working time
 - can add material for thickness
- Zinc oxide-eugenol: not for use under self-cured, resin-based composites
- Glass-ionomer advantages
 - bond to tooth structure
 - release fluoride
 - can be applied over calcium hydroxide
 - light-curing type available
- Glass-ionomer disadvantages
 - moisture sensitivity

III. CAVITY VARNISHES

- Solution of one or more resins (copal, nitrated cellulose) in solvent
- Thin multiple layers reduce leakage around margins and walls of amalgam restorations
- Do not possess mechanical strength

- Do not provide thermal insulation
- Trend away from use of cavity varnish and calcium hydroxide liners

IV. STAINLESS STEEL CROWNS

Alloy: chrome steel

- 18% chromium, 8% nickel
- Carbon content 0.8 -to 20%

Properties

- Heating does not increase strength
- Work hardens
- High chromium reduces corrosion
- Soldering with flux reduces corrosion resistance

Alloy: nickel-chrome (Ion crowns, 3M™)

- 77% nickel, 15% chromium, 7% iron

Indications and objectives (AAPD)

The dental literature supports the use of stainless steel crowns in the following situations:

1. Children at high risk exhibiting anterior tooth decay and/or molar caries may be treated with stainless steel crowns to protect the remaining at-risk tooth surfaces
2. Children with extensive decay, large lesions or multiple surface lesions in primary molars should be treated with stainless steel crowns
3. Strong consideration should be given to the use of stainless steel crowns in children who require general anesthesia
- Objectives are to restore form and function, maintain vitality where possible

Success Rates: Comparison with Class II Amalgams

Predicted Time to Failure (years)	Age < 4 Years	Age > 4 Years
1	17%	8%
5	49%	24%
10	69%	36%

Messer and Levering: Pediatr Dent 10: 1988.

Laminated anterior stainless steel crowns

- Facing held on with mesh or by etching to metal
- Most not crimpable
- Wider mesiodistally
- Laminate bulky facially
- Quicker to place but susceptible to facing loss

V. RESIN-BASED COMPOSITES AND BONDING AGENTS

Microfill

- Excellent esthetics and finishability
- Elastic-allows for tooth flexion under loading

Hybrids (bimodal or trimodal blends of fine or microfine)

- Excellent long-term results
- Good for posterior and anterior use
- Low thermal expansion and polymerization shrinkage
- Relatively high wear resistance
- May contain filler up to 70% by volume

Fine particle (often contains distributions of > 2 sizes of particles)

- High wear resistance
- Good mechanical properties
- Appropriate for posterior use
- Veneers needing strength
- Rougher surface than micro-fil or hybrid
- High filler loading (60-70% by volume, 77-88% by weight)

Objectives (AAPD)

- Restore all surfaces of anterior teeth, including those with developmental or acquired defects
- Restore form, function, and esthetics; maintain vitality

Concerns with posterior composites

- Moisture contamination
- Postoperative sensitivity
- Polymerization shrinkage
- Patency of bond with enamel and dentin
- Technique sensitive

Strip crowns

- For Class III, V restorations on anteriors
- Uses celluloid crown form, composite resin, etched enamel, dentin, bonding agent
- Not indicated for grossly decayed teeth
- Pin hole for composite release
- Prep should allow for bulk and retention

Indications

For all resin-based composite restorations, teeth must be adequately isolated to prevent saliva contamination. The dental literature supports the use of highly filled resin-based composites in the following situations:

1. small pit and fissure caries where conservative preventive resin restorations are indicated in both the primary and permanent dentition;
2. occlusal surface caries extending into dentin;

3. Class II restorations in primary teeth that do not extend beyond the proximal line angles;
4. Class II restorations in permanent teeth that extend approximately one-third to one-half the buccolingual intercuspal width of the tooth;
5. Class V restorations in primary and permanent teeth;
6. Class III restorations in primary and permanent teeth;
7. Class IV restorations in primary and permanent teeth;
8. strip crowns in the primary and permanent dentitions.

Bonding agents—enamel

- 15- to 30-sec etch with 37% phosphoric acid creates micro-retentive porosity
- Resin penetrates porous areas producing enamel tags (10-75 microns)
- Traditional resins are hydrophobic BisGMA or urethane dimethacrylate resins
- Bond strengths of enamel agents approach 20 MPa (3000 psi)
- 1 megapascal (MPa) = 150 psi

Bonding agents—dentin

- Essential to follow manufacturer's directions
- Dentin histology is a major factor in bonding concepts
Dental tubule structure affects bonding surface
- Tubule diameter increases with depth toward pulp
- Bond strength decreases with progressive depth from DEJ smear layer
- About 1-5 microns thick; dentin chips, debris, partially denatured collagen
- Extends several microns into tubules
- Permeability increased by primers

Conditioners

- Remove or modify smear layer, increase permeability (acid etches smear layer)
- Demineralize underlying dentin
- EDTA, phosphoric acid, maleic acid, nitric acid

Primers

- Hydrophilic and hydrophobic wetting agents
- Provide micro-mechanical retention to modified smear layer and dentin

Third generation bonding agents

- Use a variety of approaches
- Bonding to dentin collagen, oxalate dentin bonding, hydrophilic acidic primer modifies smear layer
- Many use mechanical means of adhesion rather than unreliable chemical bonding of earlier generations

Fourth generation bonding agents

- Often referred to as two-bottle systems - separate primer and adhesive bottles
- Similar bond strengths for dentin and enamel
- Fifth generation bonding agents
- Often referred to as single bottle systems that contains primer and adhesive

Self-etching adhesives

- rinse and no rinse (remove or not remove smear layer)

VI. GLASS IONOMER CEMENTS

Properties: GI advantages

- Bond to dentin and enamel via chelation
- Leaches fluoride: may be reservoir of F, length of release varies
- Biologically compatible with connective tissue
- Thermal expansion similar to enamel and dentin
- Low setting shrinkage
- Bond strength of 10 MPa in “sandwich” technique
- Properties: GI disadvantages
- Prone to porosity from acid substances(e.g., fluoride gels)
- Surface finish is not as smooth as resin
- Surface wear is greater than resin

VLC GI/resin hybrid restorative materials

- Improved physical properties over conventional GI cements
- Less fluoride leaching than pure GI
- Less prone to hydration problems
- Resin -modified GIC self-cures with or without light exposure
- Polyacid modified resin-based composite must be light-cured

Atraumatic Restorative Treatment (ART)

http://www.aapd.org/media/Policies_Guidelines/P_ART.pdf

- To treat superficial cavities, dysplastic enamel (hypocalcification, hypoplasia), in young “precooperative” children where pharmacological management to manage behavior is either not available, not acceptable by parents, or is delayed because of scheduling or administrative barriers
- Caries (if present) are removed; usually with hand instruments; RMGI or GIC placed (as per manufacturer), smoothed with plastic instrument and light activated
- Topical fluoride varnish should be applied; a comprehensive preventive program initiated with frequent professional monitoring
- Replacement can be discussed at future date depending on child’s behavior, stability of restoration and preventive outcomes

The dental literature supports the use of glass ionomer cement systems in the following situations:

1. Luting cement:
 - stainless steel crowns
 - orthodontic bands
 - orthodontic brackets (limited).
2. Cavity base/liner
3. Class I restorations in primary teeth
4. Class II restorations in primary teeth
5. Class III restorations in primary teeth

6. Class III restorations in permanent teeth in high-risk patients or teeth that cannot be isolated
7. Class V restorations in primary teeth.
8. Class V restorations in permanent teeth in high-risk patients or teeth that cannot be isolated
9. caries control
10. high-risk patients
11. restoration repair
12. atraumatic restorative treatment

VII. CAVITY PREPARATION IN PRIMARY TEETH

Thinner enamel and dentin than permanent teeth

- Early diagnosis important
- Restore smaller carious lesions

Pulps are larger in relation to crown

- Restore early, small lesions
- Pulpal involvement can occur more quickly

Pulp horns are closer to DEJ (especially mesiofacial)

Enamel rods in gingival third extend in occlusal direction from DEJ

- No need to bevel gingival margin

Greater constriction of crown at CEJ, more prominent cervical constriction

- Excessive gingival extension can lead to loss of gingival floor of prep
- Attempt to recover “lost” gingival floor increases risk of pulp exposure
- Built-in retention for SSC
- Contour must be re-established for gingival health
- Broader, flatter contact areas
- Clinical diagnosis of proximal caries more difficult
- Less need for contouring matrix bands
- May not need to open proximal contact with prep
- Whiter in color
- Need lighter shades of composite materials
- Shallower pits and fissures
- Less need to carve extensive occlusal anatomy
- Dentin lighter in color
- Somewhat more difficult to visualize DEJ

Relatively narrower occlusal table

Common Errors in Class II Cavity Preparations for Primary Teeth

- Failure to extend occlusal outline
- Failure to follow outline of cusps
- Isthmus too wide

- Flare of proximal wall too great
- Axial line angles too large
- Gingival contact with adjacent tooth not broken
- Axial wall not conforming to the proximal contour, gingival floor too wide

VIII. MANAGING OCCLUSAL SURFACES OF YOUNG PERMANENT TEETH

Occlusal Surface	Clinical Findings	Treatment
Recommendations*		
Sound	Pits and fissures are smooth, broad, and coalesced No caries or restorations on other occlusal surfaces Deep pits and fissures Caries or restorations present on other occlusal surfaces	No treatment necessary Sealant
Questionable	Explorer tugback but no demineralization or opacities visible on pits or grooves	Sealant or PRR
Carious	Explorer tugback with opacities or demineralization visible in pits and fissures and/or Explorer sticks and supports its weight Radiographic evidence of occlusal caries in dentin	PRR if caries small, discrete Posterior composite or Resin modified glass ionomer (RMGI)

*Treatment recommendations are based on three assumptions: the proximal surfaces are sound, the tooth can be adequately isolated, and the tooth has been erupted less than four years.

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Chapter 9: TRAUMA

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_Trauma.pdf

- I. DIAGNOSTIC WORKUP
- II. SAMPLE TRAUMA NOTE (T)
- III. TRIAGE (T)
- IV. EXAMINATION (T)
- V. RADIOGRAPHS
- VI. FUNDAMENTAL ISSUES
- VII. TREATMENT ALGORITHMS
- VIII. COMPLICATIONS
- IX. SOFT TISSUE INJURIES
- X. ORAL ELECTRICAL BURNS (T)
- XI. ADDITIONAL READINGS AND WEB SITES

I. DIAGNOSTIC WORKUP

- Triage and stabilize (injuries often complex)
 - R/O CNS injury: Note C-spine precautions if no witness to injury
 - R/O child / domestic abuse
- Documentation
 - With regard to injury ask how ? where ? when ?
 - Immunization status, particularly tetanus
 - If last tetanus booster was 5 or more years prior and wound is contaminated with soil/debris another tetanus toxoid booster is indicated.
 - For sample trauma record form:

http://www.aapd.org/media/Policies_Guidelines/G_Trauma.pdf

- Clinical photos to establish initial presentation (if practical - DO NOT delay time-sensitive treatments)

II. SAMPLE TRAUMA NOTE

Chief Complaint: (In patients (or parents) own words)
Med HX: (significant items such as bleeding disorders/seizures)
Allergies / Immunizations: (note tetanus status)
History Present Injury: (Be as detailed and specific as possible)
Time of injury: Time seen by dentist:
Extra-oral examination (note thorough head and neck exam warranted):
Intraoral examination:
Radiographs:
Diagnosis (DX):
Treatment (TX):
Medications prescribed (RX):
Follow up plan:

III. TRIAGE

- Consciousness level - Loss of consciousness, amnesia, nausea, vomiting or seizures may signal intracranial injury in children who suffered a mild head injury.
Relevant considerations/questions include;
 - Age appropriate responsiveness?
 - Patient recollection of injury; accurate identification of family members (if applicable and feasible)
 - History of nausea or vomiting?
 - Modified Glasgow Coma Scale
- Subjective comments (abuse suspicion, domestic abuse)
Historian reliability

GLASGOW COMA SCALE	
FINDING	RATING
A. Eyes	
Open spontaneously	4
Open to verbal command	3
Open to pain	2
No response	1
B. Best Motor Response	
Obeys verbal command	6
Realizes pain	5
Withdraws from pain	4
Flexion to pain (decorticate)	3
Extension to pain (decerebrate)	2
No response	1
C. Best Verbal Response	
Oriented and converses	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Minimal score=3	
Maximal score=15	

IV. EXAMINATION

- Thorough head and neck examination to R/O extra-oral injury intra & extra-oral soft tissue lacerations
- Fractured, displaced and missing teeth
- Pulp exposure
- Bony fractures (obtain Oral and Maxillofacial surgery consult)
 - Mandibular Fractures
 - Note force direction and impact point (example: chin impact = increased likelihood of condylar fracture)
 - Vertical laceration on mandibular mucosa may signal fracture
 - Signs include; change in function/occlusion, steps in continuity upon palpation of mandibular border, pain on mastication, sublingual hematoma, midline asymmetry
 - Treat quickly—healing can occur in 4-6 days
 - Fixation or conservative treatment
 - Subcondylar fractures can result in growth disturbance
 - Body fractures may need fixation
 - Panoramic, R and L oblique, A-P, Towne's views may be indicated in midface fractures
 - Zygomatic—“Tripod”—rare in children
 - Blow out—orbital floor (Water's view=cloudy antrum, enophthalmos, periorbital edema/hematoma)
 - LeForte I—maxillary separation from midface—maxilla mobile

- LeForte II—Nasomaxillary fracture
- LeForte III—Cranial base - facial separation; airway edema
- CT scan indicated for LeForte fractures
- Battles sign (mastoid hematoma=posterior cranial bone fracture)
- Raccoon sign (orbital hematoma=anterior cranial bone fracture)
- Skull fracture—CSF rhinorrhea, otorrhea (CSF less viscous than mucus)

Cranial Nerve	Examination
I Olfactory	Test sense of smell with aromatics
II Optic	Check visual acuity and light/dark
III Oculomotor:	Pupil reaction to light/ptosis
IV Trochlear:	Check eye movement
V Trigeminal:	Check muscles of mastication
VI Abducens:	Check range of movement of eyes
VII Facial	Check facial muscles and taste
VIII Auditory	Check hearing (Weber, Rinne tests)
IX Glossopharyngeal	Gag reflex
X Vagus	Check palatal function
XI Accessory	Check sternocleidomastoid, trapezius function
XII Hypoglossal	Check tongue function

V. RADIOGRAPHS

- When possible, take multiple radiographs to aid in accuracy of diagnosis
- Radiographic interpretation and documentation should include; pulp size, apical maturity, PDL space, periapical pathology, alveolar fractures, foreign bodies, developmental anomalies
- Locating foreign bodies (soft tissue radiographs: $\frac{1}{4}$ exposure time)
- Locating intruded teeth - use lateral film showing nasal area (double exposure time)
- Buccal object rule: The image of any buccally-oriented object appears to move opposite the direction from a moving x-ray source; lingual object moves in same direction as source (also SLOB: same-lingual, opposite-buccal)

VI. FUNDAMENTAL ISSUES

Primary Teeth

- Keep it simple due to behavior, lifespan of tooth
- Assess full risk of treatment and potential sequelae to permanent tooth versus functional benefit
- Advise parent of permanent tooth injury possibility

Permanent Teeth:

- Luxation injuries, particularly avulsions, dictate emergency treatment as positive outcomes diminish with time delay

VII. TREATMENT ALGORITHMS

TRAUMA TREATMENT ALGORITHM LEGEND AND ABBREVIATIONS



= Treatment decision supported by strong evidence



= Treatment decision supported by weak evidence or evidence that is conflicting



= Treatment which is time-dependant, in that delay of treatment can affect overall prognosis / outcome. **Do not delay treatment.**

Abbreviations

CaOH = Calcium Hydroxide

CHX = Chlorhexidine 0.12%

HBSS = Hanks Balanced Salt solution

NaF = Sodium Fluoride

RCT = Root canal therapy

FIGURE 1: CROWN FRACTURES - TREATMENT RECOMMENDATIONS

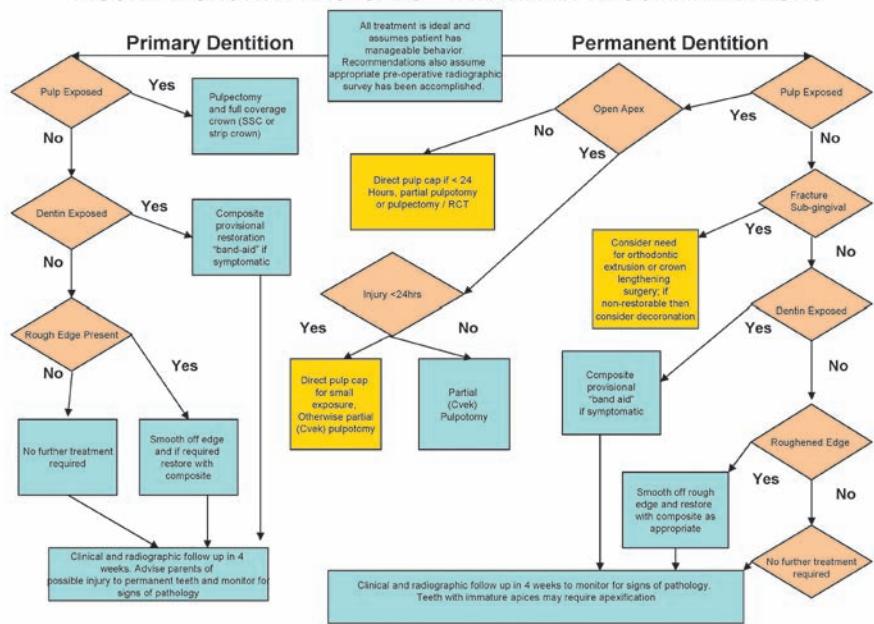


FIGURE 2: ROOT FRACTURES - TREATMENT RECOMMENDATIONS

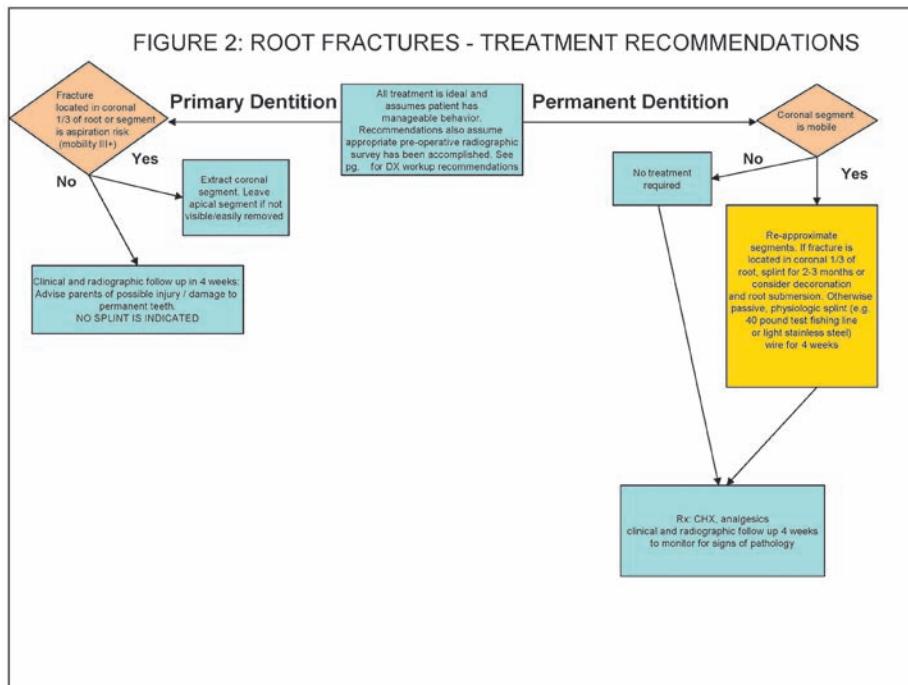
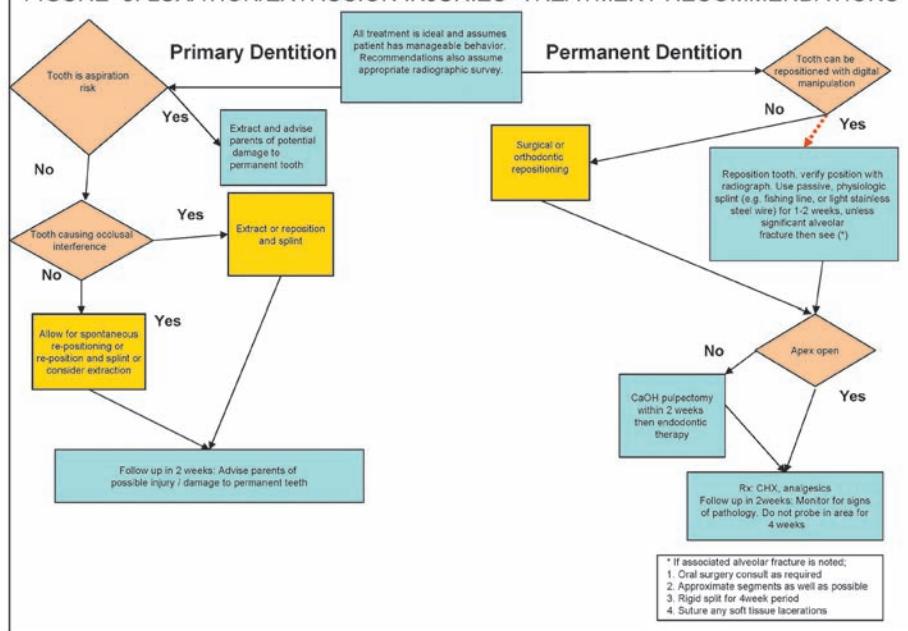
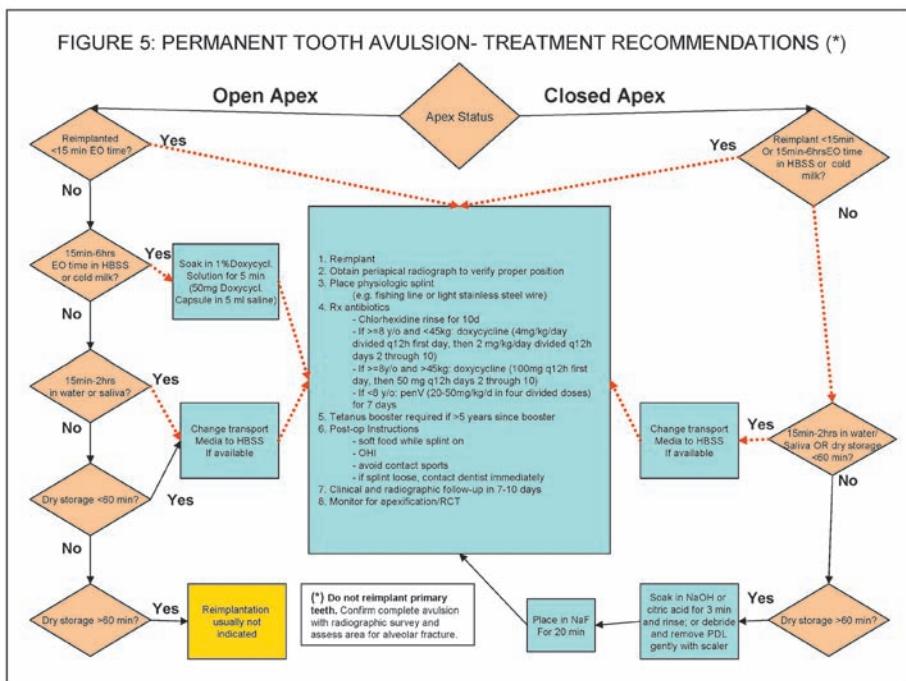
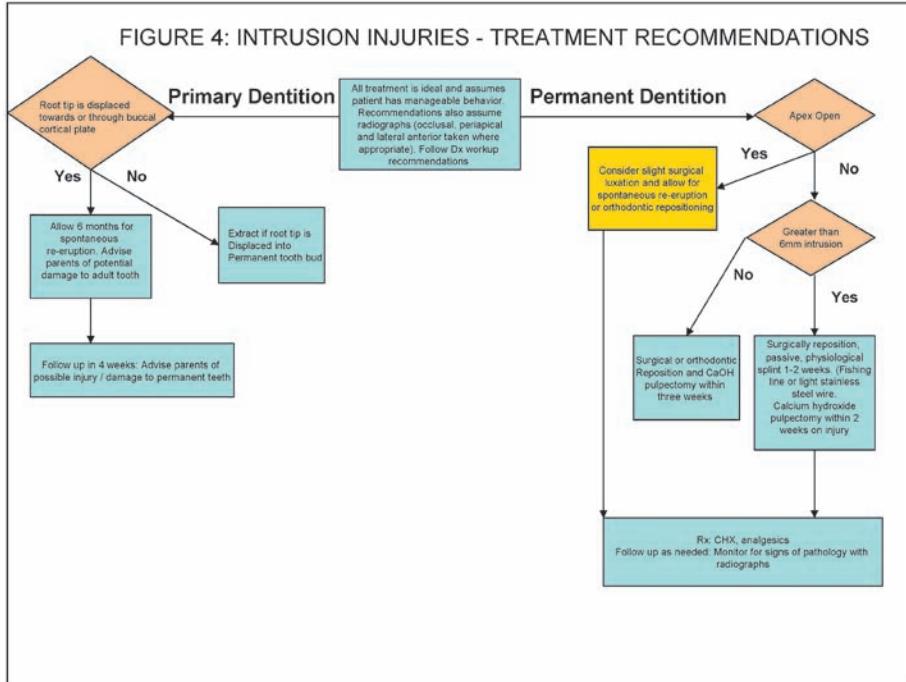


FIGURE 3: LUXATION/EXTRUSION INJURIES- TREATMENT RECOMMENDATIONS





VIII. COMPLICATIONS

- Color changes—gray/brown usually indicate pulpal necrosis/deposition of pigments. Treatment may not be indicated in primary teeth if no other signs or symptoms of pathology in a healthy child. Root canal treatment indicated in permanent teeth.
- Pulp canal obliteration—closure of canals with reparative dentin; formerly known as calcific metamorphosis—common in immature teeth
- Pulp necrosis
- Damage to developing teeth
- Ankylosis (replacement resorption)
- Resorption—surface, replacement, inflammatory

IX. SOFT TISSUE INJURIES

- Tongue laceration—suturing indicated if bleeding is not controlled
- Through and through punctures—uture both sides after debridement
- Intra/extr-oral laceration across vermillion—uture beginning with extra-oral
- Thoroughly debride gravel, other foreign bodies

X. ORAL ELECTRICAL BURNS

- Very deep burn—2000 C; often painless due to burn
- Eschar sloughs 7-10 days/bleeding from facial artery possible
- Use fixed appliance to stop contracture of wound; tooth retained with “tusks” at commissures
- Appliance worn 6-12 months
- Plastic surgery in future will often be necessary
- Wound care with appliance may involve topical antibiotic
- May need sedation/GA for impression or use extraoral device in very young child

Perioral Dimensions 12, 24, 36 Months of Age (mm)

AGE	CLOSED MOUTH BREADTH	OPEN MOUTH BREADTH	INTER- INCISAL
	(Inter-commissure)	(Inter-commissure)	
12	43.4	35.7	35.5
24	46.0	37.3	36.1
36	43.5	36.9	35.7

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Chapter 10: GROWTH AND DEVELOPMENT/MANAGEMENT OF THE DEVELOPING OCCLUSION

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_DevelopDentition.pdf

- I. BASICS OF CRANIOFACIAL GROWTH (T)**
- II. CLINICAL EVALUATION OF THE PRIMARY DENTITION**
- III. MANAGEMENT OF THE PRIMARY DENTITION**
 - SPACE MAINTENANCE
 - POSTERIOR CROSSBITE
 - ANTERIOR CROSSBITE
 - NON-NUTRITIVE SUCKING HABITS (NNS)
 - AIRWAY COMPROMISE/MOUTHBREATHING
- IV. CLINICAL EVALUATION OF THE MIXED DENTITION**

V. MANAGEMENT OF THE MIXED DENTITION

OVERVIEW OF SPACE SUPERVISION/GUIDANCE OF ERUPTION

SPACE MAINTENANCE

REGAINING LOST POSTERIOR SPACE

MANDIBULAR INCISOR CROWDING/ARCH LENGTH DISCREPANCY

ECTOPIC ERUPTION OF FIRST PERMANENT MOLARS

DENTAL/FUNCTIONAL ANTERIOR CROSSBITE

ANTERIOR OPENBITE WITH EXTRAORAL HABIT

POSTERIOR CROSSBITE

MAXILLARY CANINE ERUPTIVE DISPLACEMENT

CONGENITALLY MISSING PERMANENT TEETH

ANKYLOSED TEETH

SUPERNUMERARY TEETH

VI. TREATING SKELETAL MALOCCLUSIONS IN THE MIXED DENTITION

OVERVIEW

TRANSVERSE BASAL ARCH EXPANSION

ANTEROPOSTERIOR CLASS II

MALOCCLUSION>RETRUSIVE MANDIBLE>FUNCTIONAL APPLIANCE

ANTEROPOSTERIOR CLASS II

MALOCCLUSION>PROTRUSIVE MANDIBLE>DIRECTED HEADGEAR

ANTEROPOSTERIOR CLASS II MALOCCLUSION WITH ACCEPTABLE A-P SKELETAL/PROFILE RELATIONSHIPS

ANTEROPOSTERIOR CLASS III MALOCCLUSION

VII. ADDITIONAL READINGS

I. BASICS OF CRANIOFACIAL GROWTH

Types Of Bone Formation (Bone apposition generally occurs in osteogenic areas under tension, *not pressure*)

- Intramembranous bone formation
 - Bone formed at periosteal and sutural surfaces, facial bones (maxilla, body of mandible)
 - More modifiable in context of dentofacial orthopedics/orthodontics
- Endochondral bone formation
 - Cartilaginous precursors > cranial base and condyle of mandible
 - Less modifiable in context of dentofacial orthopedics

Growth of Facial Components

- Cranial Vault
 - Intramembranous bone formation occurring primarily at periosteum-lined contact areas, sutures
 - Remodeling occurs on inner and outer surfaces of bone to allow for expanding neurocranium
- Cranial Base
 - Endochondral bone formation
 - Synchondroses play role in early growth - *Spheno-occipital* considered principal growth cartilage of cranial base and only one remaining active during childhood growth period
- Maxilla (nasomaxillary complex)
 - Intramembranous bone formation
 - Growth occurs through balanced apposition and resorption (*remodeling-cortical drift displacement*)
 - Appositional growth predominates up and back against cranial base with growth expression projected downward and forward “from under the cranial base”
- Mandible
 - Endochondral at condyle, intra-membranous for body of mandible
 - Condyle: fibrocartilage grows by apposition (similar to epiphyseal growth plates of long bones)
 - Appositional growth predominates along posterior border of ramus with remodeling resorption along anterior border
 - Up and back growth emphasis with expression downward and forward “from under the cranial base”

Facial Growth Patterns

- Facial growth generally corresponds to somatic growth patterns
 - Females reach earlier skeletal maturity than males by about two years of age on average
 - On average, female growth spurt starts at approximately 10.5 to 11 years of age, peaks in 14 to 18 months (about 12-13 years of age), and is complete by about 13.5 - 14 years of age
 - On average, male growth spurt starts at approximately 12.5 to 13.5 years of age, peaks in 18 to 24 months (about 14 to 16 years, and is complete by about 17 to 18 years of age

- Facial form and growth patterns are maintained throughout the growth years
 - Hypodivergent / brachyfacial*: Posterior face height proportionately greater than anterior face height with counter-clockwise rotation expressed as flat mandibular plane and pronounced overbite (deep bite)
 - Hyperdivergent / dolichofacial*: Anterior vertical facial growth greater than posterior condylar growth with clockwise rotation expressed as steep mandibular plane with open bite tendency
- Dimensional Craniofacial Growth
 - Facial Height (nasion-menton): 70% complete by age 3 years, 90% prior to adolescent growth spurt
 - Width: shows *least* amount of change of any facial dimension - upper face width (bizygomatic width) increases throughout childhood and adolescence with greatest rate observed between ages 2-6 years. Lower face width (bigonial width) is 85% complete by the time first molars erupt
 - Depth (Anteroposterior): Longest growing facial dimension. May be divided into upper, middle, and lower facial dimension with areas growing at different times and rates (differential growth). Greater mandibular increments allow profile to change from convex in childhood to straighter adult profile

Facial Analysis with Lateral Cephalometrics

- Lateral Cephalograms - Examples of Common Diagnostic References *
- Maxilla to cranium: SNA, A-point to Nasion perpendicular, Maxillary length (Co-A)
- Mandible to cranium: SNB, Pogonion to Nasion perpendicular, Mandibular length (Co-Gn)
- Maxilla to mandible: ANB, Mx-Md length difference, Wit's analysis (AO-BO),
- Denture to denture: Interincisal angle, Wit's, overjet/overbite
- Incisor position: Upper incisor to cranial base (Franfort horizontal, S-N), Lower incisor to mandibular plane (IMPA), Upper and lower incisors to facial lines (NA, NB)
- Growth direction: Mandibular plane angle (FMA), y-axis to cranial base, lower face height
- Soft Tissue Profile: Angle of facial convexity, Lip profile to E-line, nasolabial fullness

*Values from composite assessments of longitudinal growth studies involving male and female Caucasians. Adult values represented in females at 14 years and males at 18 years of age; from Bishara, Am J Ortho, Jan 1981

MAXILLA	9 y.o. ($x \pm sd$)	Adult ($x \pm sd$)
SNA (°)	81 ± 3	82 ± 2
A-Na Perpendicular (mm)	0 ± 2	1 ± 2
Mx. length: Co-A (mm)	(m) 87 ± 6 (f) 85 ± 6	(m) 99 ± 6 (f) 91 ± 4

MANDIBLE

SNB (°)	76 ± 2	80 ± 2
Pg-Na Perpendicular (mm)	-6 ± 3	-2 ± 2
Md. length: Co-Gn (mm)	(m) 117 ± 6 (f) 105 ± 6	(m) 134 ± 6 (f) 120 ± 6

BASAL RELATIONSHIP: MAXILLA TO MANDIBLE

ANB (°)	4 ± 2	2 ± 2
Mx-Md Difference (mm)	(m) 20 ± 6 (f) 20 ± 6	(m) 35 ± 6 (f) 29 ± 3
AO-BO (mm)	—	(m) -1 (f) 0

FACIAL TYPE/GROWTH PATTERN

FMA (°)	29 ± 5	26 ± 4
Y-axis (°)	67 ± 3	67 ± 3
LFH (°)	47 ± 4	47 ± 4

MAXILLARY DENTITION

1 to SN (°)	105 ± 6	104 ± 6
1 to FH (°)	111 ± 6	111 ± 6
1 to NA (mm. / °)	4 ± 2 / 24 ± 5	4 ± 2 / 22 ± 4

MANDIBULAR DENTITION

IMPA (°)	95 ± 5	93 ± 5
FMIA (°)	56 ± 7	58 ± 7
1to NB (mm / °)	4 ± 2 / 26 ± 5	4 ± 2 / 25 ± 4

MAXILLARY TO MANDIBULAR DENTITION

1 to 1(°)	126 ± 9	130 ± 6
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FACIAL/SOFT TISSUE PROFILE

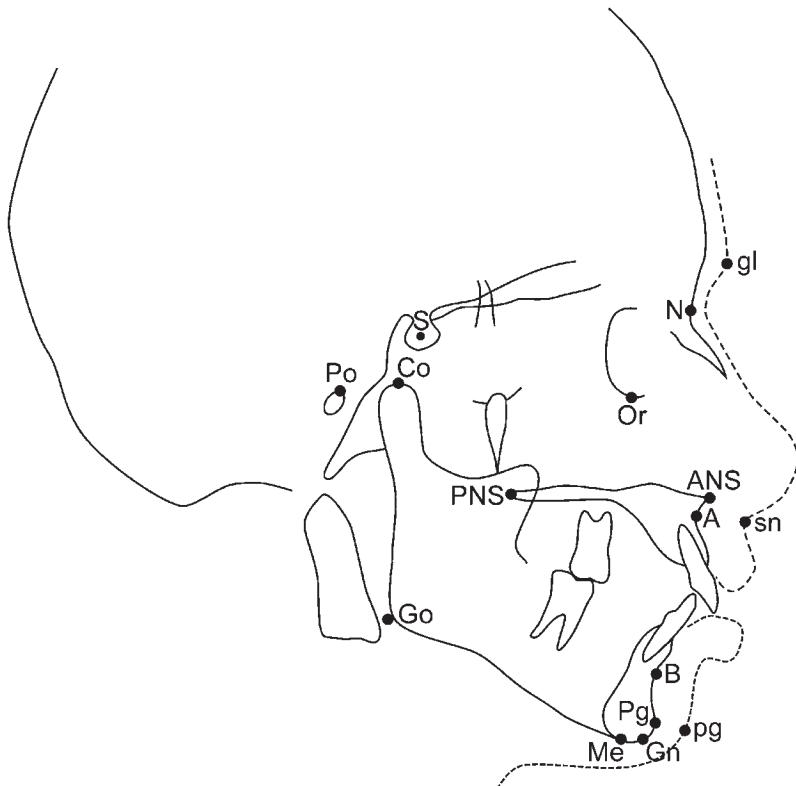
Upper lip-E line (mm)	-1 ± 2	-4 ± 2
Lower lip-E line (mm)	0 ± 2	-2 ± 2
Facial convexity gl-sn-pg(°)	169 ± 4	172 ± 6

Legend for Ceph Landmarks Illustration:

- A- A Point
- ANS- Anterior Nasal Spine
- B- B Point
- Co- Condylion
- Gn- Gnathion
- Go- Gonion
- Mc- Menton
- N- Nasal
- Or- Orbitale
- Pg- Pogonion
- PNS- Posterior Nasal Spine
- Po- Porion
- S- Sella turcica
- gl- glabella
- sn- subnasale
- pg- soft tissue pogonion



- Qualitative Cephalometrics - Eyeballing a Cephalogram
 - Head orientation: Frankfort horizontal plane should be perpendicular to vertical edge of film
 - Chin position: Nasion to Pogonion line (Facial plane) should be parallel with vertical edge of film after growth is complete. In school-age children, positioning of chin point about 4 to 6 mm. behind facial plane allows for mandibular growth differential relative to cranial base.
 - Maxilla position: N-A line should be parallel with vertical edge of film. A-point approximates facial plane at all ages - if forward suggests maxillary prognathism, if behind retrognathic position
 - Mandibular plane: Go-Gn should intersect with cranial outline at occiput. If plane “flat” extending below cranial outline into neck area, suggests brachyfacial horizontal grower. If plane angled above cranial outline towards earhole, dolichofacial vertical growth pattern expressed.
 - Maxillary incisor position: Long axis should be tangent with orbitale
 - Mandibular incisor position: Long axis should show proclination approaching 100 degrees to the mandibular plane in school-age child. Long axis should upright to slightly above a right angle after growth is complete (mean of 93 degrees)
 - Facial height: Upper face height (N-ANS) should be about equal to lower face height (ANS-Me)



II. CLINICAL EVALUATION OF THE PRIMARY DENTITION

Eruption Timing and Sequencing

- Primary teeth erupt on average from 8 months (Lower central incisors) to 30 months (Upper second primary molars) of age with a S.D. of 3 months. Sequence in both arches as follows: A - B - D - C - E
- Primary dentition occlusal relationships established by 36 months of age with minimal subsequent dimensional changes (length, width, perimeter) occurring until permanent dentition eruption

Primary Dentition Occlusion

- Spaced vs. non-spaced arches: Approximately 2/3rds of primary dentitions exhibit generalized spacing (Baume Type I) while 1/3rd are non-spaced (Baume Type II). Primate spaces mesial to upper primary canines and distal to lower primary canines commonly occur in both Baume type archforms.
 - Once established, arches remain spaced or non-spaced over course of primary dentition
 - Spaced vs. non-spaced arches related to basal arch size rather than tooth mass differences

- Primary spacing affects crowding outcome predictors into the mixed dentition:
 - spacing 3 to 6 mm. > no transitional crowding
 - spacing less than 3mm. > 20% with incisor crowding
 - no spacing > 50% with incisor crowding
 - crowded primary teeth > 100% with incisor crowding
- Molar Terminal Plane Relationships
 - Mesial step (approximately 15% incidence) - usually results in Cl I permanent molar relationship
 - Flush terminal plane (approximately 75% incidence) - majority “shift” to Cl I permanent molar relationship; but significant number stay end-on or “shift” to full Cl II permanent molars
 - Distal step—usually results in full Cl II, some shift to end-on Cl II molars
- Canine Relationships: Best predictor of sagittal relationship into the permanent dentition
 - Mesial step canines - usually results in Cl I relationship
 - Distal step/End-on canines - usually results in Cl II permanent dentition
 - Excessive mesial step (with incisor crossbite) - usually results in Cl III permanent dentition
- Incisor Relationships
 - Overbite: 2 mm (30 to 50% vertical overlap)
 - Overjet: 0-3 mm
- Ideal Primary Dentition Occlusion
 - Flush terminal plane or mesial step molar with Class I canines
 - Generalized spacing including primate spaces
 - 2 mm. overjet and 2 mm. overbite (30%)

III. MANAGEMENT OF THE PRIMARY DENTITION

Premature Loss of Primary Teeth - Space Maintenance

- Primary Incisors: Space loss unlikely if primary canines erupted into occlusion. Replacement of primary incisors (e.g. “Hollywood” bridge) is elective for cosmetic issues, not space control.
- Primary Canines: Usually due to ectopic eruption of permanent laterals as indicator of significant tooth mass discrepancy - beyond simple space maintenance. If loss of canine secondary to caries or trauma, no space maintenance generally indicated except to maintain midline symmetry.
- First primary molars: Space maintenance indicated if first permanent molars not erupted or in active eruption.
 - Usually unilateral fixed appliances from second primary molars to primary canines are recommended (e.g. crown or band and loop/arm).
 - Once first permanent molars erupted into occlusion, space loss negligible if first primary molars lost during mixed dentition and second primary molars remain to buttress first permanent molar position.
- Second primary molars: Regardless of timing of loss, space maintenance generally indicated as space loss will occur in primary or mixed dentition. Dimensional loss greater in maxillary arch - without second primary molars the maxillary first molars move forward bodily and rotate around palatal root, while lower molars

evidence mesial and lingual crown tipping. Space loss in either arch most dramatic in association with eruptive timing of first permanent molars.

- If 6-year molar not erupted or in active eruption, distal shoe from first primary molar to guide first molar eruptive positioning is generally appliance of choice.
- Once 6-year molars erupted, bilateral space maintainers advised to replace distal shoe and control molars while allowing buccal transition due to eruption patterns and potential loss of abutments
- Mandibular arch: Lingual holding arch once lower incisors erupted
- Maxillary arch: Transpalatal arch or Nance appliance

Posterior Crossbites in the Primary Dentition

- Functional Posterior Crossbite: Shift of mandible on closure results in appearance of unilateral crossbites in maximum intercuspsation. Greater than 90% of primary dentition posterior crossbites express a functional shift in the occlusion pattern.
 - Crossbite involves entire buccal segment (90+ % C-D-E segment, 2/3rds include primary lateral in pattern) with lower midline approximating a 2 mm discrepancy to the crossbite side on average
 - Shift of condyles on closure with crossbite side rotating around condylar axis and non-crossbite side projecting down and forward results in asymmetric chin positioning and Class II subdivision molar positioning (crossbite side more Class II/distal step, non-crossbite side Class I-III/mesial step)
 - Constricted maxilla, particularly intercanine width; is probable factor as first contact position with coincident midlines exhibits typical transverse end-on buccal segment cusp-to-cusp occlusion. May be associated with vertically oriented primary canine interferences, digit habit or mouthbreathing patterns.
 - Associated with asymmetric growth patterns as crossbite side exhibits shorter mandibular length and more Class II buccal relationships when untreated or when treatment delayed late into the growth years. TMD issues also suggested when lateral mandibular shifts expressed in the permanent dentition.
 - Maxillary expansion: Basic treatment to correct transverse discrepancy and eliminate functional shift. Appliances reported with success treatment rates include:
 - Fixed rapid palatal expanders (RPE of Haas, Hyrax) - over 90% success
 - Fixed archwire expanders using “slow, low-force” approach (w-arch, quad helix) - over 90%
 - Removable Schwartz Plate-type appliances - approximately 70% success
- Bilateral Posterior Crossbite: Represent true maxillary skeletal constriction with bilateral buccal segment crossbite, midline symmetry, and no notable shift of mandible
 - Reported at 2 to 3% of posterior crossbites in children, often associated with dolichofacial skeletal vertical growth, openbite malocclusion, compromised airways, and mouthbreathing patterns
 - Maxillary expansion with emphasis toward sutural separation indicated for significant transverse discrepancy (e.g. fixed palatal expanders - RPE of Haas, Hyrax). Given etiological factors with skeletal growth and airway issues, while early expansion desirable; long-term management generally requires a multiphasized comprehensive treatment plan

Anterior Crossbites in the Primary Dentition

- True Class III versus Pseudo-Class III: Anterior crossbites typically involve full incisor segment as opposed to individual teeth. Must distinguish between a pseudo-Class III and a true Class III
 - Pseudo-Class III: Incisal and canine interference produces anterior shift of the mandible on closure
 - Treatment directed at advancement of incisor segment to eliminate interference.
 - Fixed or removable maxillary appliances with finger or sweep springs to advance incisors
 - True Class III: In primary dentition presents classic skeletal and dental patterns with retruded maxilla, prognathic mandible, “adult” concave profile, retroclined lower incisors
 - Treatment directed at dentofacial orthopedic changes to correct skeletal malocclusion
 - Reverse pull headgear/facemask, chinup.
 - Both pseudo- and true Class III may require concurrent maxillary expansion (See posterior crossbite)

Non-nutritive Digit Sucking Habits (NNS)

- Normal at early age: 50% of children with NNS habit will discontinue between 24-28 months of age
 - Digit habits can last longer than pacifier habits; both produce similar affects if persist past 4 years
 - Incidence rate of 10 - 15% at age five years
- May result in anterior openbite, distorted incisor eruption, increased overjet, proclined upper incisors, linguoversion of lower incisors, posterior crossbite with constricted maxilla, possibly Class II relations
- Consider intervention prior to eruption of permanent anterior teeth approximating age 5 to 6 years if NNS habit persists and patient-parent indicate understanding of need to stop
 - Use “gentle persuasion” as beginning treatment; behavior modification can be successful
 - Cribs, rakes, “bluegrass appliance” are choices for fixed therapy to “help” child quit

Airway Compromise/Mouthbreathing

- May impact on facial growth with tendency to increase vertical orientation (skeletal openbite)
 - Similar occlusion changes as seen with deleterious extraoral habits
 - Management - Distinguish from extraoral habits. If determined to be airway directed, refer for ENT assessment for possible allergy management, tonsillectomy/adenoideectomy, palatal expansion

IV. CLINICAL EVALUATION OF THE MIXED DENTITION

Eruption Timing and Sequencing

- Permanent teeth erupt on average beginning at 6 years of age (lower central incisors, upper and lower first molars) and is complete by 12 years of age
 - Most common eruption sequence is 6-1-2-3-4-5-7 in the lower and 6-1-2-4-5-3-7 in the upper arch
 - Above sequences occur only about 50% of the time
 - Most common variation is eruption of second molars in either arch before more anterior teeth
 - Incisor transition complete by 8 years to establish the mid-mixed dentition of permanent molars and incisors along with buccal segment primary teeth (C-D-E)
 - Buccal segments undergo transition with eruption in the lower arch of the canines around 10 years of age, eruption of upper and lower first premolars approximating age 11-11.5 years, eruption of upper and lower second premolars at age 11.5-12 years and eruption of upper canines at age 12+ years
 - Second molars erupt about 12 years of age approximating lower second premolars and upper canines

Normal Mixed Dentition Occlusion and Alignment

- After incisor transition in the early mixed dentition years, the incisors normally exhibit:
 - On average 1 to 2 mm. of lower incisor “crowding” with S.D. of ± 1 mm. (i.e. “crowding” normative)
 - On average no spacing or crowding in upper incisor segment (S.D. of ± 1 mm.). “Ugly duckling” stage is normal transitional appearance with “splayed” maxillary incisors under influence of eruptive positions of adjacent incisors and canines
 - Overjet: Ideal is no overjet with incisal contact, range is 0-3 mm
 - Overbite: Ideal is about 2 mm. or 30-50% overlap, two S.D. range is 0-5 mm
- Incisor Liability
 - Permanent incisors larger than primary incisors: - 7.1 mm. in the upper and - 5.1 mm. in lower
 - Transition from primary incisors to permanent incisors possible because:
 - Interdental spacing of primary teeth if available (Baume Type I, primate spaces)
 - Increase in intercanine arch width during “growth” transition
 - Lower width increase mean of 2.4 mm. with range of 0 to 5 mm
 - Upper width increase mean of 3.0 mm. with range of 0 to 6.5 mm
 - More anterior placement of permanent incisors increasing forward arch perimeter
- Permanent Molar Relationships In the Mixed Dentition
 - Class I: Maxillary first molar mesial cusp in mandibular molar buccal groove - “Ideal”
 - End-on Class II: Majority of mixed dentition occlusions. Corrects to Class I with late mesial shift in late transition stages in most cases; but may stay end-on or even shift to full Class II
 - Full Class II: Maxillary first molar mesial cusp forward in embrasure between lower first molar and second primary molar or second premolar

- About 15% of children express full Class II molar relationship, usually as a reflection of a skeletal malocclusion involving mandibular retrognathia as the most common causative factor
- Canines also demonstrate Class II positioning with pronounced overjet of 6 mm, or more
- Cl. III: Maxillary first molar mesial cusp distal to lower first molar buccal groove
 - About 1-3% of Caucasian children, usually reflecting Class III skeletal malocclusion with mandibular prognathia and maxillary retrognathia as common causative factors
 - Discriminate from pseudo-Class III with forward shift of mandible to exaggerate discrepancy
- Permanent molar transition positioning influenced by:
 - Primary molar terminal plane relationship (See primary dentition occlusion)
 - Spacing—primary spacing may be closed by “early mesial shift” as first molars erupt
 - Size differential between primary C-D-E segment and permanent 3-4-5 segment teeth (i.e. leeway space) allows “late mesial shift” of first molars when second primary molar exfoliates and permanent first molar moves mesial, often under influence of erupting second molar
 - On average, upper leeway space is +0.9 mm. per quadrant
 - On average, lower leeway space is +1.7 mm. per quadrant
 - Mandibular growth and differential growth may affect relative A-P positioning

V. MANAGEMENT OF THE MIXED DENTITION

Overview of Space Supervision/Guidance of Eruption Concepts *

- Space supervision encompasses treatment procedures derived from clinical judgment where the clinician determines that a patient's occlusion will have a better chance of obtaining optimum development with supervised intervention than without intervention (Moyers)
- Preventive Orthodontics: To preserve and maintain normal relationships in the developing occlusion through prevention of oral disease, restorative care, and space maintenance
- Guidance of Eruption: Procedures that influence eruptive patterns of permanent teeth during transition from the primary through the mixed dentition. Applicable when overall space is adequate to accommodate a normal complement of permanent teeth with acceptable esthetics and function (Hotz)
- Interceptive Orthodontics: Recognition of developing malocclusion factors and implementation of treatment procedures to eliminate or minimize their effects on the final occlusion

* Estimates by credentialed specialists indicate approximately 50% of children would benefit from guidance and interceptive procedures beyond “preventive” interventions

- Goals Of Space Supervision/Guidance of Eruption in the Mixed Dentition
 - Improved esthetics/Incisor integrity: Satisfactory alignment of anterior teeth without significant midline discrepancy, excessive protrusion, lingual malpositioning, openbite or excessive deepbite

- Dentitional development without functional problems: Elimination of functional posterior and anterior crossbites, deleterious oral habits and temporomandibular dysfunction (TMD)
- Optimal permanent tooth eruption: Correction of eruption anomalies such as ectopic molar and canine eruption patterns, over-retained primary teeth, delayed eruption of permanent teeth, ankylosis and supernumerary teeth
- Avoid unnecessary extraction of permanent teeth: Optimal use of leeway space and arch perimeter with symmetrical molar positioned without symptomatic space loss
- Clinical Procedures in Space Supervision and Guidance of Eruption
 - Preventive and Restorative Dentistry: To preserve arch integrity and overall arch perimeter
 - Space Maintenance: Stabilization of molar and anterior tooth positions to preserve arch dimensions and prevent symptomatic loss of arch length secondary to premature loss of primary teeth
 - Disking of Primary Teeth: Reduction of mesiodistal primary tooth structure to enhance adjacent permanent tooth alignment through timely use of leeway space
 - Selective Extraction of Primary Teeth - Extension of disking concepts in the timely removal of primary teeth to enhance permanent tooth eruption and alignment positioning
 - Minor Tooth movements: Biomechanical tooth movements to return or direct developing occlusion to a “normal”, not necessarily “ideal” pattern of development. Implies minimal or simple appliance therapy over a short interval of treatment time. Conditions considered for minor tooth movement procedures within the context of interceptive orthodontic include:
 - Ectopic eruption of permanent first molars
 - Dental/Functional Anterior Crossbites
 - Posterior Crossbites
 - Oral Habits (Thumb/Digit Sucking)
 - Recognition and Correction of Dental Anomalies: Identification and elimination of anomalies and their effects on the developing occlusion to include supernumerary teeth, missing teeth, tooth size/shape anomalies, ankylosis, pathologic lesions, etc.

Space Maintenance

- Use when space loss likely, particularly in association with early loss of primary second molars (See primary dentition space maintenance)
- Work-up for space maintenance includes space analysis, presence and eruptive status of permanent teeth, molar intercuspal relationships, incisor positioning, and understanding/cooperation
- Once 6year molars erupted, bilateral space maintainers advised to control molars and allow buccal segment transition due to eruption patterns and potential loss of abutments
 - Mandibular arch: Lingual holding arch once lower incisors erupted.
 - Maxillary arch: Transpalatal arch or Nance appliance

Regaining of Lost Posterior Space

- Indicated if it simplifies, minimizes, or eliminates subsequent orthodontic treatment

- Maxillary regaining: Headgear, fixed molar “distalizing” appliances, removable appliance
- Mandible regaining: lip bumper, “active” lingual arch, removable split-saddle

Mandibular Incisor Crowding—Arch Length Discrepancies

- Lower anterior crowding considered normal as the average crowding is -1.6 ± 1.0 mm. after incisor eruption is complete. This means the vast majority of children express 0 to 4 mm. of crowding at 8 to 9 years of age and importantly, there are no future arch dimensional changes to compensate for any degree of crowding and malalignment in the lower anterior segment
 - Approach during incisor transition is to allow “wedging” effect of eruption to optimize width
 - After lateral incisor eruption, what you see is what you get
 - Arch length analysis: Can crowding be accommodated by controlled use of leeway space? If so, intervene. If excessive beyond 3 to 4 mm. of lower crowding, plan long-term arch development approach versus serial extraction. Considerations include:
 - Periodontium-thin labial gingiva or mucogingival defect
 - Profile and incisor position/angulation - incisors “most” stable where they are found
 - Vertical relationships: Non-extraction therapy opens bite; extraction therapy deepens the bite, so open bite tendency with hyperdivergent facial pattern directed toward serial extraction protocols, deepbite brachyfacial patterns directed toward non-extraction with arch expansion
 - Premature exfoliation of primary teeth (lateral incisors) and/or excessive resorption of primary canines with ectopic eruption of permanent lateral incisors
- Guidance interventions involve unraveling incisor crowding toward posterior “leeway space”
 - Disking of primary canines >>> 1 to 2 mm. of space per side can be achieved by diskng the mesiolingual corner to provide “sluice-way” for incisor alignment. Disking must go subgingival to free contacts while being careful with adjacent permanent laterals. Indicated with:
 - Less than 3 to 4 mm. of incisor crowding
 - Laterals actively erupting to alignment in the arch with eruptive width changes complete
 - Intact primary canine roots (Not ectopically resorbed or normal timing of exfoliation)
 - Incisors lingually malpositioned
 - Preferred option, especially in deepbite patterns to maintain vertical support
 - Extraction of primary canines >>> To enhance arch symmetry, coincident midlines and incisor integrity when incisor liability greater than 4 mm. with distorted incisor positioning
 - Recommended with asymmetric ectopic loss of primary canine producing midline shift - extraction of contralateral canine to sustain symmetry
 - Frequently Step One of a serial extraction program, particularly in vertically sensitive dolichofacial openbite patterns
 - Clinician must relate to parent that early lower canine extractions are not a cure-all - necessity to do so is indicative of significant problems
 - Consult indicated with clinician who will ultimately do orthodontics as

- negative effects of early lower canine removal include lingual collapse of incisors, arch length loss, bite deepening and increased overjet - all significant detriments in brachyfacial/deepbite cases
- Because of primary canine extraction's tendency to reduce the arch perimeter, placement of a lower lingual arch is strongly recommended
- “Early” Phase I arch development >>> Use of Edgewise 2X4 appliance to position incisors and molars toward favorable Class I relationships with incisor integrity, midline coincidence, and normal overbite & overjet. Discrepancies requiring extensive arch expansion to relieve incisor crowding and offset negative effects of space loss and early canine extractions are candidates for Phase I treatment
- Facial type a critical factor in decision as to extraction versus non-extraction arch development
 - Brachyfacial/Deepbite >>> Prioritize arch development/expansion
 - Dolichofacial/Openbite >>> Extraction protocol much more likely
- “Late” Supervision of Leeway Space: Use lower lingual holding arch along with selective extraction of primary molars > reserve “E-space”, control late mesial shift and lower incisor up-righting
 - Timely placement of LHA allows distal eruptive positioning of premolars and canines (on average 1.5 mm. distal placement). Provides 2 to 4 mm. of space for relief of incisor crowding
 - Applicable in two-thirds to three-fourths of children with normal crowding patterns
 - Initiation of Edgewise therapy to position incisors and molars toward Class I relationships while controlling leeway space also applicable in timing with loss of second primary molars

Ectopic Eruption of First Permanent Molars

- Resorption of distal root of second primary molars by erupting six-year molars
 - Incidence of 2-3% in maxillary arch, rare in lower arch.
 - Self-correction (reversible) reported in two-thirds of cases without significant implications
 - Irreversible ectopic molars diagnosed with lack of self-correction by dental age 7 years, eruption of opposing lower first molar above the occlusal plane. Intervention indicated as a:
 - Potential space loss with blocked first molars, loss of second primary molar
 - Asymmetric arch development - including supraeruption of opposing dentition
 - Diagnosis - Panorex or adult BWX (#2) at age 6 to 7 years; symmetry of eruption timing
- **Treatment** concepts:
 - Observation: Since approximately 2/3 self-correct, watchful waiting often legitimate approach if detected at age 5 or 6 years. Rarely self-corrects after age 7.
 - Interceptive therapy with appliances to guide or place first molar into normal position, retain favorable eruption sequence, maintain arch length, and maintain level occlusal plane
 - Brass ligature wire or elastic separators - minimal lock
 - Fixed palatal arch wire from E's with distalization spring to first molar (Humphrey appliance)
 - Fixed palatal arch wire from E's with distalization elastics to bonded button on first molar (Halterman appliance)

Dental/Functional Anterior Crossbite

- Maxillary incisors lingually positioned to lower incisors in centric occlusion
 - Dental: Lingual malpositioned upper incisors related to local tooth displacements - typically 1 or 2 teeth only. Usually over-retained primary incisors when centrals - If laterals, suggests arch size problem
 - Functional: Dentoalveolar crossbite with lingually displaced upper incisors complicated by anterior shift of the mandible to exaggerate crossbite discrepancy (pseudo-Class III). Typically incisor first contact with shift to full crossbite in maximum intercuspal position - multiple incisors involved
 - Skeletal: True Class III with prognathic mandible and retrognathic maxilla - usually involves all anterior teeth in crossbite with total anterior and posterior constriction
- Differential diagnosis: Evaluate CR - CO differences for following variables:
 - Facial Profile - With functional shift becomes more prognathic. Without shift, profile static
 - Number of Teeth - One or two probably dental; if all incisors then more likely functional or skeletal
 - Mandibular Closure : If patient can self-contact incisors edge-to-edge, functional aspect present
 - Molar Relationships - Class I > III with shift on closure. Static without shift.
 - Cephalometric Analysis: Skeletal vs. dental, Incisor inclinations a key to diagnosis -
 - Retroclined uppers, proclined lowers = dental/functional
 - Proclined uppers, retroclined lowers = skeletal
 - Familial Appearance
- **Treatment** planning for dental/functional crossbite: Almost always involves labialization of maxillary incisors with goal of satisfactory overjet and overbite, elimination of functional displacement.
 - Considerations in treatment planning include:

Space Available	Stage of eruption/access	Degree of overbite
Severity of displacements	Local etiology	Cooperation
Mandibular incisor positioning (occasionally needs retraction)		
 - Maxillary incisor directed biomechanics / appliances:
 - Tongue blade/popsicle stick - Patient self-guide incline leverage concept for minimal overlap
 - Mandibular acrylic inclined plane- Cautiously recommended due to potential trauma consequences
 - Removable Hawley with finger spring(s): labialize involved upper incisors. Incorporates posterior biteplane to seat appliance, decrease anterior interferences for labial tooth movement
 - Fixed Palatal rigid archwire from bands on first permanent molars with finger spring(s): labialize involved maxillary teeth
 - Labial archwires/Edgewise brackets - In conjunction with other major incisor alignment

Anterior Openbite With Extraoral Habit

- Prolonged digit (thumb, finger) or pacifier sucking habits into the mixed dentition produce likely malocclusion factors, dentoalveolar changes, and abnormal muscle activity
 - Effects On Occlusion
 - Flaring of maxillary incisors/lingual inclination of mandibular incisors > Increased overjet

- Anterior openbite: disrupted incisor eruption/distorted occlusal plane anteriorly
- Distortion of Maxillary Alveolar Process
- Posterior Crossbite – Definite association if habit prolonged into transitional dentition
- Class II molars with alteration of basal bone (?)
- Abnormal Muscle Activity
 - Tongue Thrust - almost always occurs in response to openbite
 - Perioral dysfunction: Lip and mentalis habit with tongue thrust and overjet/openbite
 - Mouthbreathing (?)
- Differentiate dental vs. skeletal openbite/overjet and airway complications.
- Motivation of patient/parents - The “ teachable “ moment for successful treatment
- **Treatment:** Eliminate habit and control tongue thrust to allows potential for “self-correction” of incisor malpositioning. Typical step-wise protocol dependent on patient age and cooperation:
 - Watchful Waiting: While O.K. below age 4 to 5 years, as incisor transition period approaches more aggressive management encouraged given potential malocclusion factors
 - Psychological rewards - first choice at 4 to 6 years of age using reward program for three months
 - Mechanotherapy: Palatal Crib appliance- once incisors in transition, 6 to 10 years of age
 - Crib reminds child not to engage in habit, interferes with digit placement and restrains tongue from forward positioning
 - Promotes incisor self-alignment and eruption by eliminating digit and tongue interferences
 - Planned for six-months wear, habit usually ceases within weeks
 - Corrective Edgewise appliances: Mechanically align dentition in conjunction with crib therapy
 - May be necessary in some mixed dentition cases with 2 X 4 mechanics
 - Usually necessary in late mixed dentition or adolescent patients
 - Myofunctional Therapy - only if associated speech problems

Posterior Crossbite In The Mixed Dentition

- Posterior segmental dentition (primary canines, primary molars, permanent first molars, premolars) in transverse displacement with upper positioned lingual to lower (5 - 8 % of children)
 - Dental : Isolated dental malpositioning - usually only 1 or 2 teeth
 - Functional: As in primary dentition, lateral shift of the mandible on closure usually due to inadequate maxillary arch width. Greater than 90% of posterior crossbites in mixed dentition have functional component which results in:
 - Lower midline shift to crossbite side as mandible deviates on closure
 - Unilateral lingual crossbite of entire buccal segment from canines back in C.O.
 - Asymmetric Class II molars on crossbite side, Class I - III on non-crossbite side
 - Asymmetric condylar position: rotates around condylar axis on crossbite side, slides down and forward on non-crossbite side
 - Facial asymmetry - mandible shorter on crossbite side, longer on non-crossbite side
 - Skeletal: True transverse discrepancy, usually bilateral presentation with severely constricted upper arch, high palatal vault, midline coincidence in CO and CR positions

- If upper dentition buccal to lower, classified as buccal crossbite or “Brodie Bite” (< 1 %)
- **Treatment:** Directed at establishing normal intraarch symmetry and shape, coordinated interarch relationships transversely and to eliminate functional displacements (i.e. allow normal closure)
 - Treatment planning considerations include:

• Stage of Dentition	• Eruption Status/Appliance Retention
• Arch Length/Circumference	• Amount of Expansion Needed
• Nature of Expansion - Orthodontic/Orthopedic	• Patient Age
• Molar Rotations	• Habits/Overbite
• Facial Type	• Associated Alignment Objectives
• Cooperation	• Personal Preference
 - Maxillary biomechanics/expansion appliances:
 - Cross-arch elastics: Isolated dental displacements
 - Removable Schwarz Plate: Provides arch expansion limited to lateral dental up-righting
 - W-arch/Quad-helix: Functional crossbites in early to mid-mixed dentitions
 - Palatal Expander/Hyrax: Bilateral posterior crossbite to optimize orthopedic sutural separation throughout mixed dentition. Indicated for increased anchorage requirements for functional and bilateral crossbites in late mixed dentition once first bicuspids erupted

Maxillary Canine Eruptive Displacement

- Maxillary permanent canines exhibit impaction demonstrating palatal displacement in 1 - 2% of population (females affected three times more frequently than males). Much more common is severe mesiolabial displacements secondary to segmental crowding
 - Labial and palatal malpositioning often associated with atypical resorption of permanent incisors
 - Ectopic eruption or impaction may be associated with constricted maxillary intercanine width, agenesis or microdontia of the lateral incisor, and arch length deficiency
- Early Recognition
 - Approximating 10 to 12 years, radiographic evaluation with periapical or panoramic radiographs
 - Clinical signs: lateral incisor distal inclinations pronounced, small maxillary lateral incisors present, primary canines not appropriately mobile, eruptive bulging of canines atypical
- **Treatment** Intervention
 - Early: Excessive canine mesial orientation may be redirected to more distal and vertical eruption path through removal of primary canine around time canine has approximately two-thirds root development
 - If displaced permanent canine overlap of adjacent lateral incisor not beyond midline long axis of the lateral, chances for canine normal repositioning after primary canine extraction 85 to 90%. - If canine overlap beyond lateral incisor long axis, successful repositioning approximately 60%
 - Successful progress determined at one-year follow-up = most problems with palatal canines
 - Late: Edgewise appliances to ensure space for positioning and establish

anchorage for orthodontic eruption. Uncover canine, engage with attachments to orthodontically erupt and align into archform

Congenitally Missing Permanent Teeth (excluding third molars)

- Incidence of missing permanent teeth is about 4% with no gender differences
 - Affects two or more teeth in about half of cases, frequently symmetrical pattern involving antimeres.
 - Most frequent are lower second bicuspids, upper lateral incisors, and upper second bicuspids
 - Consideration: cone-shaped teeth are characteristically seen in association with missing teeth
- Key issue involves consequences and considerations to long-term arch alignment
 - Disruption of normal dimensions can result in adjacent teeth drifting, supraeruption of opposing teeth, and improper space accommodation with attendant aesthetic and functional disharmony
 - First question involves decision whether to maintain primary tooth or to allow closure of the spaces
 - Location of missing teeth: Mesial eruption paths and alignment tendencies of adjacent permanent canines may compensate for missing maxillary lateral incisors. Such “self-correction” adjustments are less likely to occur in bicuspid areas unless severe arch length discrepancy present and extra space needed for alignment
 - Number of missing teeth: When bilateral antimeres missing, space closure is often desirable to obviate the need for prosthetic replacement. If a single tooth is missing, maintenance of space and eventual prosthetic management may be more likely for symmetrical harmony
 - Patient age: Early recognition of significance as planning options are broader in terms of occlusion development, supervision of arch integrity, and the status of the primary predecessor
 - Tendency for overlying deciduous tooth to persist until young adulthood.
 - Predictive range is great with individuals demonstrating premature loss of the primary tooth or, in others; persistence for decades
 - For primary molars with missing premolars, ankylosis a common factor relative to arch relationships
- **Treatment options**
 - Missing maxillary permanent laterals:
 - Maintain primary lateral and/or manage and open space orthodontically with long-term plan for implants or Maryland bridge. This is usual emphasis in non-crowded arches with Class I and Class III occlusion patterns
 - Enhance movement of permanent canines forward into lateral position with “early” extraction of primary laterals, later orthodontic alignment for “canine” replacement. This is more likely the emphasis in crowded arches and Class II occlusion patterns
 - Missing second premolars:
 - Maintain primary molar and/or manage space with long-term plan for implants or bridge. This is usual emphasis in non-crowded arches with Class I and Class II occlusion patterns
 - Enhance movement of permanent first molars forward with “early” extraction of primary second molar, later orthodontic alignment for full space closure. This

- is more likely the emphasis in crowded arches and Class III occlusion patterns
- Important to keep in mind that case reports exist showing second premolar tooth germ development / calcification not until as late as 10 years of age

Ankylosed Teeth

- Lower first primary molars most commonly affected, followed by upper first primary molars, lower second primary molars and upper second primary molars
 - Also occurs secondary to traumatized primary and permanent anterior teeth
 - Resorption of ankylosed molars usually proceeds in normal mode with 95% of premolars erupting into proper occlusion with normal periodontal health and alveolar bone height
 - Most common sequelae is simply delayed transition as to timing
- Static retention of ankylosed tooth often results in clinical “submersion”, supraeruption of opposing tooth and tipping of adjacent teeth with accompanying loss of space. Severity related to how early ankylosis occurred, which tooth involved, which arch
 - Second primary molars of much greater significance to arch integrity than first primary molars
 - Particularly true when ankylosis occurs prior to eruption of first permanent molar
 - Greater vertical development of maxillary processes can effectively “bury” ankylosed upper second primary molar with more severe consequences. Often requires complicated surgical removal
- **Management/Intervention**
 - Early on, clinician may choose to monitor as tooth often shows normal exfoliation. Mesiodistal width and occlusal relationships may be maintained with composite build-ups, stainless steel crowns
 - Eventual treatment often involves extraction of involved primary molar at later stages if exfoliation is delayed or deflected eruption of the succeeding permanent tooth is manifest
 - In case of significant vertical disharmony, extraction of ankylosed molar may enhance outcomes by avoiding excess arch collapse and less complicated extraction. Appropriate space maintainers should be considered unless there is sufficient crowding to justify a serial extraction plan

Supernumerary Teeth

- Reported in up to 3.6 % of children
 - Occur approximately ten times more frequently in maxilla than mandible; boys twice as often as girls
 - Mesiodens: 80% single, 20% two or more. Usually present cone-shaped crown with single root
 - More than 90% are lingually or palatally malpositioned
 - Approximately 3 of 4 remain unerupted and need surgical removal
 - Can be responsible for delayed eruption of permanent teeth, over-retention of primary teeth, displaced teeth, diastemas, abnormal root resorption, follicular or dentigerous cysts, and resultant malocclusion
- Remove when no harm will come to developing permanent teeth
 - Prefer to wait until one-half to two-thirds root development of adjacent permanent teeth

- Patient age and potential for cooperation also factors in delaying surgical intervention
- Watchful waiting allows time for possible eruption of supernumerary, avoidance of surgical exposure
- When removed, exposure of permanent teeth with provision of eruption channel recommended
 - Up to 80% of permanent maxillary teeth spontaneously erupt after supernumerary removed
 - Orthodontic treatment often necessary to make room for unerupted teeth and to position properly

VI. TREATING SKELETAL MALOCCLUSIONS IN THE MIXED DENTITION

Overview of Dentofacial Orthopedics

- Biomechanical treatment directed at altering the relationships of the jaws and the activity patterns of orofacial muscles to affect changes in facial proportions
- Rationale: Objectives of facial and dental esthetics with functional harmony can rarely be achieved without compromise unless basal arch relationships are in orthognathic position with Class I molar and canine relationships, acceptable overbite and overjet, coordinated transverse archforms
- Significance: By definition, to modify growth one must treat during active growth periods - i.e. in conjunction with the pubertal growth spurt or earlier. More severe discrepancy—earlier the treatment
- **Treatment** techniques should employ dentofacial orthopedics that most directly attack the area of discrepancy to modify skeletal growth patterns in all three planes of space
 - Most Class II, Division 1 malocclusions present with normal maxilla, notable mandibular retrognathia (85%), vertical growth tendency, narrowed upper arch, good lower arch. Full Class II, ANB > 6 degrees
 - Most Class II, Division 2 malocclusions present with normal maxilla, mild mandibular retrognathia, strong chinpoint, deepbite growth tendency, broad archforms, end-on Class II, ANB < 6 degrees
 - Class III malocclusions present with combination of maxillary retrognathia, mandibular prognathia, negative ANB, vertical growth patterns, transverse maxillary deficiency, retroclined lower incisors

Transverse basal arch expansion

- Basal orthopedic maxillary expansion involving sutural separation much more readily achieved prior to anatomical “interlocking” of mid-palatal sutures approximating age 12 years. See posterior crossbites for highlights on maxillary expansion appliance options
- Optimizes developmental environment for mandibular growth and function in approximately 50% of Class II, Div. 1 malocclusions. Frees mandible from constricted retrusion to allow advancement
- Almost all Class III patients require expansion as an essential component of correction to coordinate arch widths, relieve crowding, and enhance forward development of maxilla

Anteroposterior Class II malocclusion > Retrusive Mandible > Functional Appliances

- Promote mandibular growth by advancing with protrusive bite appliance, restrain maxillary forward development for “catch-up”
 - Most studies indicate increased mandibular skeletal length on the order of 1 mm. above normal changes over course of one year of treatment with most appliance approaches
 - Holding mandible forward produces reactive forces on adjacent structures. These reactive forces combine with any enhanced mandibular growth toward the correction of Class II malocclusion by:
 - moving (or restricting) the upper teeth backward
 - moving the lower teeth forward
 - restraining maxillary skeletal growth (i.e., the so-called “headgear effect”)
- Conditions for favorable use of functional appliances include:
 - Treatment carried out during periods of active growth (i.e. on upward slope of growth spurt)
 - Favorable growth pattern (i.e. horizontal growth) - Functional appliances increase lower face height and are thus contraindicated in cases with a large LFH (i.e. dolichofacial grower)
 - Nasal airway not compromised (dolichofacial patterns are problematic)
 - Symmetrical dental arches without major anomalies in position or crowding of the teeth
 - Cooperation
- Examples of functional advancement appliances include:
 - Bionator/orthopedic corrector: Removable acrylic design allows eruptive guidance by selective grinding of appliance
 - Activator: Full maxillary coverage restrains maxillary development. Acrylic design allows eruptive guidance of dentition by selective grinding of appliance
 - Frankel: Shield design reduces compressive forces of buccal musculature and abnormal lip positioning for arch expansion
 - Herbst: Telescope design displaces mandible forward with lower dental protraction, distalizing affect on upper molars and restraining effect on maxilla. Fixed design offers less problems with cooperation, more appliance emergency problems

Anteroposterior Class II Malocclusion > Protrusive Maxilla > Directed Headgear

- Promote restraint of maxillary dental and skeletal forward and vertical development, distalize upper arch, and allow normal mandibular growth - orthodontic and orthopedic effects possible
- Cervical-pull Headgear: Optimize molar distalization, redirect vertical development, influence maxillary skeletal growth, decrease overbite. Primarily works by:
 - promoting molar extrusion and distalization of crowns - average 3 mm./year distalization
 - “shearing” effect at sutures enhances displacement of maxillofacial complex with about 0.5 to 1.0 mm. in distal movement of A point
 - increases lower face height as FMA increased, pogonion drops down as mandible rotates

- High-pull Headgear: Optimize orthopedic restraint of maxillary growth and minimize vertical eruptive development, enhance overbite
 - promotes horizontal and bodily dental movement of molars, distalization effects are minimal
 - restrains vertical/forward development of molars (i.e. relative “intrusion” of molars)
 - restrains downward/forward growth of maxillary complex, more posteriorly at PNS than at ANS
 - minimizes FMA and lower face height changes to reduce bite opening.
- Indications for extraoral headgear beyond maxillary protrusion include:
 - Retraction will not compromise nasolabial profile
 - Distalization of buccal segments to gain arch length and optimize Class I molars
 - Anchorage support for incisor retraction
 - Symmetrical A-P positioning
 - Active growth: on upward slope of growth curve
 - Arch expansion desirable to enhance forward movement of mandible
 - Cooperation/understanding of patient/family

Anteroposterior Class II Malocclusion with Acceptable A-P Skeletal/Profile Relationships

- Promote corrective changes by restraining / distalizing upper dentition, protracting lower dentition
- Class II elastics: possible orthodontic and orthopedic effects, requires Edgewise appliances
- Distalize maxillary posterior segments (e.g. headgear, distal jets, springs, etc.)
- May incorporate selective permanent tooth extractions to camouflage A-P discrepancy
- Control of excess vertical facial development
 - Maxillary restraint with high-pull headgear
 - Maxillary restraint with full coverage functional appliances (Combination Activator-Headgear)
- Class II Division 2 malocclusion usually presents normal maxilla, acceptable mandible, severely retroclined incisors and deepbite. The occlusal patterns may be “locking” the mandible in a retruded position by the vertical angulation of the maxillary incisors. Treatment options directed at:
 - Generally avoid extraction protocols given tendency to deepen bite
 - Unlock mandible by correcting maxillary incisor position as targeted first phase of treatment
 - If mandible repositions, level and align
 - If Class II persists, consider function appliance, headgear therapy, Class II elastics

Anteroposterior Class III Malocclusion

- Restrain mandibular growth
 - Chin-cup therapy
 - Restraint of true mandibular growth length not documented by long-term studies
 - Primarily redirect mandibular growth direction more vertically rather than in desired A-P sagittal direction. Usually contraindicated given typical dolichofacial growth pattern

- Protract the maxillary complex
 - Extraoral reverse-pull headgear (facemask)
 - “Redirections” or enhances maxillary growth with forward protraction
 - Affects on teeth and orthopedic changes depend on force direction, magnitude, duration, time, appliance anchorage, and patient age
 - Allows transitional dentition treatment
 - Expand maxillary arch to “unlock” occlusion for enhanced maxillary forward movement and possibly restrict mandibular forward development
 - Expansion and occlusion changes may reduce forward mandibular positioning
 - Concurrent mesial movement of upper molars and incisors to establish overjet and overbite
 - May eliminate abnormal muscle function
 - Application of facemask in primary and early mixed dentition consistently shown to produce most dramatic results for Class III correction in the shortest time period
 - Treatment concurrent with incisor eruption to optimize growth and occlusal relationships
 - Early correction concepts allude to idea that early orthopedic correction of malocclusions ultimately incorporated into future craniofacial growth patterns
 - Supplemented with concurrent rapid palatal expansion may aid skeletal changes
 - Results from reverse-pull headgear wear attributed to:
 - Mesial movement of maxillary molars/incisors - up to 3 mm./year forward movement
 - “Shearing” effect at sutures to maximize displacement of the maxillofacial complex - about 1.0 mm. forward movement of A point
 - Increased lower face height, FMA is increased, pogonion drops down and back as bite opens to enhance A-P correction - may worsen vertical growth
 - increases lingual uprighting of lower incisors

VII. ADDITIONAL READINGS

1. Brennan MM, Gianelly AA. The use of the lingual arch in the mixed dentition to resolve incisor crowding. Am J Orthod Dentofac Orthop 2000; 117: 81-85.
2. Bishara SE, Jakobsen JR, Treder J, and Nowak A. Arch width changes from 6 weeks to 45 years of age. Am J Orthod Dentofac Orthop 1997; 111 (April): 401-409.
3. Dean JA. Management of the developing occlusion. In: McDonald RE, Avery DA, and Dean JA eds. *Dentistry for the Child and Adolescent*. 8th ed. St. Louis, MO: Mosby; 2004:625-683.
4. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Eur J Orthod 1988; 10: 283-295.
5. Terlaje RD, Donly KJ. Treatment planning for space maintenance in the primary and mixed dentition. J Dent Child 2001; 68: 109-114.
6. Lagravere MO, Heo G et al. Meta-analysis of immediate changes with rapid maxillary expansion treatment. JADA 137:44-53, 2006.
7. Petren S, Bondemark L et al. A systematic review concerning early orthodontic treatment of unilateral posterior crossbite. Angle Orthodontist 73:588-596, 2003.

Chapter 11: RECORD KEEPING AND FORMS

AAPD POLICIES:

http://www.aapd.org/media/Policies_Guidelines/G_Recordkeeping.pdf

http://www.aapd.org/media/Policies_Guidelines/G_Informed%20Consent.pdf

- I. GENERAL INFORMATION AND PRINCIPLES**
- II. PATIENT INFORMATION SECTION**
- III. MEDICAL AND DENTAL HISTORY**
- IV. EXAMINATION AND TREATMENT PLANNING**
- V. TRAUMA ASSESSMENT**
- VI. PHARMACOLOGICAL/BEHAVIOR GUIDANCE**
- VII. PREVENTIVE RECALL**
- VIII. RESTORATIVE**
- IX. COMPREHENSIVE ORTHODONTIC**
- X. CONSULTATION REQUEST**
- XI. INFORMED CONSENT**
- XII. ADDITIONAL READINGS AND WEBSITES**

I. GENERAL INFORMATION AND PRINCIPLES

- Patient charts must contain informed consent
- Front of chart must contain medical alerts
- Chart entries must be made using black ink (preferred) rather than pencil
- Individual patient chart (not several family members in the same chart)
- Consultations when indicated must be in the chart
- Date of each visit noted
- Information given to the parent at the each visit is noted
- Chart entry contains the name of the staff member making the note
- Chart entry contains the name/initials of the doctor or identifying designation
- Essentials of a dental record: medical history, dental history, clinical assessment, diagnosis, treatment plan, progress notes
- Supplemental parts (as indicated): radiographs and assessment, sedation records, orthodontic records, consultations, laboratory results, special assessments
- Update medical history at each treatment or diagnostic visit

II. PATIENT INFORMATION SECTION

Information section should contain

- patient name and nickname
- date of birth/age
- date of entry
- parent or responsible party home telephone number
- parent or responsible party work telephone number
- chief complaint/reason for visit

III. MEDICAL AND DENTAL HISTORY

Medical history should contain

- allergies
- idiosyncratic or adverse drug reactions
- immunizations
- current medications and dosage
- recurrent headaches
- congenital birth defects
- seizures
- mental retardation/developmental delays
- behavioral/learning problems
- history of blood transfusions and date
- history of abnormal bleeding
- problems with:
 - Heart
 - Kidney
 - Liver/GI
 - Endocrine system
 - Breathing/lung
 - Hearing

Sight

History of

- cancer, tumors, blood dyscrasias
- hospitalizations, surgeries, or injuries
- infections of bacterial or viral origin
- name and address of physician
- date of last physical exam
- Substance use
- Sexually transmitted disease
- Pregnancy
- a question to allow the parent/responsible party to relate any other significant problem
- Family history
- Social history

Dental history should include:

- TMJ/TMD history
- fluoride usage/exposure history
- previous dental experience: routine and emergency
- date of last dental visit/radiographs
- dental related habits: oral hygiene, diet
- bottle usage
- a question, regarding social development
(personality/temperament)
- family dental history

IV. EXAMINATION AND TREATMENT PLANNING

- Examination record should include:
 - occlusal classification /canine relationship
 - occlusal classification /molar relationship
 - overbite relationship
 - overjet relationship
 - facial profile noted
 - midline shift noted
 - amount of midline shift and responsible arch noted
 - presence of cross-bites and description
 - presence of crowding noted
 - presence of oral habits noted
 - dental development (advanced or delayed?)
 - behavior of child
 - intraoral soft tissue examined/pathology noted
 - Tongue
 - Floor of mouth
 - Palate and oropharynx
 - Buccal mucosa
 - Gingiva
 - TMJ/TMD evaluation
 - Assessment of oral hygiene
 - Intraoral hard tissue examined
 - Presence of caries
 - Presence of existing restorations/sealants/applications
 - Morphology of teeth/hypoplasia

- Presence of dental pain
- Behavior rating (Frankl)
- Caries Risk Assessment (CAT)
- Treatment Plan should contain:
 - Problem list or diagnoses
 - Tooth-by-tooth plan
 - Prioritized sequence of treatment by specialty area
 - Behavior guidance
 - Preventive plan
 - Other considerations
 - Consultation with appropriate health care provider for medically compromised patients

V. TRAUMA ASSESSMENT

- Entries must include (see AAPD Trauma Form)
 - how?
 - what?
 - where?
 - when?
 - classification of crown fracture if present
 - degree of root formation
 - root fracture or absence of
 - integrity of alveolar bone
 - pulpal involvement
 - mobility and or displacement
 - sensitivity to temperature
 - sensitivity to percussion
 - soft tissue damage
 - mental status /gross neurological assessment
 - tetanus immunization status
 - radiographs taken
 - prognosis
 - plan for follow-up treatment and intervals

VI. PHARMACOLOGICAL/BEHAVIOR GUIDANCE

Record (excluding N₂O/O₂ alone) must contain:

- monitoring and recording of vital signs in accordance with AAPD guidelines
- health status update
- type and dose of sedation used
- type and dose of local anesthetic used.
- percentage of N₂O/O₂used
- weight of patient
- informed consent for behavior guidance techniques employed
- documentation of the need for sedation
- time sedation given
- NPO recommendation
- time N₂O/O₂ started and discontinued
- time of dismissal
- condition of child upon dismissal (ambulatory, talkative, etc.)
- effectiveness of sedation

- presence of complications
- use of restraints
- pre and post-operative instructions

VII. PREVENTIVE/RECALL

Examination record must contain same information as new exam

VIII. RESTORATIVE

Patient record note in progress note section should include:

- identification of the teeth restored
- identification of areas/surfaces of restoration
- use and type of base material/liner
- restorative material
- type and dose of anesthetic used
- documentation of materials used in pulp therapy
- behavior guidance used and result

IX. COMPREHENSIVE ORTHODONTIC

Record elements may include:

- lateral cephalogram with name and age of patient and date taken
- pretreatment panoramic radiograph or full mouth radiographic survey
- pretreatment study casts with name of patient and date
- diagnosis of problem
- sequential detailed treatment plan
- history at exam to include:
 - Presence of allergies
 - Airway assessment
 - Mouth breathing
 - Missing teeth
 - TMJ/TMD evaluation
 - Occlusal plane
 - Arch length analysis
 - Sequential detailed treatment objectives
 - Sequential detailed treatment time estimation
 - Photos of patient with date
 - Post-treatment study casts with name and date
 - Post-treatment cephalogram and panoramic survey

X. CONSULTATION REQUEST

A request for a consultation from another service should have:

- Service being consulted (eg, Cardiology)
- Patient Identifiers: name, telephone, address
- Specific question being asked and nature of clinic service:
 - Consultation only, assume management of the patient, assume management in consultant's field
 - Consultant's brief confirmation of understanding of request
 - Examination and historical findings

- Assessment of patient
- Recommendations for the requesting provider
- Identification of consultant
- Patient history and relevant findings for consultant
- Name and contact information of requesting provider
- HIPPA releases
- Special considerations: urgency, patient management issues

XI. INFORMED CONSENT

- Every patient has the right to be informed of the nature of their condition, the risks of undergoing the proposed treatment, alternative treatments available and their risks, and the risk of foregoing any treatment whatsoever
- The information to be given to the patient is that information that the average, reasonable patient should have in order to make a rational, informed decision
- Factors to consider in determining which risks to inform the patient about are, the severity of the risk and the probability of the risk occurring (the greater the severity or the greater the probability, the more necessary it is to inform the patient)
- Four steps in obtaining a patient's (or parent's) informed consent:
 - give the necessary information to the patient verbally, visually, by printed material or by any appropriate combination
 - insure that the patient fully understands the information received and has ample opportunity to question
 - obtain the patient's consent; ideally, it should be unequivocal consent
 - document having obtained informed consent by having the patient sign an appropriate consent form and incorporating that form into the record either physically or by reference or adequately document in the record the procedure by which the appropriate informed consent was obtained
- A signed, printed form is not specifically required by law, but is usually the most appropriate way to ensure that all necessary information was given to the patient
- The presumption that informed consent has been obtained because the dentist possesses a form signed by the patient (or parent) is a rebuttable presumption and will not prevent a lawsuit from proceeding
- Printed consent forms should be neither too general nor too complicated
- No patient or parent can give valid consent for any treatment that is otherwise negligent
- Failure to obtain informed consent can be the basis for both a criminal charge of a battery and for a civil malpractice lawsuit and appropriateness of treatment is irrelevant in such a claim
- In *emergencies*, a limited privilege exists for a dentist to provide treatment if the patient may otherwise suffer irreparable harm, if actual consent is unobtainable, and if the average, reasonable patient would not otherwise refuse (To relieve a patient of pain, to reduce significant swelling, to control hemorrhage, or to prevent serious permanent deformation are examples)
 - no privilege exists for the dentist to provide subsequent, remedial treatment
- In providing treatment to handicapped individuals ascertain whether that individual has the mental capacity to provide a valid informed consent and if not, determine who is responsible to provide consent for the patient

- In most states, per statute, the age of majority is eighteen—an emancipated minor is a minor who has not yet attained the chronological age of majority but is considered, legally, to be an adult, and therefore capable of giving consent (courts of law will consider a minor emancipated if living outside of the parental home and capable of self-support)
- A dentist may be requested by a parent to provide dental treatment to a child who, although not legally emancipated, is mature enough to fully understand the nature and consequences of the treatment (the mature minor) and does not wish it to be performed (i.e., some teenagers); if treatment is elective, it is probably best delayed until parent and child agree
- Good Samaritan Laws vary by state, but they usually provide a defense against liability should a dentist provide emergency care if there exists no duty to provide such care (i.e., if no doctor-patient relationship) and if there is no expectation of remuneration
- Because many parents may not be familiar with commonly used behavior guidance methods, it is imperative that their consent be obtained prior to using any of the methods, in accordance with AAPD Guidelines

XII. ADDITIONAL READINGS AND WEB SITES

AAPD Trauma Form:

http://www.aapd.org/media/Policies_Guidelines/G_Trauma.pdf

AAPD Caries Risk Assessment Tool

http://www.aapd.org/media/Policies_Guidelines/RS_CAT.pdf

Health Insurance Portability and Accountability Act:

<http://www.hhs.gov/ocr/hipaa/>

Chapter 12: INFECTION CONTROL

AAPD ORAL HEALTH POLICY:

http://www.aapd.org/media/Policies_Guidelines/P_InfectionControl.pdf

- I. GUIDELINES FOR EXPOSURE DETERMINATION AND PREVENTION
- II. USE OF PERSONAL PROTECTIVE EQUIPMENT (T)
- III. INFECTION CONTROL CATEGORIES OF PATIENT CARE INSTRUMENTS (T)
- IV. METHOD FOR STERILIZING AND DISINFECTING PATIENT-CARE ITEMS AND ENVIRONMENTAL SURFACES (T)
- V. MAJOR METHODS OF STERILIZATION (T)
- VI. GUIDE FOR SELECTION OF APPROPRIATE DISINFECTION METHODS FOR ITEMS TRANSPORTED TO OR FROM THE DENTAL LABORATORY (T)
- VII. ADDITIONAL READINGS AND WEB SITES

I. GUIDELINES FOR EXPOSURE DETERMINATION AND PREVENTION

Listed below are the minimum requirements recommended during controlled situations to protect the health care worker from potentially infectious agents. These lists are not all inclusive, so judgment is required on the part of the health care worker to access the need for additional barrier protection in less controlled situations.

- Hospitals may impose varying regulations for different types of patients:
 - immunocompromised
 - those with infectious diseases (tuberculosis)
 - those potentially infective (e.g., exposed to chickenpox)
- Non sterile gloves are to be used unless it is a strict or aseptic procedure
 - refer to your hospital policy and procedure manual
- Sharps
 - recap needles with one-handed/scoop or use recapping device
 - dispose of sharps in puncture-proof container
 - don't bend needles
- Hand pieces and Air/Water Syringes
 - high speeds: clean and sterilize after each patient; discharge air and H₂O for 20-30 secs after each patient; antiretraction valves in H₂O line
 - slow speeds: clean and sterilize all attachments (except motor); disinfect motor cover
 - air/ H₂O syringe: sterilize tips or use disposables; disinfect/sterilize syringes handle
- Dental unit waterlines
 - (1) regulatory standard for safe drinking water (EPA and APHA/AWWA) ≤ 500 CFU/mL
 - (2) methods of microbial control include:
 - (a) self-contained water systems combined with chemical treatment
 - (b) in-line microfilters
 - (c) autoclavable water delivery systems
 - (d) other water treatment strategies including UV, reverse osmosis, super-heating of entrance water
 - (e) combinations of the above
 - (3) Simply using source water containing ≤ 500 CFU/mL of bacteria in a self-contained system will not eliminate bacterial contamination in treatment water if biofilms in the water system are not controlled. Removal or inactivation of dental waterline biofilms requires the use of chemical germicides.
 - (4) The majority of recently manufactured dental units are engineered to prevent retraction of oral fluids (e.g., through handpieces, ultrasonic scalers, or air/water syringes). Older dental units with antiretraction valves - flushing of devices for a minimum of 20-30 seconds after each patient is recommended.
 - (5) Periodic monitoring of dental unit water quality should occur

- Hand piece sterilization protocol:
 - (1) Flush hand piece air/H₂O before removal from line (bur in)
 - (2) Clean and dry instrument
 - (3) Apply hand piece cleaner and /or lubricant if required
 - (4) Expel excess lubricants (bur in)
 - (5) Clean fiber optics
 - (6) Bag and heat process hand piece
 - (7) Flush air/H₂O lines (20-30 secs) in hose before attaching
 - (8) Open bag (lube if needed) attach hose and expel excess lube (bur in)

II. USE OF PERSONAL PROTECTIVE EQUIPMENT

<i>Situation</i>	<i>Hand hygiene</i>	<i>Glove</i>	<i>Fluid Resistant Gowns</i>	<i>Goggles/ Mask</i>
Clean surfaces contaminated by blood/body fluids (minor)	x	x	x	x
Contact with blood saliva, mucous membranes	x	x	x	x
Contact with blood soiled items, body fluids	x	x	x	x
Examining all oral lesions	x	x		x
When splattering or splashing blood or other body fluids is likely (cavity preparation, prophylaxis)	x	x	x	x
Performing decontamination procedures on soiled instruments	x	x	x	x

X = Required

III. INFECTION CONTROL CATEGORIES OF PATIENT CARE INSTRUMENTS

Category	Definition	Dental instrument or item
Critical	Penetrates soft tissue, contact bone, enters into or contacts the bloodstream or other normally sterile tissue	Surgical instruments, periodontal scalers, scalpel blades, surgical dental burs
Semi critical	Contacts mucous membranes or nonintact skin; will not penetrate soft tissue, contact bone, enter into or contact the bloodstream or other normally sterile tissue	Dental mouth mirror, amalgam condenser, reusable dental impression trays, dental hand pieces*
Noncritical	Contacts intact skin	Radiograph head/cone, blood pressure cuff, face bow, pulse oximeter

*Although dental hand pieces are considered a semi critical item, they should always be heat-sterilized between uses and not high-level disinfected

IV. METHOD FOR STERILIZING AND DISINFECTING PATIENT-CARE ITEMS AND ENVIRONMENTAL SURFACES

Health-care application					
Process	Result	Method	Examples	Type of patient-care item	Environmental surfaces
Sterilization	Destroys all microorganisms, including bacterial spores	Heat automated High temperature	Steam, dry heat, unsaturated chemical vapor	Heat-tolerant critical and semi-critical	Not applicable
		Low temperature	Ethylene oxide gas, plasma sterilization	Heat-sensitive critical and semi-critical	
		Liquid immersion	Chemical sterilants Glutaraldehyde, glutaraldehydes with phenol, hydrogen peroxide, hydrogen peroxide with peracetic acid, peracetic acid	Heat-sensitive critical and semi-critical	

High-level disinfection	Destroys all microorganisms, but not necessarily high numbers of bacterial spores	Heat-automated	Washer-disinfector	Heat-sensitive semi-critical	Not applicable
		Liquid immersion	Chemical sterilants/ high-level disinfectants Glutaraldehyde, glutaraldehydes with phenol, hydrogen peroxide, hydrogen peroxide with peracetic acid, ortho-phyhalaldehyde		
Intermediate level disinfection	Destroys vegetative bacteria and the majority of fungi and viruses. Inactivates <i>Mycobacterium bovis</i> . Not necessarily capable of killing bacterial spores.	Liquid contact	U.S. Environmental Protection Agency (EPA)-registered hospital disinfectant with label claim of tuberculocidal activity (e.g., chlorine containing products, quaternary ammonium compounds with alcohol, phenolics, iodophors, EPA-registered chlorine-based product)	Non-critical with visible blood	Clinical contact surfaces; blood spills on house-keeping surfaces

Low -level disinfection	Destroys the majority of vegetative bacteria, certain fungi and viruses. Does not inactivate <i>Mycobacterium bovis</i>	Liquid contact	EPA-registered hospital disinfectant with no label claim regarding tuberculocidal activity.** The Occupational Safety and Health Administration also requires label claims of human immunodeficiency virus (HIV) potency for clinical contact surfaces(e.g., quaternary ammonium compounds, some phenolics, some iodophors	Non-critical without visible blood	Clinical contact surfaces; housekeeping surfaces
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V. MAJOR METHODS OF STERILIZATION

Method	Temp	Pressure	Cycle Time	Advantage	Disadvantage
Steam Autoclave	121°C (250°F)	15 psi	15-20 min	Rapid turn around, good penetration, wide range of materials	Corrosion, dulling of unprotected instruments, packages may remain wet
Dry Heat Oven	160°C (320°F)	n/a	2 hours	Does not corrode or dull instruments, no toxic or hazardous chemicals, low cost per cycle	Long cycle time, destroys heat-labile items (plastics)
	170°C (340°F)	n/a	1 hour		
Rapid Heat Transfer	375°F	n/a	12 min for wrapped and 6 min for unwrapped, unwrapped items quickly contaminate	Short cycle, items are dry, small capacity	Cannot sterilize liquids, damage to heat-labile items, cannot open door during cycle

Unsaturated Chemical Vapor	131°C (270°F)	20 psi	30 min	Short cycle time, less corrosive on instruments	Uses toxic or hazardous chemicals, requires fume ventilation, destroys heat-sensitive plastics
Ethylene Oxide	25°C (75°F) Room temp.	n/a	10-16 hours	Suitable for most materials including dental appliances / instruments	Very long cycle time, uses toxic / hazardous chemicals, requires special ventilation

VI. GUIDE FOR SELECTION OF APPROPRIATE DISINFECTION METHODS FOR ITEMS TRANSPORTED TO OR FROM THE DENTAL LABORATORY

Item	Method	Recommended disinfectants	Comments
Casts	Spray until wet or immerse	Chlorine compounds or iodophors	Disinfectant can be prepared using slurry water (saturated calcium sulfate) Probably should not be disinfected until fully set (24 hours)
Impressions	Immersion disinfection preferred		Heat sterilize reusable impression trays Discard plastic trays after use
Irreversible hydrocolloid (alginate)	Disinfect by immersion with caution. Use only disinfectants with short-term exposure times (no more than 10 min for alginates).	Chlorine compounds or iodophors	Short-term immersion in glutaraldehydes has been shown to be acceptable; but time is inadequate for disinfection
Impression compound		Iodophors or chlorine compounds	Phenolic sprays can be used

Prostheses	Immerse in disinfectant Use caution to avoid corrosion of metal Can also be sterilized by exposure to ethylene oxide gas	Rinse thoroughly after disinfection	Clean “old” prostheses by scrubbing with hand wash antiseptic or sonication before disinfection
Removable (acrylic/porcelain)		Chlorine compounds or iodophors	Rinse thoroughly after disinfection; store in diluted mouthwash

Note: Exposure time to disinfectant should be that recommended by the disinfectant manufacturer. All items must be thoroughly rinsed (15 seconds minimum) under running tap water after disinfection.

From Merchant VA: Dental laboratory infection control: OIC update, Dent Learn Syst 3:1-8, 1995.

VII. ADDITIONAL READINGS AND WEB SITES

CDC: Guidelines for infection control in dental health-care settings, MMWR Morb Mortal Wkly Rep 52(No. RR-17) 2003; 8-9, 20 and 65-66.

<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5217a1.htm>

Chapter 13: BEHAVIOR GUIDANCE

AAPD GUIDELINES:

http://www.aapd.org/media/Policies_Guidelines/G_BehavGuide.pdf

http://www.aapd.org/media/Policies_Guidelines/G_Nitrous.pdf

http://www.aapd.org/media/Policies_Guidelines/G_Sedation.pdf

http://www.aapd.org/media/Policies_Guidelines/G_AnesthesiaPersonnel.pdf

I. BEHAVIOR THEORIES

II. BEHAVIOR GUIDANCE PRINCIPLES

III. BEHAVIOR GUIDANCE TECHNIQUES

IV. SEDATION (T)

V. SEDATION ROUTES

VI. MEDICATIONS

VII. PRESEDATION PREPARATION

VIII. MONITORING PRINCIPLES

IX. EMERGENCIES

X. ADDITIONAL READINGS AND WEB SITES

I. BEHAVIOR THEORIES

Psychoanalytic Theory

- Behavior shaped by unconscious processes
- Freud—psychosexual development and orientation
- Erikson—psychosocial development and orientation

Behaviorism

- Relationship between stimulus and response
- Pavlov—classical conditioning-reflex
- Skinner—operant conditioning and selective reinforcement
- Social learning theory—modeling

Cognitive Theory

- Individuals think and choose
- Thoughts influence future actions and ideas
- Piaget—how children think vs. what they know

II. BEHAVIOR GUIDANCE PRINCIPLES

- Considerations that must be evaluated in any clinical setting in which a child treated for oral health issues are:
 - Assessment of the child's developmental level, dental attitudes, and temperament (parental attitudes and communication are important)
 - Predict the child's reaction to treatment
 - Dentist's behavior and attitude
 - Communication principles
 - Barriers to communication and guidance
 - Deferring treatment and
 - Gaining informed consent for behavior guidance
- Behavior guidance is based on scientific principles
- Execution of behavior guidance is more than pure science and requires skills in communication, empathy, coaching, and listening
- Thus, behavior guidance is a clinical art form and a skill built on a foundation of science
- The goals of behavior guidance are:
 - establish communication
 - alleviate fear and anxiety
 - deliver quality dental care
 - build a trusting relationship between dentist and child
 - promote the child's positive attitude toward oral/dental health and oral health care
- The urgency of the child's dental needs must be considered when planning treatment
- Deferral or modification of treatment sometimes may be appropriate
- All decisions regarding use of behavior guidance techniques must be based upon a benefit vs risk evaluation
- Parents share in the decision-making process regarding treatment of their children
- Dental staff must be trained carefully as well

III. BEHAVIOR GUIDANCE TECHNIQUES

Communication and communicative techniques

- TSD: tell, show, do
- Distraction
- Non-verbal communication
- Positive reinforcement
- Voice control
- Parental presence/absence

Advanced techniques – needs consent that is documented

- Protective stabilization (must be safe, time limited, non-punishing)
- Hold and go (although no defined as a technique, it is commonly performed; best for very brief, limited procedures or those not technically demanding)

Pharmacologic Management - needs consent that is documented

- Minimal and moderate sedation
- Deep sedation
- General anesthesia

IV. SEDATION

Principles

- Careful patient evaluation and selection
- Thorough knowledge of selected medications and maximum doses
- Required informed consent from guardian
- Well equipped office - monitors, positive pressure O₂, hi-speed suction, back-up lighting, suction and emergency kit
- Practitioner trained and capable of “rescue” for any unforeseen event associated with sedation
- Back up emergency service
- Pre, intra, post operative written documentation
- Maximum Recommended Dose (MRD) should be calculated by weight and not exceeded
- Immobilization devices (e.g., Papoose Board) should not interfere with adequate ventilation

Goals

- Facilitate the provision of quality care
- Minimize the extremes of disruptive behavior
- Promote a positive psychological response to treatment
- Promotes patient welfare and safety
- Return the patient to a physiologic state in which safe
- discharge is possible (as determined by recognized criteria)

Caveats of minimal and moderate sedation

- Loss of consciousness unlikely
- Patient independently and continuously maintains airway
- Responds to light tactile stimulation or preferably verbal command
- Reflex withdrawal, although a normal response to a painful stimulus, is not considered as the only age-appropriate purposeful response
- Intact cough and gag reflex
- Minimal effect on cardiovascular (CV) and respiratory systems

Caveats of deep sedation

- Patient cannot be easily aroused but respond purposefully after repeated verbal or painful stimulation (eg, purposefully pushing away the noxious stimuli)
- Ability to independently maintain ventilatory function may be impaired
- May be accompanied by partial or complete loss of protective airway reflexes
- Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate
- Cardiovascular function is usually maintained
- Should have an intravenous line placed at the start of the procedure or have a person skilled in establishing vascular access in pediatric patients immediately available
- Precordial stethoscope or capnograph for patients difficult to observe to aid in monitoring adequacy of ventilation

Patient Selection

- Traditional techniques unsuccessful in managing behavior
- Patient is ASA 1 or 2
- Patient below age of reason (pre- or uncooperative)
- Extent of treatment
- Needle phobic; excessively fearful older child
- Older child with poor experiences or coping abilities
- Distance traveled even for patients without behavior problems
- Developmental delay or handicapping condition/ medical problem

History

- Allergies/asthma/croup must be considered
- Current meds including over the counter may include depressants
- Diseases - CV, CNS, pulmonary, liver, kidney, pregnancy status
- Malignant hyperthermia, preemie on ventilator (bronchopulmonary dysplasia)
- Sleep apnea - snoring suggests tonsil/airway problem
- Previous sedations/GA/hospitalization
- Family history of diseases

Physical Assessment

- General physical condition (gait, wheelchair, coordination)
- Vital signs—HR, RR, BP

- Vital statistics - age and weight
- Airway—tonsils, neck, nose, tongue must not be potential airway obstacle
- Mouth breather/C-spine/nasal speech
- Midfacial hypoplasia
- Risk assessment - ASA status III, IV contraindicated
- Obesity
- Communication ability

Level of Sedation	Cognitive Function	Physiological Function	Monitor	Personnel
Minimal	May be impaired	Not affected	Observation only; intermittent	Not specified
Moderate	Depression; Responds to light tactile stimulation	Patent, self-correcting airway; ventilation and cardiovascular function is adequate	O2 sat HR Intermittent BP & RR (no designated period of recording) EKG & Defib available (should)	Person responsible for monitoring other than operator; may do other tasks
Deep	Depression; Cannot be easily aroused	Potential loss of airway reflexes; cardiovascular may be affected	O2 sat HR Intermittent BP & RR (recorded q5min)	Person responsible solely for monitoring

V. SEDATION ROUTES

Inhalation

- Delivery system—continuous, regulate, min 25% O₂, fail safe feature
- Scavenger system
- Equipment calibration annually
- Concentration can be titrated—very desirable characteristic
- 100% O₂ possible
- O₂ analyzer in portable systems if less than 25% O₂ can be delivered

Oral

- Most accepted by children—no needles
- Taste—syringe administration/aspiration—requires partial patient cooperation; syringe use may cause aspiration if done too rapidly

- Can't titrate—not predictable
- Onset and recovery may be prolonged/variable absorption
- Adding more medication after initial dose not recommended for any reason

Intranasal

- Easily administered to a resistant patient with just brief discomfort
- Atomizer may be preferred
- Very effective with quick onset/short acting meds
- Reduces time in office

IM

- Fear of needle and pain; injection painful
- Can't titrate, not predictable
- Use vastus lateralis/gluteus muscles
- Prolonged duration

IV

- Very predictable, can titrate
- IV may be difficult to start in children, especially overweight (to find vein)
- Rapid onset/shorter recovery
- Complications at venipuncture site include bleeding and hematoma

VI. MEDICATIONS

Nitrous Oxide

- CNS depression—minimal CV or respiratory effect
- Anxiolytic, minimal analgesia
- Induction 5 min—recovery 10 min
- 20 – 70 % concentration – open system; nasal hood
- Chronic exposure/abuse can occur by in dental professionals
- Use with local anesthesia
- Relative contraindication during wheezing (moderate-severe asthma), nasopharyngeal obstruction, TB (tuberculosis)
- Side effects of sweating, nausea, GI discomfort, vomiting @ high or rapidly varying nitrous levels

Chloral Hydrate—Sedative/Hypnotic

- CNS depression—minimal CV or respiratory effect
- Gastric irritation a side effect; unpleasant taste may need flavoring to mask taste
- Onset 30-60 min, peak 60 min, duration 5 hours; working time up to 60 minutes
- 25–50 mg/kg orally to 1 g maximum
- Metabolized to trichlorethanol in liver; excreted by kidney
- Arrhythmias in higher doses (>75 mg/kg)
- No reversal agent
- Often combined with other agent; and dose should be lowered

Diazepam (Valium)—Sedative/Hypnotic (Benzodiazepine)

- CNS depression—minimal CV or respiratory effect
- Amnesia, ataxia (acts in cortex, limbic system, thalamus, hypothalamus)
- Onset 45-60 min, peak 60 min, 1/2 life 20–40 hours
- 0.25 - 0.5mg/kg orally (<10mg total dose)
- Flumazenil reversal (dose – 0.01 mg/kg; IV preferred but can be IM or submucosal)
- Contraindication: narrow angle glaucoma
- Half-life 20-40 hrs with sedative metabolite

Midazolam (Versed™)—Sedative/Hypnotic (Benzodiazepine)

- CNS depression—minimal CV or respiratory effect
- Amnesia
- Onset 15 min, 30–40 min working time
- 0.5 – 0.75 mg/kg to 15 mg total – orally
- Flumazenil reversal (dose – 0.01 mg/kg; IV preferred but can be IM or submucosal)
- 3 - 4x potency of diazepam
- Respiratory depression with higher doses or rapid IV bolus

Hydroxyzine (Vistaril™)—Antiemetic/Antihistaminic

- CNS depression—Anxiolytic, bronchodilator
- Analgesia—dry mouth
- Onset 15–30 min, 2–4 hours duration
- 0.6 mg/kg orally
- Used often with chloral hydrate or meperidine

Meperidine (Demerol®)—Narcotic

- CNS, CV, respiratory depression—Naloxone reversal (dose 0.01-0.1 mg/kg; IV preferred but can be IM or submucosal)
- Sedation, analgesia, lowers seizure threshold
- Exercise caution in patients with pulmonary complications; head trauma, seizures, hepatic/renal disease, airway obstruction
- Onset 30 min, peak 1–2 hours, duration 2–4 hours
- 1.0 - 2.0 mg/kg orally to adult dose; 50 mg max PO, SM, IM
- Metabolized by liver, excreted by kidney
- Side effects: dizziness, xerostomia, sweating, nausea/vomiting, seizures, respiratory depression

Lidocaine (Xylocaine)—Amide Local Anesthetic

- CNS depression—minimal CV effect
- 2% vs. 1% concentration
- 4.4 mg/kg with or without vasoconstrictor
- Minimize dosage - record maximum dose prior to administration

Combinations

- Utilized for different pharmacologic and synergistic effects
- Combine techniques (e.g., oral and inhalation)
- Dosage considerations/potentiation (usually lower dosage)
- Deeper sedation may result
- Often used under 3 y/o

VII. PRE-SEDATION PREPARATION

Informed Consent

- In writing, witnesses
- Disclose the problem
- Discuss treatment, alternatives including no treatment
- Risks—those significant for decision including alternatives and no treatment

Pre-sedation Instructions

- Verbal and written
- Dietary precautions
- Postoperative behavior
- Guardian must accompany patient (2 are suggested)

Dietary Precautions

- No solid foods, non-human milk, and infant formula up to 6 hours
- No breast milk up to 4 hours
- Clear liquids up to 2 hours before procedure for children ages 6 months and older.

Day of Sedation Assessment

- NPO status/taking over the counter (OTC) medication or Rx?
- Upper respiratory infection/nasal discharge/large tonsils
- Fever/cough
- Bladder empty
- Baseline vital signs

VIII. MONITORING PRINCIPLES

- Increases patient safety
- Early recognition of potential problems
- Continuous/continual (e.g., oxygen saturation/blood pressure)
- Quieter the patient/more important the monitoring
- Know state laws covering education and monitoring

Monitoring Clinical Level of Consciousness

- Patient response to commands and tactile stimuli
- Level of conversation
- Body movement and body language
- Eyes open or closed

Monitoring Airway

- Head position—keep chin elevated
- Airway sounds—snoring - reposition head
- Respiratory sounds and rate from pre-tracheal stethoscope
- Watch oxygen saturation on pulse oximeter
- Reservoir bag movement
- Patient color, chest movement
- Capnograph is desirable for moderate and deep sedation

Monitoring Cardiovascular System

- Heart rate - stethoscope, manual, automatic devices
- Blood pressure - manual and automatic
- Small cuff – spuriously high BP value, large cuff – spuriously low BP value; use correct sized cuff

Monitoring the Respiratory System

- Chest movement—rate change from baseline
- Listen for RR through stethoscope
- Watch oxygen saturation on pulse oximeter
- Patient color, reservoir bag movement

Documentation

- A medico-legal necessity
- Pre-sedation written instructions
- Informed consent, pre treatment history must be included in record
- Current weight, base-line vital signs
- Intraoperative time based record of vitals, drugs—route, dose, time
- Patient condition at discharge—may include vitals, time of D/C
- Include N₂O/O₂ levels

Discharge Criteria

- Vital signs and airway stable; no nausea/fever
- Oriented to surroundings, recognizes guardian
- Can walk, talk, and support head
- Post treatment instructions in writing
- Guardian is available

IX. EMERGENCIES

AAPD Resource: http://www.aapd.org/media/Policies_Guidelines/RS_MedEmergencies.pdf

Operating Facility

- Full face mask with positive pressure O₂
- Emergency kit

- High speed suction
- Pretracheal stethoscope
- Sphygmomanometer (auto or manual)
- Pulse oximeter
- Other as required by state law

Risk Management

- Use familiar drugs, and techniques, and protocols
- Informed consent
- Limit use of sedation to patients who require it
- Comprehensive preoperative evaluation
- Continuous monitoring/documentation
- Have an emergency management system
- Treat high risk patients in the hospital
- Personnel trained in BLS, monitoring, emergency procedures
- Parent or caretaker must be present
- Not for kids with URI within two weeks of sedation
- NPO verified
- Rubber dam is asset
- Mouth prop assures access to oropharynx
- Judicious use of H₂O in cavity preparation

Emergency Management Principles

- Recognize the problem/stop treatment
- Remain calm
- Assess consciousness
- Initiate rescue procedures as needed (e.g., ABC)
- Get help—activate EMS
- Keep on-site treatment basic unless trained to assess and manage

Potential Emergent Situations

- Respiratory rate depression or arrest
- Upper airway obstruction
- Allergic reactions/overdose
- Vomiting
- Seizures

Emergency Management System

- Appropriate drugs—O₂, Epi, Naloxone, Analgesic, Flumazenil
- Equipment—O₂ delivery system, suction, syringes
- Trained support staff—maintain proficiency
- Emergency medical back-up

XI. ADDITIONAL READINGS AND WEB SITES

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http://www.aapd.org/searcharticles/current.asp?PUBLICATION_ID=98
2. Newman DH, Azer MM et al. When is a patient safe for discharge after procedural sedation? The timing of adverse effect events in 1,367 pediatric procedural sedations. Ann Emerg Med 42:627-635, 2003.
3. American Society of Anesthesiologists. Practice Guidelines for Sedation and Analgesia by Non-Anesthesiologists. Anesthesiology 96:1004-1017, 2002.
4. Krauss B and Green SM. Procedural sedation and analgesia in Children. Lancet 367: 766-780, 2006.
5. AAPD. *Clinical Guideline on Monitoring and Management of Pediatric Patients During and After Sedation for Diagnostic and Therapeutic Procedures*.
http://www.aapd.org/media/Policies_Guidelines/G_Sedation.pdf

Chapter 14: PAIN CONTROL

AAPD CLINICAL GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_LocalAnesthesia.pdf

- I. INDICATIONS
- II. TECHNIQUES OF LOCAL ANESTHESIA
- III. MAXIMUM RECOMMENDED DOSAGES (T)
- IV. LOCAL ANESTHETIC OVERDOSE
- V. COMPLICATIONS OF LOCAL ANESTHESIA
- VI. ANALGESIA FOR CHILDREN
- VII. ADDITIONAL READINGS

I. INDICATION

- Children feel pain, although they may not be able to report the severity or location
- Special consideration must be given to local anesthetic dosages, especially in small children
- The maximum recommended dosage of local anesthetic must not be exceeded which may preclude multiple quadrant dentistry

II. TECHNIQUES OF LOCAL ANESTHESIA

- Maxillary anesthesia
 - supraperiosteal infiltration in the mucobuccal fold
 - intrapapillary injection for palatal anesthesia
- Mandibular anesthesia
 - supraperiosteal infiltration (effective for incisors and canines and occasionally effective for minor operative procedures on primary molars)
 - inferior alveolar nerve block (effective for extensive operative procedures or oral surgical procedures on primary (or permanent) molars)
 - PDL injection (useful for single tooth anesthesia but not routinely for pediatric procedures)

III. MAXIMUM RECOMMENDED DOSAGES

Local Anesthesia

Drug	Mg/kg	Mg/lb	Absolute Maximum Dose
Ariticane	7.0	3.2	500
Lidocaine	4.4	2.0	300
Mepivacaine	4.4	2.0	300
Prilocaine	6.0	2.7	400
Bupivacaine	2.0	0.9	90
Etidocaine	8.0	3.6	400

If conscious sedation is employed, then dosage of anesthetic should be well below the MRD so that potentiation of cardiorespiratory depressant effects does not put the patient at risk.

- Epinephrine prolongs the action of the lidocaine by constricting the blood vessels in the area which increases the potential for post-treatment soft tissue trauma from biting or scratching
- Epinephrine prevents rapid systemic uptake of lidocaine so it is generally used in dentistry for children

MAXIMUM DOSE

PATIENT WEIGHT (KG/LB)	MG	NO. CARTRIDGES
10/23	44	1.2
15/34.5	66	1.8
20/46	88	2.4
25/57.5	100	2.7
30/69	132	3.6
40/92	176	4.8
50/115	220	6.1
60/138	264	7.3
70/161	300	8.3

IV. LOCAL ANESTHETIC OVERDOSE

- Causes
 - intravascular injection
 - excess dosage delivered to patient
- Effects
 - central nervous system depression seizures disorientation loss of consciousness
 - cardiovascular system

Decreased myocardial contractility

Decreased cardiac output

Cardiovascular collapse

V. COMPLICATIONS OF LOCAL ANESTHESIA

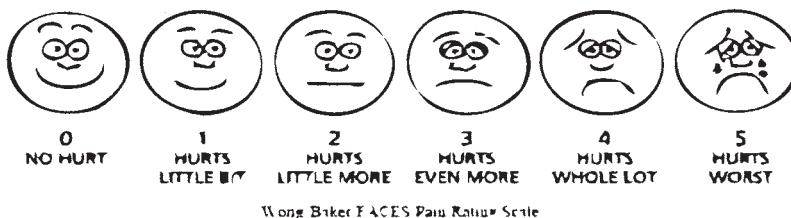
- Soft tissue injury
 - accidental lip, cheek or tongue biting or scratching from prolonged soft tissue anesthesia
 - more common in very young or developmentally disabled child
 - warn patient and parent of possible injury; advise parent to watch patient to prevent injury

VI. ANALGESIA FOR CHILDREN

- Pain assessment in the preverbal child or one with limited verbal skills
 - physiologic response: increased heart rate, blood pressure, respiratory rate
 - behavioral response:
 - persistent crying
 - crying with oral stimulation, eg, sucking, eating, etc.
 - inability or refusal to eat or drink
 - awakening from sleep

Analgesics Commonly Prescribed for Children

ANALGESIC	RECOMMENDED DOSAGE-ORAL ROUTE	ADVANTAGES	DISADVANTAGES	HOW SUPPLIED
Acetaminophen (Tylenol, Tempra, Panadol)	10-15mg/kgq4h	Antipyretic; rare side effects	No anti-inflammatory action;mild pain relief	Drops:80mg/0.8ml calibrated dropper; suspension:160mg/5ml; chewable tabs:80mg tabs; tablets:325 and 500mg tabs
Aspirin	Not recommended for children			
Ibuprofen (Advil, Motrin)	2-8mg/kg,q6-8 h	Anti-inflammatory, good relief for moderate to severe pain, antipyretic	Gastric irritant, may impair clotting	Suspension:100mg/5ml Tablets:200mg
Naproxen (Naprosyn)	3-7mg/kg,q8-10h	Anti-inflammatory, good relief for severe pain	Gastric irritant, may impair clotting, delayed onset	Suspension:125mg/ml Tablets:250,375, 500mg
Acetominophen with codeine(Tylenol with codeine)	Codeine:0.5mg/kg 3-6yrs:12mg,q4-6h 7-12 yrs:24mg, q4-6h	Good relief for severe pain, antipyretic	CNS and respiratory depression, constipation, cramping, potentiates sedative drugs effects, contraindicated with head trauma	Suspension:12mg/5ml codeine with 120mg Tylenol Tablets all contain 300mg Tylenol #1:7.5mg codeine #2:15mg codeine #3: 30mg codeine #4: 60 mg codeine

**VII. ADDITIONAL READINGS**

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3. Malamed, SF. Sedation: A guide to patient management, 4th edition, CV Mosby Co, St. Louis, 2003.

4. Moore TJ et al. Methemoglobinemia linked to topical benzocaine use. *Arch Int Med* 164:1192-1195, 2004.
5. Brown RS and Rhodus NN. Epinephrine and local anesthesia revisited. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 00:401-408, 2005.
6. Bieri D, Reeve R, Champion G et al. The Faces Pain Scale for the self assessment of the severity of pain experienced by children: development, initial validation and preliminary investigation. *Pain* 41:139-150, 1990.

Chapter 15: HOSPITAL DENTISTRY AND GENERAL ANESTHESIA

AAPD POLICIES AND GUIDELINES:

http://www.aapd.org/media/Policies_Guidelines/P_HospitalStaff.pdf

http://www.aapd.org/media/Policies_Guidelines/P_HospitalizationInfants.pdf

http://www.aapd.org/media/Policies_Guidelines/P_3rdPartySedGA.pdf

http://www.aapd.org/media/Policies_Guidelines/G_Sedation.pdf

http://www.aapd.org/media/Policies_Guidelines/G_AnesthesiaPersonnel.pdf

- I. HOSPITAL OPPORTUNITIES**
- II. REQUIREMENTS FOR MEDICAL STAFF
MEMBERSHIP AND HOSPITAL PRIVILEGES**
- III. GOALS OF GENERAL ANESTHESIA**
- IV. PRE-OPERATIVE DENTAL EXAMINATION AND
CONSULTATION (T)**
- V. PRE-ANESTHETIC PHYSICAL EXAMINATION**
- VI. SURGERY DOCUMENTATION**
- VII. OPERATING ROOM PROTOCOL**
- VIII. POST-SURGICAL ORDERS**
- IX. OPERATIVE REPORT**

- X. DISCHARGE CRITERIA (T)**
- XI. POST-OPERATIVE INSTRUCTIONS (RX)**
- XII. POST-SURGICAL COMPLICATIONS**
- XIII. ADDITIONAL READINGS**

I. HOSPITAL OPPORTUNITIES

- Provide essential services to patients within an operating room setting
- Provide consultative and emergency services
- Participate within the organizational structure through committee memberships of either clinical or administrative purpose

II. REQUIREMENTS FOR MEDICAL STAFF MEMBERSHIP AND HOSPITAL PRIVILEGES (CREDENTIALING)

- Graduation from an accredited dental school
- A state dental license where the facility is located
- High ethical and moral standards
- Advanced training may be necessary
- Professional liability insurance
- Some hospitals require board certification

Medical-Dental Staff Member Responsibilities

- Patient care within the limits of approved clinical privileges
- Participation in emergency department on-call rotations
- Timely medical records completion
- Compliance with the rules and regulations of the medical staff and the policies and procedures of the hospital

III. GOALS OF GENERAL ANESTHESIA

General Anesthesia is used to provide safe and comprehensive dental care for the pediatric patient with behavior, medical, or other problems that preclude treatment in the office setting by eliminating cognitive, sensory, and skeletal motor activity in order to facilitate the delivery of quality comprehensive diagnostic, restorative, and /or other dental services.

Indications

- Patients with certain physical, mental, or medically compromising conditions
- Patients with dental restorative or surgical needs for whom local anesthesia is ineffective because of acute infection, anatomic variations, or allergy
- The extremely uncooperative, fearful, anxious, physically resistant or uncommunicative child or adolescent with substantial dental needs and no expectation that the behavior will soon improve
- Patients who have sustained extensive orofacial and/or dental trauma
- Patients with immediate comprehensive oral/dental needs who otherwise would not receive comprehensive dental care
- Patients requiring dental care for whom the use of general anesthesia may protect the developing psyche and/or reduce medical risks

Contraindications

- General anesthesia risk
- Respiratory infection

- Active systemic disease with elevated temperature
- NPO guideline violation
- A healthy cooperative patient with minimal dental needs

Selection of Operating Room Facility:

Outpatient Ambulatory (Surgery Center/Hospital Care Area)

Patient selection-healthy and free of significant medical disorders

ASA I/II

Advantages

- more efficient
- better tolerated by family and providers
- more patient friendly
- decrease cost for consumers and 3rd party payers

Disadvantages

- requires reliable parents/guardians

Inpatient

Patient Selection

- ASA III and above
- patients with significant medical conditions
- children from remote areas with rampant decay
- questionable parental compliance with pre/post operative instructions
- possible need for 23 hour admission

IV. PREOPERATIVE DENTAL EXAMINATION AND CONSULTATION

Initial Dental Examination

- Obtain a complete and comprehensive health history to include medications and abnormal medical findings
- Determine the family history and social background
- Identify the chief complaint
- Obtain a complete oral, clinical, and radiographic exam, if possible

Parent/guardian Consultation

- Discuss the reason/need for general anesthesia
- Risks/benefits associated with general anesthesia
- Anticipated post-operative behavior and limitation of activities
- Cost estimate for the procedure
- Need for medical consultation
- Need for a physical examination
- Need for laboratory tests as determined by a complete medical history and physical
(See Resource Section for lab values)
- Third party payment pre-authorization

- Admission process to the hospital
- Pre-surgical and post-surgical dietary precautions.

NPO Guidelines

	6-36 Months	>36 Months
Clear Liquids	2 Hours	2 Hours
Breast Milk	6 Hours	NA
Formula	6 Hours	NA
Solids/Milk	6 Hours	8 Hours

Informed Consent

- Each patient, parent and or other responsible individual is entitled to be informed regarding benefits, risks, and alternatives to general anesthesia and to give written consent
- Patient record must document appropriate consent obtained per state/institutional requirements at the initial exam and reaffirm the day of the surgery

Child's Psychological Management:

Child/parent anxiety over impending procedures may be modified by:

- Involving the child on the operating room tour
- Allowing the child to bring along a favorite doll or toy
- Giving pre-induction sedation
- Providing a non-threatening environment
- Giving post-sedation as needed
- Allowing parents to rejoin their children as early as possible in the recovery area.
- Reduce parent stress
 1. detailed pre-surgery consult
 2. prior tour of OR facility
 3. updating parents of child's status throughout the procedure

Pre-Surgical Evaluation/Work Up:

Health history:

- Age in years and months
- Name, address, and telephone number of the child's pediatrician or family physician
- Allergies and previous allergic or adverse drug reactions, i.e., latex, surgical tape, drugs, food
- Current medications (prescription, over the counter, and herbal) including dose, time, route, and site of administration
- Diseases, disorders or physical abnormalities and pregnancy status
- Child's previous hospitalizations and anesthesia/ surgical procedures
 1. Patient's age and type of procedure performed
 2. Complications
 3. Recovery
- Family history of diseases or disorders especially those which might impact sedation and general anesthesia
- Perinatal Problems
 1. Need for prolonged hospitalization (date, purpose, course)
 2. Need for supplemental oxygen or intubation
 3. History of apnea or bradycardia

Systems Review:

Respiratory

- exposure to environmental tobacco smoke
- obstructive apnea, breathing irregularities, and cyanosis
- history of snoring or obstructive breathing
- recent or recurrent upper respiratory infection
- history of laryngotracheitis (Croup)
- asthma or wheezing

Cardiovascular

- murmurs, arrhythmias, syncope, cyanosis

Gastrointestinal

- reflux/vomiting
- diet, feeding difficulties, appetite, failure to thrive
- liver disease

Neurologic

- seizure disorders, convulsions, paralysis
- developmental delay, neuromuscular disease
- headaches, mental conditions

Hematologic

- anemia, bleeding disorders, hemoglobinopathies
- prior blood transfusions

Renal

- renal insufficiency, oliguria, anuria
- fluid and electrolyte imbalances

Immunologic

- immunocompromised

Psychosocial

- drug, alcohol, tobacco abuse
- family dysfunction, physical/sexual abuse
- previous surgical experiences
- psychosis, anxiety, depression

American Society of Anesthesiologist Risk Assessment Classification

Class I	A normally healthy patient with no organic, physiologic, biochemical or psychiatric disturbance or disease
Class II	A patient with mild-to-moderate systemic disturbance or disease
Class III	A patient with severe systemic disturbance or disease.
Class IV	A patient with severe and life-threatening systemic disease or disorder
Class V	A moribund patient who is unlikely to survive without the planned procedure
Class VI	A declared brain dead patient whose organs are being removed for donor purposes

V. PRE-ANESTHETIC PHYSICAL EXAMINATION

Within 30 days of the procedure

Vital Signs

- Temperature, Blood Pressure, Pulse (Heart Rate), Respiratory Rate

Measurements

- Height, Weight

General Observations

- alert, dull, chronically ill, in distress, color, nutrition, gait

Organ Systems

Head and Neck

- shape, size, proportion, and symmetry of head and face

Eyes

- position, prominence, blinking, movement
- eyelids, conjunctiva, cornea, pupils

Ears

- symmetry, position, tympanic membranes

Nose

- shape, symmetry, position
- secretions, mucosa, nasal airway patency

Mouth and Throat

- lips, tongue, gingiva, mucosa, palate, teeth
- tonsils, salivary secretions

Lungs and Chest

- auscultation, percussion, palpation
- chest development, symmetry of movement

Cardiovascular

- blood pressure, pulses, size
- rate, rhythm, sound quality, murmurs or thrills

Abdomen

- inspection, palpation, auscultation
- masses, viscera

Genitalia

- male testes
- female introitus

Skin

- color, pigmentation, cyanosis, edema, petechiae, elasticity
- lesions (macules, papules, vesicles, pustules, ulcers)

Lymph Nodes

- location, enlargement, tenderness, symmetry

Skeleton (extremities, muscles, joints, spine)

- proportionality, posture, stance
- development, tenderness, spasm

Nervous System

- state of consciousness, reflexes
- balance, gait, paralysis

Summary List of Problems/Tentative Diagnosis

VI. SURGERY DOCUMENTATION

Dates Required

- Date of the surgery
- Date of preoperative anesthesia consultation

Surgery Location

- Hospital OR
- Ambulatory surgery center

Type of Admission

- OPPA (Out Patient Possible Admit)
- OPOB (Out Patient Observation: 23 Hours)
- OPDA (Out Patient Definite Admit)
- Inpatient admission

Patient Information

- Name, medical record number, telephone number, admitting physician

Completed Forms

- Procedure scheduling request form
- History and physical (legible, dated, and signed)
- Surgical consent (signed and dated)
- Pre-operative physician's orders (as applicable)

Additional Forms (as applicable)

- Laboratory test results
- Consults
- Power of attorney/guardianship
- Charts/records from other facilities

VII. OPERATING ROOM PROTOCOL (DAY OF SURGERY)

Pre-Op Evaluation

- Weight in kilograms
- Review patient's history and physical
- Vital signs, (blood pressure, pulse, and respiratory rate)

- Airway evaluation
- NPO status

Medical Chart Entries

- Admitting Note
 1. Current medical status
 2. Diagnosis
 3. Proposed treatment
- Verification of pre-anesthetic medications given and NPO status
- H&P (legible, dated, and signed)
- Surgical consent (reaffirmed, signed and dated)
- Pre-operative physician's orders (as applicable)
- Lab tests results, consults, and guardianship (if applicable)

Dental Personnel

- All operating room personnel must follow OSHA guidelines
- Dental procedures are considered clean rather than sterile procedures
- Wear appropriate attire to prevent contamination
 1. Scrubs, surgical gowns, head and foot covers
 2. Eyeglasses and/or face shield, surgical mask for the nose and throat
- Standard "Scrub" technique and wear sterile gloves
- Use barrier technique to prevent cross-contamination

Anesthesia Protocol

- Consider requests by the surgeon (naso-endotracheal versus oral intubation)
- Start the IV, perform the intubation, stabilize the tube
- Assess and maintain patient hydration throughout the procedure
- Secure monitoring equipment
- Precordial stethoscope, automatic blood pressure cuff
- Electrograph leads, temperature probe
- Pulse oximeter, and capnograph monitors
- Eye protection to include taping
- Administer medications IV (antibiotic premedication, use American Heart Association recommendations)

Dental Preoperative Protocol

- Obtain appropriate X-rays prior to scrub and throat pack
 1. Lead protection for the patient and all involved personnel
 2. Digital X-rays preferred due to low radiation and immediate feedback
- Patient Protection
 1. Special care to protect the patient's eyes (tape, eye guards)
 2. Shoulder roll and pliant head rest (stabilization)
 3. Padding for pressure points (especially the special needs patients)
 4. Secure and stabilized naso-endotracheal tube
 5. Cleanse the perioral area and dry
 6. Position the surgical sheet and drape accordingly

- Throat Pack
 1. Prevent anesthetic gases backflow and prevent debris from falling into the nasopharynx
 2. Prior to insertion, thoroughly irrigate and suction the oro/nasopharynx
 3. Use a moistened sterile gauze or vaginal pack and pack from side to side to occlude the entire pharyngeal area (do not disturb the tube)
 4. Indicate both oral and written throat pack placement and time to ensure removal upon procedure completion

Dental Operative Procedure

- Perform a thorough debridement, prophylaxis, and detailed oral exam
- Instruments are the same as used in outpatient procedures
- Mouth prop used to maintain opening (be careful not to impinge the lips or tongue)
- Quadrant isolation with a rubber dam is preferred
- Local anesthetic may be used to minimize post-operative pain and bleeding
- Provide treatment that will give the greatest longevity and require the least maintenance
- Possible intra-operative complications
 1. Dislodged or obstructed endo/nasotracheal tube
 2. Disconnected or infiltrated IV
 3. Nasal bleeding
 4. Lips and/or tongue edema
- Procedure Completion
 1. Give a 10-15 minute warning prior to completion to prepare anesthesia for extubation and recovery room personnel for patient arrival.
 2. After thorough irrigation and suction, remove the throat pack and indicate the time both oral and written
 3. Post-operative pain control usually IV NSAID or narcotic (administered by anesthesia)

VIII. POST-SURGICAL ORDERS

- Maintain the IV (Lactated Ringer's or D5W) 2-3ml/kg/hr until the patient is stable OR
- Use 4:2:1 Rules for replacement fluids:
 $1^{\text{st}} \text{ } 10 \text{ kg} \times 4 \text{ ml}$
 $2^{\text{nd}} \text{ } 10 \text{ kg} \times 2 \text{ ml}$

Remaining kgs \times 1 ml each

(IV rate = ml/hr)

EXAMPLE: 25 kg child would receive $40 + 20 + 5 = 65\text{ml/hr}$

- Discontinue IV fluids once the child is fully awake, alert, and has taken PO fluids
- Encourage PO intake of fluids once the patient is alert and awake
- PO intake may be delayed if extractions or oral surgery was performed
- Clear liquids are most commonly ordered
- Pain medications if none were given intra-operatively
- Vital signs

IX. OPERATIVE REPORT

Required within 24 hours

- Patient's name and hospital number and date of birth
- Date of the procedure
- Attending doctor and assistant's names
- Pre-operative and post-operative diagnoses
- Surgical procedures
- Surgical indications
- Anesthesia
 1. Preoperative medications
 2. OR room and time
 3. Type of intubation
 4. Anesthetic agents
 5. IV site and fluids
- Dental Procedures
 1. Radiographs taken and time
 2. Draping procedure and throat pack placement and time
 3. Dental procedures for each tooth (restorations, pulp therapy, extractions)
 4. Surgical procedures and appliance impressions
 5. Dental prophylaxis and fluoride application
 6. Type and amount of intraoperative fluids utilized
- Estimated blood loss and hemostasis
- Patient condition at the conclusion of the procedure
- Patient condition on arrival in recovery
- Prescriptions written
- Post-operative instructions
- Prognosis

X. DISCHARGE CRITERIA

- Cardiovascular function is satisfactory and stable
- Airway patency uncompromised and satisfactory
- Patient easily aroused with protective reflexes intact
- Patient adequately hydrated
- Pain and bleeding controlled
- No nausea or vomiting
- Patient can sit unaided, and ambulate with minimal assistance
- Pre-sedation level of responsiveness for the very young and special needs patient
- Responsible adult(s) available

ALDRETE POST ANESTHESIA RECOVERY SCORE**>9 Required for Discharge**

Activity – Voluntarily or on Command	
Moves 4 Extremities	=2
Moves 2 Extremities	=1
No Motion	=0
Breathing	
Deep Breath, Cough, Cry	=2
Dyspnea or Shallow	=1
Apnea	=0
Blood Pressure	
Within 20% of Preanesthetic Value	=2
Within 20-50% of Preanesthetic Value	=1
50% Outside Preanesthetic Value	=0
Color	
Pink	=2
Pale, Blotchy, Dusky	=1
Cyanotic	=0
Consciousness	
Fully Aware, Responds	=2
Arouses to Stimulus	=1
Unresponsive	=0

XI. POSTOPERATIVE INSTRUCTIONS (ORAL AND WRITTEN)

- Diet/Activity
 1. Clear liquids 3-4 hours post op
 2. Soft diet day of surgery if clear liquids tolerated
 3. Diet as tolerated > 24hours post operative
- Limited activity day of surgery
- Oral hygiene instructions
 1. Use moistened gauze or Toothettes 1-2 days post-op
 2. Regular brushing and flossing by 3-4 days post-op
- Pain, nausea, vomiting management
 1. Children's Tylenol/Motrin q4-6h prn for pain

Tylenol (acetaminophen): 10 mg/kg q 4-6 h, max daily 65 mg/kg

Motrin (ibuprofen): 10 mg/kg q 4-6 h, max daily 40 mg/kg

2. Phenergan suppositories or Tigan for persistent nausea and vomiting
Phenergan (promethazine): .25 mg/kg to .5 mg/kg PR, max 25 mg
Tigan (trimethobenzamide): <15 kg - 100 mg tid/qid (suppository)

- 24 hour phone contact number if post-op problems
- Follow-up appointment as indicated

XII. POST-SURGICAL COMPLICATIONS

- Immediate post anesthetic (recovery room)
Nausea, vomiting, croup, hypoxia, bleeding, and laryngospasm
- Post discharge
Persistent emesis (Rx antiemetics eg; Phenergan)
- Fever
 1. Low grade is common (treat with acetaminophen)
 2. >38.5 C possible dehydration or infection; Call the dental provider
 3. Moderate to severe post-operative pain (use higher levels of analgesia)
 4. Sore throat (use ice chips or Popsicles)

XIII. ADDITIONAL READINGS

1. American Academy of Pediatric Dentistry Reference Manual. Guidelines on the Elective Use of Minimal, Moderate and Deep Sedation and General Anesthesia for Pediatric Dental Patients. Pediatr Dent 2005; 27(suppl):110-118.
2. McDonald R, Avery D, Dean J. Dentistry for the Child and Adolescent. 8th Edition 2004 Mosby.
3. Munro H. Postoperative nausea and vomiting in children. J Perianesthesia Nursing 2000;15(6): 401-7.
4. White H, Lee JY and Vann WF. Parental evaluation of quality of life measures following pediatric dental treatment using general anesthesia. Anesth Prog 50:105-110, 2003.

Chapter 16: MEDICAL EMERGENCIES

- I. PREPARATION FOR EMERGENCIES**
- II. PREVENTION OF EMERGENCIES**
- III. MANAGEMENT OF EMERGENCIES-GENERAL PRINCIPLES**
- IV. COMMON MEDICAL EMERGENCIES**
- V. SUMMARY (T)**
- VI. ADDITIONAL READINGS**

I. PREPARATION FOR EMERGENCIES

- BLS training and retraining
- ALS training and retraining
- Emergency medications
 - Oxygen
 - Important to have portable oxygen system separate from central system capable of delivering high flow 100% O₂ for 30 minutes
 - Can use central system via nasal hood, face mask or blow-by
 - Used for all medical emergencies to increase inspired oxygen concentration
 - Epinephrine 1:1000
 - Anaphylaxis
 - Acute asthma resistant to inhaled bronchodilator
 - Sympathomimetic: α, β1, β2 effects
 - EpiPen® (0.3mg), EpiPen Jr.® (0.15mg)
 - Albuterol
 - Respiratory distress (not obstructive)
 - Acute asthma attack
 - β₂ agonist (bronchodilator)
 - Metered dose inhaler
 - Suspension for nebulizer
 - Proventil®, Ventolin®
 - Glucose
 - Hypoglycemia
 - Tube of sugar
 - ONLY IF CONSCIOUS AND ABLE TO MAINTAIN GAG REFLEX
 - Glucagon
 - Hypoglycemia/ Insulin shock if unconscious
 - Converts hepatic glycogen to glucose
 - 1 mg with 1 ml vial of diluent
 - IM
 - Diazepam, Midazolam
 - Status epilepticus
 - Benzodiazepine
 - Sedative, anticonvulsant, anxiolytic, amnestic
 - Diastat® rectal delivery 5 or 10 mg prefilled system
 - Midazolam 5 mg/ml ampule for IM

Diphenhydramine (Benadryl®)

- Allergic reactions
- Antihistamine with anticholinergic and sedative side effects
- 12.5 mg chewable tabs or liquid
- 25 mg caps
- 50 mg/1 ml ampule or Tubex® cartridge for deep IM
- Aspirin
 - MI
 - Anticoagulant (inhibits platelet aggregation), analgesic, antipyretic, anti-inflammatory
 - Baby aspirin (80 mg) chewable

- Nitroglycerin
- Chest pain (angina pectoris)
- Vasodilator- ↓myocardial O₂ demand (decreases preload and afterload), - ↑O₂ supply (dilates coronary arteries)
- 0.4 mg sublingual tab or spray
- Nitrous Oxide
 - MI
 - Analgesic, sedative
 - 30- 50% mixed with oxygen via nasal hood or face mask
- Emergency Equipment
 - Portable oxygen tank with regulator (E cylinder)
 - Automated external defibrillator (AED)
 - Pediatric and adult bag-valve-masks- (Ambu® bag)- used to artificially ventilate the patient in severe respiratory distress or respiratory arrest
 - Pocket mask- easier to use than the Ambu bag when ventilating a patient and more hygienic than mouth to mouth ventilation
 - Pediatric and adult non-rebreathing masks- used to supplement inspired oxygen concentration in spontaneously breathing patients
 - Oropharyngeal/ nasopharyngeal airways in various sizes- used to assist the unconscious or semi-conscious patient in maintaining an open airway
 - Glucometer
 - BP cuffs and stethoscope
 - Pulse oximeter
 - Tuberculin syringes, 1" needles, Tubex® injector, alcohol gauze
 - Nebulizer chamber
 - All equipment and meds together in readily accessible portable cart/box
 - Staff should know location and use
 - Check regularly for use and expiration dates

II. PREVENTION OF EMERGENCIES

- Thorough medical history
- At each visit check Hx (any recent problems?)
- Pt. to bring meds (bronchodilator)
- Never treat a stranger
- Refer or treat in hospital when appropriate
- Consider rescheduling if acute exacerbation likely
- Avoid precipitating acute event
- Reduce stress

III. MANAGEMENT OF EMERGENCIES — GENERAL PRINCIPLES

- Stop treatment and assess situation
 - Base assessment on medical history, recent events and clinical signs and symptoms
- Position patient
 - If conscious, position of comfort
 - If unconscious, position to increase cerebral blood flow, supine with legs levated; or lateral recumbent to prevent aspiration

- If in status epilepticus, position to protect patient
- Activate EMS
 - Staff member should call and inform emergency personnel as to nature of emergency, age of patient., location, emergency treatment being administered
- ABC's of life support/ AED
 - Airway- check for patency, open if needed with head tilt, chin lift
 - Breathing- check respirations for quality, rate, depth, use of accessory muscles, sounds
 - Circulation- check pulse for quality, rate, adequacy of perfusion
 - Apply AED
 - Treat as you go as per BLS guidelines
- Supplemental oxygen
 - Blow by oxygen
 - Nasal hood
 - Non-rebreather mask
 - Bag- valve- mask
 - 100% O₂
- Calm, reassure and comfort
 - Stress increases oxygen demand and worsens prognosis
- Vital signs (monitor and record)
 - If appropriate, monitor and record every 5 minutes:
 - BP
 - Pulse (rate and quality)
 - Respirations (rate and quality)
- Drugs if appropriate (limited ALS interventions)

IV. COMMON MEDICAL EMERGENCIES

- Syncope
- Airway obstruction
- Hyperventilation syndrome
- Respiratory distress/ acute asthma
- AMS (altered mental status)/ CVA (stroke)/ TIA (transient ischemic attack)
- Chest pain (angina pectoris or MI)
- Cardiac arrest
- Allergic reactions
- Seizures
- Hypoglycemia
- Local anesthetic overdose

1. Syncope

Prevention

- Reduce pain and anxiety
- Upright patient slowly
- Hypoglycemic patients should have light meal pre-appointment

Recognition

- Vasovagal- fear, pain, anxiety
- Orthostatic- rising quickly from supine
- Metabolic- hypoglycemia
- Transient, sudden loss of consciousness due to transient cerebral ischemia
- Nausea, pallor, diaphoresis
- Tachycardia followed by bradycardia and hypotension

Management

- Stop tx, assess and recognize
- Position to increase cerebral blood flow
- BLS with supplemental O₂
- Determine cause (rapid glucose test)
- Treat if hypoglycemic or if in doubt
- Vital signs
- If not self-limiting, activate EMS
- Physician referral

2. Airway obstruction**Prevention**

- Carefully titrate sedative drugs and monitor respiration
- Use rubber dam, gauze drapes, proper positioning

Recognition

- Soft tissue obstruction- snoring, hypoxemia, chest retraction, cyanosis
- Foreign body obstruction- coughing, hypoxemia, chest retraction, cyanosis

Management

- Stop tx, position patient
- BLS, head tilt, chin lift, abdominal thrusts for foreign body, supplemental O₂ for soft tissue obstruction
- EMS transport- all post choking patients should be transported for evaluation

3. Hyperventilation syndrome**Prevention**

- Reduce pain and anxiety
- Sedation

Recognition

- Rapid, shallow breathing, confusion, dizziness, parasthesia (numbness, tingling), carpo-pedal spasm (cramping of hands or feet)
- Differentiate from diabetic acidosis (history and glucometer)

Management

- Stop treatment, position patient
- BLS

- Calm, comfort and reassurance
- Vital signs
- Have patient hold breath for 10 second intervals
- Consider O₂ mask (instead of paper bag)
- EMS if no resolution

4. Acute asthma

Prevention

- Check history (emphysema, COPD, frequency, duration, last episode, meds)
- Have patient's meds ready
- Reappoint if necessary
- Reduce pain and anxiety

Recognition

- Hyperactive airway triggered by allergy, exercise, stress, URI, irritants
- Bronchospasm, mucosal edema, mucous plugging, expiratory wheezing, productive cough, chest tightness

Management

- Stop treatment, calm and position patient
- BLS with supplemental O₂
- Inhalant albuterol 1-2 puffs or nebulizer dose (may be repeated after 5 min.)
- If severe asthma, epi 1:1000: EpiPen ®(0.3mg) or EpiPen Jr.® (0.15mg)
- Vital signs
- Activate EMS if needed or physician referral

5. AMS/CVA/TIA

Recognition

- Weakness, confusion, dizziness, aphasia, dysphagia, nausea, paralysis, loss of consciousness

Management

- Recognize, stop treatment, position patient
- BLS with supplemental O₂
- Rapid glucose test- treat hypoglycemia
- EMS
- Vital signs

6. Chest pain/Angina pectoris/MI

Prevention

- Reduce pain and anxiety
- Supplemental oxygen
- Sedation (nitrous oxide)

Chest pain/Angina pectoris- Recognition

- History of angina
- Chest pain, may radiate to anywhere above waist
- Stress, anxiety $\Rightarrow \uparrow$ myocardial oxygen demand, insufficient blood supply to myocardium
- Chest pain relieved by rest, nitro
- If no history of angina or patient claims this episode is atypical, treat as MI

Myocardial infarction- Recognition

- \downarrow blood supply to cardiac muscle causing infarct, ASHD (atherosclerotic heart disease)
- No prior history of angina
- Severe, possibly radiating chest pain not relieved by rest, nitro
- Rapid, thready pulse, palpitations, dyspnea, diaphoresis, weakness, feeling of impending doom

Chest pain/Angina pectoris- Management

- Recognize, stop treatment, position patient
- BLS with supplemental O₂
- Calm, comfort and reassurance
- Vital signs
- Nitro 0.4 mg SL tab or spray (may be repeated q 5 min to max of 3 times) only if systolic BP > 100
- Consider N₂O
- If no relief after 3 doses of nitro, assume MI

Myocardial infarction- Management

- Recognize, stop treatment, position patient
- BLS with supplemental O₂
- Call EMS
- Calm, comfort and reassurance
- Vital signs
- 2 baby aspirin (160 mg) chewable
- Nitro 0.4 mg SL tab or spray if systolic BP > 100
- Nitrous oxide 50% until EMS arrival

7. Cardiac arrest

Recognition

- Unconscious, no pulse

Management

- Recognize, call for EMS
- BLS with supplemental O₂
- AED, defibrillate if indicated

8. Allergic reactions

Prevention

- Identify patients
- Avoid offending agents

Recognition

- Hypersensitivity due to allergen
- Pruritis and urticaria
- Dyspnea, wheezing
- Pallor, cyanosis
- Tachycardia leading to weak pulse
- Hypotension
- Eventual respiratory and cardiovascular arrest

Management

- Recognize, stop treatment, calm and position patient
- BLS with supplemental O₂
- Vital signs
- Benadryl 25-50 mg PO or deep IM (1 mg/kg)
- Epi 1:1000 IM if anaphylactic: EpiPen ® (0.3mg) for 5 yrs and older, EpiPen Jr.® (0.15mg) for younger
- Repeat epi every 5-10 min. as needed
- Activate EMS, or physician referral if mild and self-limiting

9. Seizures

Prevention

- Medical history (meds)
- Avoid medication overdose
- Reduce stress

Recognition

- Transient alteration of consciousness, behavior, autonomic function, sensation or motor activity caused by sudden, transient disturbances of cerebral function
- Origin is either idiopathic or acquired (fever, infection, head trauma, metabolic disorders, drug overdose)

Grand Mal Seizures-Recognition

- Tonic-clonic
- Prodromal phase (30% of patients have aura)
- Ictal phase- tonic contractions of extremities followed by clonic movements
- Postictal phase- unconsciousness leading to confused awakening (most dangerous phase)

Management

- Position, prevent injury
- BLS with supplemental oxygen

- Monitor vital signs
- Rapid glucose test- treat hypoglycemia
- Status epilepticus: Diazepam 0.3 mg/kg to 10 mg max PR or midazolam 5-10 mg IM
- EMS or physician referral

10. Hypoglycemia

Prevention

- Appropriate medical management of patient
- Light food prior to appointment

Recognition

- Palpitations, diaphoresis, confusion leading to unconsciousness
- Rapid glucose test to differentiate from other causes of syncope (serum glucose <50mg/100ml)
- Type 1 diabetics or other metabolic disorders
- Fasting

Management

- Stop treatment, position patient
- BLS with supplemental oxygen
- Rapid glucose test
- If conscious- sugar PO
- If unconscious- glucagons 1 mg IM
- Monitor vital signs
- Activate EMS

11. Local anesthetic or other drug overdose

Prevention

- Know maximum dose based on body weight and health
- Use smallest dose for needed effect
- Aspirate local anesthetic

Recognition

- Local anesthetic overdose- CNS excitation followed by seizure and CNS depression
- Unconsciousness
- Cardiorespiratory arrest

Management

- Recognize, stop treatment, position patient
- BLS with supplemental O₂
- EMS
- Monitor vital signs

12. Fluoride toxicity or other poison ingestion

Prevention

- Know maximum dose based on body weight and health
- Use suction and position patient appropriately
- Use smallest dose needed
- Never leave child alone in room

Recognition

- History of ingestion
- Nausea, vomiting, abdominal pain
- Excessive salivation, abnormal taste
- Tremors, weakness, convulsions
- Shallow respirations

Management

- Recognize, stop tx, position patient
- CALL POISON CONTROL IMMEDIATELY: **(800) 222-1222** from anywhere in the U.S. Information needed: pt's age, weight, condition, time and amount ingested. This number can be used for emergency or for information.
- BLS with supplemental O₂
- EMS
- DO NOT GIVE IPECAC!
- Monitor vital signs

V. SUMMARY

- Be prepared
- Prevent problems
- Recognize emergency, position patient
- BLS with supplemental oxygen/ AED
- Meds when appropriate
- Monitor vital signs
- ALS
- EMS or MD referral

Summary of BLS ABCD Maneuvers for Infants, Children, and Adults

MANEUVER	ADULT Adolescent and older	CHILD 1 year to adolescent	INFANT Under 1 year of age
ACTIVATE Emergency Response Number (lone rescuer)	Activate when victim found unresponsive If asphyxial arrest likely, call after 5 cycles (2 minutes) of CPR	Activate after performing 5 cycles of CPR For sudden, witnessed collapse, activate after verifying that victim unresponsive	
AIRWAY	Head tilt-chin lift Suspected trauma, use jaw thrust		
BREATHS Initial	2 breaths at 1 second/breath	2 effective breaths at 1 second/breath	
Rescue breathing without chest compressions	10 to 12 breaths/min (approximately 1 breath every 5 to 6 seconds)	12 to 20 breaths/min (approximately 1 breath every 3 to 5 seconds)	
Rescue breaths for CPR with advanced airway	8 to 10 breaths/min (approximately 1 breath every 6 to 8 seconds)		
Foreign-body airway obstruction (conscious)	Abdominal thrusts	Back slaps and chest thrusts	
CIRCULATION Pulse check (≤ 10 sec)	Carotid	Carotid or femoral	Brachial or femoral
Compression landmarks	Center of chest, between nipples		Just below nipple line
Compression method Push hard and fast Allow complete recoil	Heel of 1 hand, other hand on top	2 Hands: Heel of 1 hand with second on top or 1 Hand: Heel of 1 hand only	1 rescuer: 2 fingers 2 rescuers: 2 thumb-encircling hands
Compression depth	1½ to 2 inches	Approximately 1/3 to ½ the depth of the chest	
Compression rate	Approximately 100/min		
Compression- ventilation ratio	30:2	1 rescuer: 30:2 2 rescuers: 15:2	
DEFIBRILLATION (AED)	Use adult pads. Do not use child pads/ child system. For out-of-hospital response may provide 5 cycles/2 minutes of CPR before shock if response >4 to 5 minutes and arrest not witnessed.	Use child pads/child system if available. If child pads/system not available, use adult AED and pads. Use AED as soon as available for sudden collapse and in-hospital. After 5 cycles of CPR (out- of-hospital).	No recommendation

(Newborn/Neonatal Information Not Included)

“Highlights of the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.” Currents in Emergency Cardiovascular Care Winter 2005-2006; 16(4): 15.

VI. ADDITIONAL READINGS

1. American Academy of Pediatrics. Poison Treatment in the Home. Pediatrics 112:1182-1185, 2003.
2. Hazinski MF,Nadkarni VM et al. Major changes in the 2005 AHA Guidelines for CPR and ECC. Circulation 112:IV206-211, 2005.

Chapter 17: ALLERGIC AND IMMUNE DISORDERS

- I. ANAPHYLAXIS**
- II. ALLERGIC RHINITIS**
- III. ATOPIC DERMATITIS**
- IV. URTICARIA AND ANGIOEDEMA**
- V. HEREDITARY ANGIOEDEMA**
- VI. FOOD ALLERGY**
- VII. LATEX ALLERGY (T)**
- VIII. ASTHMA**
- IX. JUVENILE ARTHRITIS**
- X. VASCULITIDES IN CHILDREN**
- XI. SYSTEMIC LUPUS ERYTHEMATOSUS**
- XII. CONGENITAL AND ACQUIRED IMMUNODEFICIENCIES**
- XIII. ADDITIONAL READINGS AND WEBSITES**

I. ANAPHYLAXIS

DEFINITION

Anaphylaxis is an overwhelming, immediate systemic reaction due to an IgE mediated release of mediators from tissue mast cells and peripheral blood basophils. This reaction occurs rapidly and can be fatal. Anaphylactoid reactions are similar in appearance but are not mediated by IgE.

EPIDEMIOLOGY/CAUSATION

Anaphylaxis is responsible for 500-1000 fatalities yearly. The most common causes of anaphylaxis are:

- Food - especially peanut, nut, egg, milk, wheat, soy, fish and shellfish
- Medications – antibiotics, NSAIDs, local anesthetics, others
- Insects – bees, wasps, hornets, fire ants
- Latex exposure in those previously allergic (new onset becoming less common)
- Exercise
- Idiopathic

COURSE OF DISEASE

Mild reactions may occur with only:

- Scattered hives
- Pruritus
- Nausea

Significant reactions include:

- Widespread hives
- Vomiting, diarrhea
- Tongue/lip/throat swelling
- Wheeze, cough, stridor
- Anaphylactic shock
- Patients with asthma are at greater risk for a severe reaction.
- Up to 20% of patients will have a bi-phasic reaction with symptoms recurring 2-8 hours (up to 72 hours) later

DIAGNOSIS

- Other conditions may appear similar to anaphylaxis:
 - Vasovagal reaction
 - Flushing episode
 - Anxiety
 - Cardiac events
- To make the diagnosis of anaphylaxis
 - Look for a triggering event within 2 hours of onset (usually more rapid)
 - If in doubt, treat for anaphylaxis to prevent serious consequences
- **It is important to identify previously known allergies before treating a patient in the dental office**

TREATMENT

This is a medical emergency, prompt treatment is mandatory.

If the patient has a few hives, mild nausea:

- Diphenhydramine, 1-2 mg/kg (max 50 mg), repeat every 6 hours for 24 hours

For a significant reaction (widespread hives, angioedema, tongue/lip, cough, wheeze, vomiting, diarrhea):

- Monitor vital signs q 15 minutes, sooner if concerns
- IM epinephrine - 0.01 ml (0.01 mg)/kg body weight up to maximum of 0.5 ml. Give this immediately. May repeat q 15 minutes × 2 doses, then q 4h if needed
- Diphenhydramine, 1-2 mg/kg (max 50 mg) IM or IV. Give this at the same time as the epinephrine
- Corticosteroids IV – Hydrocortisone sodium succinate (Solu-Cortef) or equivalent, 1-2 mg/kg if not responding to the above medications
- Inhaled beta-adrenergic bronchodilator if pt has bronchospasm
- If there are questions about the response of the patient, call 911 immediately

For life-threatening reactions (includes throat swelling, severe asthma, arrhythmia, hypotension, loss of consciousness):

- Call 911
- Place patient supine with feet elevated and monitor vital signs closely
- Give epinephrine IM or SQ as above
- Intravenous epinephrine, 1:10,000 or 1:100,000 if condition worsens, titrated at 1 mcg/min continuous drip
- Oxygen
- Inhaled beta-adrenergic bronchodilator if pt has bronchospasm
- CPR including IV fluids

II. ALLERGIC RHINITIS

CLINICAL PRESENTATION

- seasonal or perennial in nature
- nasal congestion
- sneezing
- nonpurulent rhinorrhea
- pruritis of eyes, nose, and palate

ETIOLOGY AND PATHOGENESIS

Inflammation of the nasal mucous membranes resulting from an IgE-mediated allergic reaction to the protein/glycoprotein of inhaled aeroallergens.

DIAGNOSIS

- Focused history
- Physical examination with correlation of symptoms with positive skin-prick tests

MANAGEMENT: 3 steps

- Avoidance of inciting allergens,
- Pharmacotherapy: intranasal corticosteroids, antihistamines, cromolyn sodium and decongestants
- Immunotherapy for patients who do not receive adequate relief with pharmacotherapy

For those with comorbid conditions (asthma, sinusitis, chronic otitis media) specific treatment is targeted to these coexisting medical conditions.

COMPLICATIONS

- Acute/chronic sinusitis
- Recurrent otitis media with hearing loss and Eustachian tube dysfunction,
- Impaired speech development in children
- Nasal polyps
- Sleep apnea
- Aggravation of asthma
- Increased likelihood of developing asthma

DENTAL CONSIDERATIONS

- None with adequate symptom relief
- In severe cases mouth breathing may predispose to orthodontic problems
- Mouth breathing without a diagnosis should alert the dentist to allergic rhinitis; refer the patient to an allergist for testing
- For sedation dentistry, if airway patency is a concern and there are signs of nasal obstruction, consult the patient's physician

III. ATOPIC DERMATITIS (eczema)

CLINICAL PRESENTATION

- Chronic dermatosis characterized by pruritis and relapsing inflammation
- Typical lesions begin acutely with erythema and excoriations triggered by scratching
- Uncontrolled itching and rash takes the chronic appearance of lichenification and hyperpigmentation without erythema
- Affects infants/ young children along the extremity extensor surfaces, cheeks, forehead and neck
- In older children/ adults lesions occur in flexural areas i.e. antecubital/ popliteal fossa

ETIOLOGY

- Distinct causal relationships are ill-defined
- Sensitization to foods or aeroallergens may contribute to presentation of eczema
- One third of cases in children may be exacerbated by at least one implicated food
- A strong familial association exists, including those with asthma and allergic rhinitis

DIAGNOSIS

- Made by history and physical examination
- 90% of cases present before age 5 years, with most resolving by puberty
- Diagnostic criteria include:
 - pruritis
 - pattern of skin involvement
 - personal/family history of atopic disease
 - young age of onset

- elevated serum IgE and total eosinophil counts, especially in children with asthma
- Many diseases present with lesions similar to atopic dermatitis
- Differential diagnosis in children:
 - neoplastic conditions
 - immunodeficiencies
 - metabolic defects

MANAGEMENT

- education
- trigger avoidance
- skin hydration (lukewarm baths+ topical emollients)
- itch control (antihistamines), and topical steroids for flares
- Newer steroid sparing anti-inflammatory therapies i.e. calcineurin inhibitor preparations are to be used with caution in children

DENTAL CONSIDERATIONS

None except for patients on high dose corticosteroids which necessitates consultation with the child's physician.

IV. URTICARIA & ANGIOEDEMA

CLINICAL PRESENTATION

- Urticaria is characterized by
 - extremely pruritic, erythematous, raised lesions affecting the superficial dermal layers that blanch with pressure
- Angioedema is similar pathologically
 - swelling is deeper
 - primarily affects the face, extremities, genitalia with occasional tongue enlargement or laryngeal edema
- Urticaria is associated with angioedema in 40% of cases
- Acute urticaria typically lasts <6 weeks with an identifiable trigger s.a. foods, medications, insect stings, infection
- Chronic urticaria lasts more than 6 weeks and mainly idiopathic

DIAGNOSIS

Depends on:

- focused clinical history
- identifying triggers
- a systems review

Further investigations and work-up include:

- blood tests to look for underlying abnormality including disorders of complement, hypo-/hyper-thyroidism, hepatitis, autoimmune diseases, malignancies
- biopsy where the clinical presentation is highly suspicious of an underlying etiology
 - lymphoproliferative neoplasms
 - connective tissue disorders

MANAGEMENT

- avoidance of triggers
- antihistamines
- in severe refractory cases oral steroids
- Other helpful medications include:
 - Montelukast, zafirlukast, colchicine, dapsone, azathioprine, cyclosporine, ketofin

DENTAL CONSIDERATIONS

- avoid treating patients in the active phase
- be aware of any medication triggers

V. HEREDITARY ANGIOEDEMA**ETIOLOGY**

- autosomal dominant disorder resulting from a deficiency in functional C1 esterase inhibitor

CLINICAL PRESENTATION

- submucous or subcutaneous edema that may be triggered by:
 - trauma
 - medical or dental procedures
 - emotional stress
 - menstruation
 - infections
 - oral contraceptives and other medications
- Edema typically lasts for 2-5 days before resolving spontaneously. Edema is characteristically:
 - non-pitting
 - tensely swollen, painful
 - not erythematous, warm or pruritic
- Areas most commonly affected:
 - lips, eyelids, tongue
 - other extremities, genitalia
- Most ominous symptoms relate to oropharyngeal and laryngeal edema resulting in:
 - airway obstruction with frank stridor occurring in 30 minutes. Typically, swelling does not extend beyond the larynx

MANAGEMENT

- Daily prophylactic anabolic attenuated androgens (danazol, stanozolol) for all patients. Occasionally antifibrinolytic agents used
- Acute attacks:
 - rapid administration of anabolic androgens
 - tracheotomy is potentially lifesaving
 - Epinephrine and antihistamines are NOT useful in managing the edema of hereditary angioedema
- Fresh frozen plasma is occasionally used prior to major surgical procedures
 - always consult with the patient's physician beforehand

- A routinely well managed patient is not a contraindication for dental treatment
- Some perioral swelling may occur following dental procedures, this should not discourage the dentist from treating these patients

VI. FOOD ALLERGY

CLINICAL PRESENTATION

- Cutaneous reactions:
—urticaria, angioedema, (not pathognomonic)
- Gastrointestinal manifestations especially in children:
—Nausea, vomiting, diarrhea, abdominal pain
- Oral symptoms:
—tongue, lip and perioral edema
—pruritis of the palate or lips
- Respiratory symptoms present as part of a generalized anaphylactic reaction:
—sneezing, rhinorrhea, nasal pruritis, bronchoconstriction, laryngeal edema

ETIOLOGY AND PATHOGENESIS

- Aberrant immune response induced by exposure to a particular food protein
- May be IgE-mediated or cell-mediated or both
- IgE mediated responses occur in 6-8% of children < 3 years of age
- Overall prevalence in the general population is ~2%
- Common food allergens in children include: eggs, peanuts, cow's milk, soy, tree nuts, fish, shellfish, wheat
- Most children outgrow their allergies by age 5 years except for the following allergies which usually persist into adulthood: peanuts, tree nuts, fish, shellfish
- Up to 35% of children with moderate to severe atopic dermatitis have confirmed food allergies

DIAGNOSIS

- thorough history taking
- a temporal association (best within 2 hours of ingestion of suspected food)
- reproducibility of symptoms on every exposure
- clinical features
- Diet history and ingredient labels need careful review.
- Skin-prick-tests (SPT) or blood radioallergosorbent tests (RAST) confirm the diagnosis of IgE-mediated food allergy

MANAGEMENT

- Avoidance is the key principle
- Read labels carefully for hidden ingredients
- Patients should carry Epi-pen at all times; instruct significant others on its proper use

DENTAL CONSIDERATIONS

- Avoid major food allergens when making dietary recommendations
- Children, especially those highly sensitized to peanuts, may react to air-borne food allergens and even to contact from someone who has recently consumed these products, an important consideration for maintaining patient safety in the dental office

VII. LATEX ALLERGY(LA)**ETIOLOGY AND PATHOGENESIS**

- LA is a reaction to certain proteins in latex rubber
- The amount of latex exposure needed to produce sensitization or an allergic reaction is unknown
- Latex products are manufactured from a milky fluid derived from the rubber tree, *Hevea brasiliensis*
- The true prevalence of sensitivity is unknown with estimates for the general population from (5-10%) and that for health care workers from 0.5-17%

CLINICAL PRESENTATION

Adverse reactions following exposure may be categorized as:

- Irritant contact dermatitis (ICD)
 - non immunological mediated dermatitis (non-allergic) characterized by:
 - dry, itchy, irritated areas of skin, usually of the hands
 - accounts for ~80% of hand dermatitis
 - causative factors:
 1. maceration and abrasion from constant glove wearing
 2. repeated hand washing and incomplete hand drying
 3. use of cleaners and sanitizers
 4. exposure to powders added to gloves
 5. other workplace chemicals and products
- Allergic contact dermatitis (ACD)
 - delayed hypersensitivity reaction caused primarily by accelerators, promoters, and antioxidants added to natural rubber latex during harvesting, processing and manufacturing
 - T-cell mediated immune response
 - clinically characterized by a rash, redness, and itching, 24-48 hours after contact with the offending products
 - rash may progress to oozing skin blisters and may spread to skin untouched by latex (similar to that of poison ivy)
 - Cutaneous manifestations are similar to ICD
 - allergy patch testing distinguishes the Type IV hypersensitivity reaction of ACD from the non allergic reaction of ICD
- Immediate allergic reaction (ICD)
 - the progression from skin rash (ACD) to more serious consequences is unknown
 - Some patients may develop ACD, then urticaria, allergic rhinitis, sneezing, scratchy throat, conjunctivitis, angioedema, wheezing, asthma (coughing, difficult breathing), and rarely anaphylaxis.
 - Immediate allergic reactions are all IgE-mediated and the hallmark symptoms are swelling, redness and itching

DIAGNOSIS OF LATEX ALLERGIC

- thorough clinical history
- skin puncture testing with a standardized latex reagent
- standardized assay testing for latex-specific IgE antibody
- provocative *in vivo* latex challenge

AT-RISK POPULATIONS:

- patients who undergo multiple surgical procedures where extensive or chronic contact of latex products occur with mucosal surfaces:
 - spina bifida, with or without myelomeningocele, complete avoidance due to high risk of allergy (18-73%)
 - spinal cord trauma
 - urogenital and gastrointestinal malformations
 - neurogenic bladder
 - hydrocephalus internus with ventriculo-peritoneal shunts
 - first surgery before one year of age
- Patients reporting any of the following symptoms on previous exposure to latex:
 1. Rhinitis
 2. Conjunctivitis
 3. Urticaria
 4. Angioedema
 5. Coughing
 6. Shortness of breath
 7. Wheezing

Obtain a complete medical history, promptly refer patients for further evaluation

- Atopic individuals
 - individuals predisposed to multiple allergies such as a family history of hay fever, asthma, dry skin, or eczema
- Occupational exposure – despite the lack of evidence-based consensus, it is generally accepted that health care workers may be at an increased risk of developing severe latex allergy compared to the general population
- Persons with food allergies
 - some of the latex proteins share a similar epitope to that of proteins found in fruits such as avocados, bananas, chestnuts, figs, kiwis, turnips, tomatoes, potatoes
 - It is estimated that a patient with a history of fruit allergy has an 11% risk of concurrent latex allergy

PREVENTIVE STRATEGIES

- General strategies:
 - use reduced-protein, powder-free latex gloves or non-latex varieties
 - use non-latex dental products
 - avoid oil-based hand creams/ lotions unless known to reduce latex-related problems
 - perform adequate hand hygiene after using latex gloves
 - frequently clean ventilation filters and areas contaminated with latex dust
- Strategies for oral health care workers:
 - ACD – a trial of reduced-protein, powder-free, additive free or latex free gloves may resolve dermatitis
 - suspected latex allergy- avoid direct latex contact until evaluated by a physician

- evidence of immediate hypersensitivity reaction to latex
 - avoid all contact with all latex products
 - avoid areas with high latex aeroallergen content
 - follow physician's instructions for dealing with allergic reactions
- Strategies for prevention of adverse reaction to latex products in patients:
 - identify allergic or high-risk patients
 - Patients allergic to latex must be treated in a latex free environment:
 - schedule for first appointment in the day
 - use latex-free gloves, other latex-free devices, latex free procedure tray
 - latex-free emergency kit

TREATMENT OF AN ACUTE ALLERGIC REACTION TO LATEX:

Immediately stop exposure to latex and follow specific treatment depending on the type of allergic reaction:

- Allergic contact dermatitis
 - apply high-potency topical corticosteroids.
- Allergic rhinitis
 - administer topical intranasal corticosteroids
- Acute urticaria
 - oral H1-receptor antagonists: one dose of Benadryl 25-50mg (adult), 1-1.25 mg/kg for children <12 years of age.
- Asthmatic reactions - see section on asthma
- Anaphylaxis – see section on anaphylaxis

LATEX DENTAL PRODUCTS AND THEIR LATEX-FREE ALTERNATIVES

Latex product	Alternative
Gloves	Vinyl, nitrile, neoprene, thermoplastic, elastomer, styrene-based copolymer, methyl methacrylate, polyurethane
Prophy polishing cups	Non-latex prophy cups, prophy brush on metal base
Dental rubber dam	Non-latex dental dam (Hygenic, Akron, OH, USA)
Orthodontic elastics	Ligature wires (3M unitek chain / Alastik ligatures), closing springs
Adhesive tape	Plastic, silk, 3M-Micropore
Anesthetic carpule	Glass ampules
Bitewing tabs	Paper loops
Impression materials containing latex	Alginate, blu-mousse, Impregum
Masks with elastic	Non-latex cone shaped and tie-on masks
Gutta percha	No good alternative, avoid protruding gutta percha through apex
Bite block	Molt mouth prop with silastic wrap
Pacifier	Silicone pacifier
Elastic ligature thread	Elastomeric thread
Stethoscope	Astroscope
Pulse oximeter probe	Non-latex oxisensor
Disposable syringes	Glass syringes

VIII. ASTHMA

DEFINITION

- Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, neutrophils, and epithelial cells
- In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and in the early morning
- These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment.
- The inflammation also causes an associated increase in the existing bronchial hyper-responsiveness to a variety of stimuli (NHLBI 1997)

EPIDEMIOLOGY

Asthma is the most common chronic medical condition of childhood. The CDC's 2003 National Health Interview Survey indicated a lifetime prevalence of asthma of 12.5%, and a current asthma prevalence of 8.5% in children \leq 18 years old. There is a difference in prevalence based on race, socioeconomic status and location. Asthma is more common in African-Americans (13.4%), and urban dwellers (7.1%), particularly those of lower socioeconomic status.

CAUSATION

Asthma symptoms are caused by airways narrowing secondary to:

- Airway muscle constriction
- Mucosal edema
- Airway mucous accumulation
- Inflammatory infiltrate in the submucosa and basement membrane thickening
- Viral infections

Triggers for this process include:

- Allergens, exercise, cold air, gastroesophageal reflux disease, tobacco smoke, strong odors, pollutants, sinusitis, stress

COURSE OF DISEASE AND PROGNOSIS

- Fifty to eighty percent of children with persistent asthma will have onset of their symptoms before they are 5 years old
- In children with early onset (<3 years old) wheezing, there are two patterns:
 1. Wheeze primarily with viral infections and resolve by the time they are 5-7 years old
 2. Persistent wheezing
- Children with early, frequent wheezing ($> 3x/year$) are more likely to develop persistent wheezing if they have:
 1. Concurrent atopic dermatitis or a parent with asthma
 2. Two or more of: allergic rhinitis, peripheral blood eosinophilia, and wheezing unassociated with viral infections

COMPLICATIONS

Comorbidities include: Allergic rhinitis, chronic sinusitis, gastroesophageal reflux disease, food allergy, atopic dermatitis.

In the dental office, the most concerning complication would be the onset of:

- Acute asthma attack -it could occur secondary to an exposure including latex, smoke, strong odors, reflux, metabisulfite, or as part of a medication reaction
- Adrenal suppression does NOT usually develop in children with < 700 micrograms of inhaled beclomethasone (or equivalent) daily, but if present, must supplement for extensive surgical procedures

DIAGNOSIS

- Chronic asthma is diagnosed by a combination of:
 1. History
 2. Physical examination
 3. Pulmonary function testing either by spirometry (≥ 6 y.o.) or peak flow measurements (≥ 4 y.o.)
- In children, the primary symptoms of asthma may be cough and dyspnea, without associated wheeze. A child with known asthma presenting with an acute asthma attack may have: chest tightness, cough, wheeze on auscultation (not required if other signs present), dyspnea, anxiety

DIFFERENTIAL DIAGNOSIS

Poorly responsive childhood asthma must be distinguished from: congenital abnormalities of the airway, cystic fibrosis, immunodeficiency diseases, gastroesophageal reflux disease, foreign body, malignancy.

An acute asthma attack should be distinguished from :

- Vocal cord/laryngeal dysfunction secondary to stress, reflux disease, mouth breathing
- Anxiety attack (Panic attack)

MEDICAL TREATMENT

Chronic asthma medications are divided into:

- Controllers, those given daily to keep asthma in control
 1. All patients with chronic asthma should be on a controlling medication
 2. First-line controller therapy is Inhaled corticosteroid (ICS)
 3. Other controllers include leukotriene antagonists, theophyllines, cromolys, long-acting beta agonists – added to inhaled corticosteroid when control not achieved with ICS alone
- Relievers, those used to relieve an acute asthma episode.
 1. Short-acting beta-agonist (albuterol)
 2. Anticholinergic (ipratropium) used occasionally in combination with beta-agonist (rarely alone)
 3. All patients with asthma should have a reliever available and should bring that to the dental appointment

MANAGING AN ACUTE ASTHMA ATTACK: This is a medical emergency.

- Get the patient into a comfortable position and help them to relax
- Check HR, RR, oxygen saturation, blood pressure (if tolerated)
- Listen for air movement. If the chest is very quiet with little air movement and/or the patient is extremely anxious, call 911
- Give the child's "rescue medication" if available. Usually, this is 2 puffs of inhaled albuterol delivered through a metered dose inhaler. The child should take one

puff, wait one minute and repeat, holding the breath 10 seconds after each puff. This may be repeated in 30 minutes

- If this is unavailable, give 2.5 mg. albuterol in 3cc normal saline, nebulized (This is available as a premixed ampule). This may be repeated in 30 minutes
- If neither of the above are available, give 0.01 mg/kg. 1:1,000 epinephrine subcutaneously (maximum 0.5 mg). This is also available as an auto-injector in 2 doses (Epi-pen® or Twinject®): 0.15 cc for children < 30 kg, and 0.3 cc for children > 30 kg. Epinephrine may be repeated in 20-30 minutes
- Administer oxygen to keep the oxygen saturation reading greater than 95%. Monitor vital signs. Tremor and increased heart rate are common side effects of short-acting beta-agonists

DENTAL CONSIDERATIONS

Oral Findings:

- Possible increased prevalence of dental caries and tooth wear
- Oral candidiasis- well documented side effect following steroid inhaler use
- Decreased salivary flow rates (SFR) with prolonged beta-adrenergic inhaler use
- Gingivitis from mouth breathing and elevated aminopeptidase / myeloperoxidase levels in the gingival crevicular fluid

Prevention:

- good oral cleanliness
- age-appropriate topical fluoride use
- healthy non-cariogenic diet
- avoidance of acidogenic drink consumption following inhaler use, especially at night-time when SFR is negligible
- drink or rinse with water after inhaler use

DELIVERING SAFE DENTAL CARE:

Medical History Review

- A child is more likely to develop an asthma attack in the dental office if their asthma is **not** in good daily control. Take a thorough medical history and review at periodic intervals. Any indication of the following is suggestive of poor asthma control and necessitates referral to the child's physician:
 1. Use of rescue medication (albuterol) > 2×/week
 2. Nighttime awakening with symptoms > 2×/month
 3. Concurrent upper respiratory illness causing asthma symptoms
- Safe dental management depends on:
 1. pulmonary status
 2. level of asthma control, i.e. , frequency of attacks, date of last asthma attack, latest ER visit, frequency of changes in medication protocol, precipitants
- Most recent scheduled dose of anti-asthma medication to be taken before treatment
- Patient may need steroid supplementation only prior to **major** dental treatment: consult with the patient's physician if: systemic glucocorticoids were used in the last month, the patient had >4 brief oral steroid courses in the last year, or oral steroids were used for 10-14 days in the past year
- Postpone dental treatment if the asthma is poorly controlled with evidence of: nocturnal wheezing, frequent severe attacks, uncontrolled exercise-induced bronchospasm, poor pulmonary function

SEDATION ISSUES:

- Recommended drugs are hydroxyzine, benzodiazepines
- Avoid: barbiturates, narcotics (morphine & mepiridine)
 - both drugs stimulate histamine release leading to bronchospasm
- Nitrous oxide is effective in mild to moderate asthmatics, but avoid prolonged periods of use, it is delivered anhydrous, can be irritating to the airway drying the bronchial secretions
- Prior medical consultation for sedation of severe asthmatics
- Light sedation or general anesthesia may be the best choice
- IV sedation to be used with extreme caution

Anecdotal triggers, associated allergies and cautions:

- Anecdotal dental triggers: dentrifrices, fissure sealants, tooth enamel dust, methyl methacrylate, fluoride trays, cotton rolls, sulfites
- Local anesthetics (LA) with vasoconstrictors are safely used unless there is a suspected or known allergy to sodium metabisulfite; vasoconstrictors in the LA may potentiate the effect of beta-agonist inhalers with the rare possibility of palpitations, increased blood pressure, arrhythmias
- ~4% of asthmatics are allergic to aspirin and other non-steroidal anti-inflammatory drugs, use acetaminophen in these cases
- Patients on theophylline medication for their asthma should not receive erythromycin - raises theophylline blood levels to a toxic range, but theophylline is rarely used today
- Managing an acute asthma attack in the dental office – see above section on managing an acute asthma attack

IX. JUVENILE ARTHRITIS

DEFINITION/EPIDEMIOLOGY/CLASSIFICATION

Previously referred to as juvenile rheumatoid arthritis with three classifications (systemic, pauciarticular, polyarticular), the juvenile idiopathic arthritides are now classified into separate categories which are further divided into subgroups to better explore etiologies, determine prognosis and evaluate therapies (Durban criteria).

- Systemic arthritis (Still's Disease): 10-15% of cases: rash, fever and arthritis of any number of joints
- Pauciartthritis: 50% cases: two groups: Involvement of less than five joints within six months of onset, or less than 5 joints in 6 months with later involvement of additional joints.
- Polyarthritis: 30-40% of cases: Involvement of more than four joints in first six months of illness, divided according to presence or absence of Rheumatoid Factor
- Enthesitis-related arthritis (Spondyloarthropathies): Arthritis plus 2 or more of: sacroiliac joint tenderness, inflammatory spinal pain, HLA-B27, positive family history of anterior uveitis with pain, a spondyloarthropathy, or inflammatory bowel disease, anterior uveitis associated with pain, redness, or photophobia (13% of cases in one study), seldom occurs before second decade of life

- Psoriatic Arthritis: Two groups – (1) arthritis and psoriasis or (2) arthritis and positive family history of psoriasis in a first degree relative, plus dactylitis or appropriate fingernail abnormalities (eg, pitting or onycholysis)

PROGNOSIS AND COMPLICATIONS

This is variable within each group, depending on the subgroups. An overall view of each group will be given, there may be distinct variations within the subgroups.

- Systemic arthritis
 - Arthritis resolves completely in 40-50% patients
 - 1/3 have prolonged illness with chronic disease including fever and rash, possibly leading to destructive arthritis with associated complications
- Pauciartthritis: Most cases benign, resolving in 6 months and major complication is uveitis and leg length discrepancy
- Polyarthritis has early onset has guarded prognosis with expected persistence of disease, adolescents probably represent early-onset adult-type disease
- Complications are flexion contractures, weakness, difficulty with ambulation, TMJ involvement with micrognathia, uveitis
- Enthesis-related arthritis
 1. Absence of HLA-B27 – often mild, especially if no elevation of sedimentation rate
 2. Presence of HLA-B27 – increased risk of developing juvenile ankylosing spondylitis, possibly severe with disability. More guarded prognosis if associated with inflammatory bowel disease and psoriasis
- Psoriatic arthritis
 1. Arthritis varies from mild (distal arthritis or asymmetric oligoarthritis) to severe, destructive and deforming (arthritis mutilans) and spondyloarthropathy.
 2. Associated with psoriatic skin disease and uveitis

MEDICAL TREATMENT

Specific therapy depends on the type of arthritis, general guidelines:

- Non-steroidal anti-inflammatory drugs or selective cox-2 inhibitors are first line therapy for the arthritides
- Second-line therapies are immunosuppressive, including but not limited to: corticosteroids, methotrexate, hydroxychloroquine, sulfasalazine, gold
- Intra-articular corticosteroids are also used
- Newer, biologic therapies include: etanacertpt or infliximab (anti-tumor necrosis factor)

DENTAL CONSIDERATIONS

- Patients are usually in chronic pain with often overprotective parents
- The patient may have limited movement, including the cervical (head and neck) area, an important consideration when positioning the patient in the dental chair
- A pillow may be used to provide support for limbs and neck
- Short appointments are preferred because of discomfort caused by sitting in a chair for prolonged periods of time
- Possible TMJ involvement with diminished mouth opening, decreased mandibular growth leading to orthodontic problems, open bite and ankylosis have been associated with destruction of the condyles, the articular cartilage has no blood supply and little reparative capacity and once destroyed degenerative changes occur giving rise to progressive joint destruction which may require surgical intervention to regain lost function

- Young children may refer to TMJ pain as an earache
- Children with JRA may be on multiple medications and potential drug interactions should be monitored
- These children may have difficulty cleaning their teeth if hands or arms are involved requiring that a toothbrush may be modified to build a larger hand grip or extended handle to make brushing easier

Surgical consideration

Consult with a rheumatologist for the necessary work-up

- For children on aspirin determine PT and PTT prior to surgical procedures
- Patients on oral steroids may require supplemental steroids to prevent adrenal suppression during traumatic and stress inducing treatment procedures
- A CBC should be taken, especially if patient is planned for treatment with general anesthesia or if the patient is taking gold or penicillamine, because of possible bone marrow suppression.

X. VASCULITIDES IN CHILDREN

WEGENER GRANULOMATOSIS

- Wegener granulomatosis is a systemic vasculitis involving medium and small arteries, venules, and arterioles. It is extremely rare in children (1 in 2 million annual incidence)
- Clinical findings include: nasal or oral inflammation – ulcers, bloody discharge, subglottic stenosis, nodules, fixed infiltrates or cavities on CXR, pulmonary hemorrhage, microscopic hematuria or red cell casts, granulomatous inflammation of artery or perivascular area on biopsy
- Laboratory finding: presence of circulating anitneutrophil cytoplasmic antibodies (ANCA) against proteinase 3 (not exclusive to Wegener's)

BEHGET'S DISEASE

- Behcet's disease is very rare in children
- Clinical findings include: oral aphthous ulcers, often extensive and multiple, occurring more than 3 times per year, genital ulceration occurs in 75% patients, cutaneous lesions in 75% patients, variable from acneiform lesions to palpable purpura, ocular disease in 25-75%: uveitis , neurologic disease in less than 20%, large vessel vasculitis in 33%, small vessel vasculitis common, less commonly involves cardiac, gastrointestinal and pulmonary systems
- Laboratory findings: No specific, diagnostic laboratory abnormalities

TREATMENT OF THE VASCULITIDES IN CHILDREN

- Specific treatment varies, depending on the severity and chronicity of the disorder
- Agents include: corticosteroids, NSAIDs, cytotoxic agents
- Morbidity and mortality in acute phase related to severity of active disease, in the chronic phase often related to complications of therapy

XI. SYSTEMIC LUPUS ERYTHEMATOSUS

DEFINITION/EPIDEMOIOLOGY

- Chronic inflammatory disorder of unknown cause

- Multi-organ system involvement
- Rare in childhood, affecting 5-10,000 children in the US
- More common in females greater than 5 yo
- Incidence and severity vary: Caucasian < Hispanic < African-American < Asian

CLINICAL PRESENTATION

- Presentation is variable, from full-blown severe disease to chronic, low-grade symptoms. Most common presentation in childhood: fever, malaise, failure to thrive
- Classic malar rash absent in 2/3 patients
- Other common presentations in childhood include: hematologic: anemia, cytopenia, and/or thrombocytopenia, mucocutaneous: malar rash and/or oral ulcers, musculoskeletal: arthritis or arthralgia, fever, nephritis, abdominal complaints

DIAGNOSIS

- SLE is diagnosed according to American College of Rheumatology Criteria in both children and adults. Four or more of the following criteria present simultaneously or serially is diagnostic for SLE (>2 possible, >3 probable, >4 classic):
 1. Malar rash – erythema, flat or raised over malar eminences
 2. Discoid rash – erythematous raised patches, keratotic scaling, follicular plugging
 3. Photosensitivity
 4. Oral ulcers – or nasopharyngeal, painless
 5. Arthritis – 2 or more peripheral joints
 6. Serositis – pleuritis, pericarditis
 7. Renal disorder – persistent proteinuria, cellular casts
 8. Neurologic disorder – seizures or psychosis
 9. Hematologic disorder – hemolytic anemia, leucopenia, lymphopenia, thrombocytopenia
 10. Immunologic disorder – positive antiphospholipid antibody, anti-DNA antibody, anti-Sm antibody, false positive test for syphilis
 11. Antinuclear antibody

COURSE OF DISEASE/PROGNOSIS/COMPLICATIONS

- With appropriate care, the prognosis of SLE in children is good
- Poor prognosis related to: poor compliance with treatment regimens, neurologic complications, intercurrent infections (often secondary to therapies), renal disease, particularly diffuse proliferative glomerulonephritis
- Survival rate nearly 100% at 5 years and 83% at 10 years
- Increased mortality associated with:
 1. Socioeconomic status
 2. Disease activity
 3. Renal and CNS involvement
- Long-term complications include:
 1. Malignancy secondary to therapeutic regimens
 2. Cardiovascular disease
 3. Organ failure (especially renal) secondary to disease process

MEDICAL TREATMENT

- Therapy based on extent of organ involvement in disease
- Maximize compliance, decrease side effects, especially secondary to corticosteroids

- Medications include:
 1. Corticosteroids – low dose in mild, to high dose
 2. NSAIDs
 3. Hydroxychloroquine in mild
 4. Steroid sparing agents in moderate/severe SLE:
 - Azathioprine
 - Methotrexate
 - Mycophenolate mofetil
 5. Cytotoxic (cyclophosphamide) agents to induce remission in poorly controlled disease

DENTAL CONSIDERATIONS

- Increased susceptibility to infection
- Establish the patient's ability to fight infection, C2/C4 complement deficiency as patients may have treatment induced depletion of antibodies
- Assess the need for bacterial endocarditis prophylaxis for secondary heart damage
- Supplemental steroids to prevent adrenal suppression for major surgical procedures or severely anxious patients
- Patients may be on multiple medications – consider possible drug interactions before prescribing any additional medication
- Assess kidney function as renal complications are common – beware of drugs metabolized and excreted by the kidney
- Patients with musculoskeletal symptoms may be uncomfortable if required to stay in the same position for long time – consider shorter appointments
- Sjogren syndrome is a secondary complication of SLE with xerostomia as a clinical manifestation

XII. CONGENITAL AND ACQUIRED IMMUNODEFICIENCIES (see complete table in resource section)

Disorders of the immune system lead to the inability to effectively fight infections.

- Congenital, or primary, immunodeficiencies occur secondary to genetic defects and usually lead to increased susceptibility to infection at birth or in early childhood, but may not be evident until later
- Acquired, or secondary, immunodeficiencies develop later in life (but may still be present in childhood), usually as a consequence of infection (HIV or AIDS), malnutrition, malignancy, or immunosuppressive medications
- Syndromic immunodeficiencies are those in which the immunodeficiency is part of an involvement of a number of organ systems
- Immunodeficiencies can affect all arms of the immune system: B-cells, T-cells, complement, phagocytes

ORAL MANIFESTATIONS

T cell defects and neutrophil deficiencies (see table for detail)

- oral candidiasis
- severe gingivitis/ prepubertal periodontitis

- gingivostomatitis
- recurrent aphthous ulceration
- recurrent herpes simplex virus infection
- premature exfoliation of primary teeth

B Cell deficiencies (see table for detail)

- few oral complications (see table)
- recurrent bacterial infections, especially pneumonia and skin lesions

DENTAL CONSIDERATIONS

- Aggressive prevention and regular review
- Maintain up-to-date medical history
- Close liaison with patient's physician
- May need complete blood cell count, white cell differential and platelets prior to any invasive dental procedure
- Prophylactic antibiotics other than Penicillin due to preponderance of Penicillin resistant organisms resulting from its chronic use in these patients
- Extraction of pulpally involved teeth to prevent septicemia
- Acyclovir for recurrent herpes simplex virus
- Antifungal – Nystatin, Amphotericin B (topical and systemic)
- Chlorhexidine 0.2% mouthwashes

XII. ADDITIONAL READINGS AND WEB SITES

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Chapter 18: CHILDHOOD CANCER

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_Chemo.pdf

- I. INCIDENCE AND OUTCOMES
- II. ORAL COMPLICATIONS OF CHEMOTHERAPY AND RADIOTHERAPY
- III. ORAL AND DENTAL MANAGEMENT
- IV. ADDITIONAL READINGS AND WEB SITES

I. INCIDENCE AND OUTCOMES

The most common cancers in children 0-19 years of age

- Leukemia
 - represented 31% of all cancer cases before 15 years of age; two major types: Acute Lymphoblastic Leukemia (75%) and Acute Non-Lymphocytic Leukemia (19%)
 - highest incidence rate found among children 1-4 years
 - leading cause of cancer death among children less than 1 year and between 10-19 years
 - overall rates are substantially higher for white children
 - overall survival for children now is approximately 80% for ALL and 50% for ANLL
 - most favorable outcome observed for children with ALL is between 1 and 10 years
 - with the exception of prenatal exposure to x-rays and specific genetic syndromes (eg., Down Syndrome), little is known about the causes of childhood ALL
- Central nervous system (CNS) cancer
 - represented 16.6% of all malignancies of childhood and adolescence
 1. Second most common malignancy and the most common solid tumors of childhood*
 2. Highest incidence found among children 1-4 years
 - leading cause of cancer death among children 1-5 years, especially for infants with ependymomas and primitive neuroectodermal tumors (PNET)

Other childhood cancers

- Lymphomas
 - 3rd most common form of childhood cancer: 15% of childhood malignancies
 - two major types: Hodgkin's disease (HD) and non-Hodgkin's lymphoma (NHL)
 - for younger children, NHL was more frequent than HD; the reverse was true in adolescence
 - 5-year survival rate: 91% for HD and 72% for NHL (1994)
- Sympathetic nervous system tumors
 - arise from the neural crest tissue
 - 7.8% of all cancers among children younger than 15 years
 - 97% were neuroblastomas: they occur almost exclusively in infants and very young children and most commonly in the adrenal gland and is the most common malignancy in the first year of life
 - 5-year survival rate: 55% (1994)
- Soft tissue sarcomas
 - 7.4% of cancer cases in children younger than 20 years of age
 - derives from primitive mesenchymal cells and arises primarily from the connective tissues of the body (10% in the head and neck)
 - congenital anomalies and genetic conditions are the only known risk factors
 - rhabdomyosarcoma was the most common soft tissue sarcoma of childhood, with most cases developing in children under 10 years: two types are embryonal (75%) and alveolar

* Most statistics do not include low grade CNS tumors. If they are accounted for, CNS cancers then become the most common form of childhood malignancy and the leading cause of cancer-related morbidity and mortality.

- overall 5-year survival rate: 64% (1994); younger children had higher survival rates and the embryonal form had a more favorable prognosis
- Renal tumors
 - 6.3% of cancer among children younger than 15 years
 - predominant form: Wilm's tumor which occurs most commonly before 5 years of age
 - Overall 5-year survival rate: 92% (1994)
- Malignant bone tumors
 - 6% of cancer cases in children younger than 20 years; peak incidence: 15 years
 - most common types: osteosarcoma (56%) and Ewing's sarcoma (34%)
 - osteosarcomas derive from primitive bone-forming mesenchymal stem cells and most often occur near the metaphyseal portions of the long bones, especially in the lower limbs
 - Ewing's sarcomas are believed to be of neural crest origin, occurring between evenly between the extremities and the central axis
 - 5-year survival rate: 63% (1994), higher for osteosarcoma than for Ewing's sarcoma
 - to date, no factor has emerged to explain even a small proportion of cases

Other considerations:

- major cause of mortality for 5-yr. survivors: primary cancer recurrence with subsequent progression
- cancer mortality has declined dramatically for children
 - < 4% of total deaths for children under 20 years in 1995
- there is an increased risk of second malignant neoplasms following all childhood cancers, particularly female sex, childhood cancer at a younger age, childhood HD or soft tissue sarcoma, exposure to alkylating agents

II. ORAL COMPLICATIONS OF CHEMOTHERAPY AND RADIOTHERAPY

The mouth is the most frequently documented source of sepsis in the cancer patient with pre-existing oral disease and poor oral hygiene being major contributing factors.

Oral complications may lead to discomfort, pain, bleeding, nutritional difficulties, infection and septicemia, increasing the costs of care and impacting the patient's quality of life as well as treatment outcomes.

Early and radical dental care and aggressive oral hygiene measures reduce the risk for oral problems and associated systemic complications such as:

- a. Mucositis
- b. Secondary infections
- c. Salivary gland dysfunction: sialadenitis*, xerostomia
- d. Neurotoxicity: taste dysfunction, dentinal hypersensitivity
- e. Bleeding
- f. Mucosal/muscular fibrosis*
- g. Osteoradionecrosis*
- h. Soft tissue necrosis
- i. Temporomandibular dysfunction
- j. Craniofacial and dental developmental problems, especially in children under

* complication of radiotherapy only

6 years of age: tooth agenesis, microdontia, crown disturbances (size, shape, enamel hypoplasia, pulp chamber abnormalities), root disturbances (early apical closure, shape, length), reduced mandibular length, reduced alveolar process height

- k. Oral graft vs. host disease (in hematopoietic stem cell transplantation only - HSCT)

III. ORAL AND DENTAL MANAGEMENT

- A. Before the initiation of cancer therapy and transplantation conditioning
 - identify, stabilize or eliminate existing and potential sources of oral infection and local irritants
 - the dental and medical history should be reviewed with special attention to the treatment protocol, hematological status, presence of a central line, and, in HSCT, the type of transplant, conditioning, and GVHD prophylaxis

PREVENTIVE STRATEGIES

toothbrushing	regular or electric soft brush 2 to 3 times daily, regardless of the hematological status, during the entire cancer or HSCT therapy toothbrushes should be air-dried
flossing oral rinses	once daily if patient is properly trained chlorhexidine only if the patient has poor oral hygiene or periodontal disease
diet	encourage a non-cariogenic diet and alert the caretakers to the high sucrose content of pediatric oral medications
fluorides	fluoridated paste and other agents for patients at risk for caries and xerostomia
trismus	daily oral physical therapy for patients who will receive radiation to the face
lip care	lanolin-based creams and ointments; avoid petrolatum products
education of patients and caretakers	the importance of optimal oral care and the acute and chronic sequelae of the cancer therapy

HEMATOLOGICAL PARAMETERS

Absolute neutrophil count	> 1,000/mm ³ : no need for supplemental antibiotics
(ANC)	< 1,000/mm ³ : defer elective care until it rises
Platelets	> 75,000/mm ³ : no additional support needed but be prepared to treat prolonged bleeding < 75,000 mm ³ : consult physician before providing dental care
Coagulation tests	may be in order for individual patients, especially those with liver or coagulation problems

DENTAL PROCEDURES

- ideally, all dental care should be completed before the therapy starts. When that is not feasible, prioritize procedures and place temporary restorations until the patient is stable. HSCT patients will be immunosuppressed for a very long time, therefore it is important that all the most important dental work is accomplished prior to the transplant. Bacterial endocarditis (BE) prophylaxis is warranted if central line is present

—prioritizing procedures:

1. infections, extractions, scaling, and sources of tissue irritation
2. carious teeth, root canal therapy and replacement of faulty restorations
 - the risk of pulpal infection and pain determines which carious lesions should be treated first

Endodontics

primary teeth	failure of pulpotomies/pulpectomies during immunosuppression can have significant impact on medical therapy. If not sure about the pulpal status, extraction is indicated.	
permanent teeth	symptomatic	root canal tx at least 1 week before initiation of cancer tx. If not possible, extract
	non-vital	
	asymptomatic	root canal tx can be delayed until hematological status is stable (except in HSCT)
	non-vital	

Orthodontics

Smooth, well fitting appliances	good oral hygiene	keep appliances
full mouth braces	poor oral hygiene remove if patient is at risk for moderate/severe mucositis or has poor oral hygiene	remove appliances

Periodontics

pericoronitis	prevention through excision of overlying tissue or extraction of 3 rd molars if indicated
scaling and polishing	before initiation of cancer therapy

Oral Surgery

Extractions	at least 7 – 10 days prior to therapy initiation no clear recommendations for use of antibiotics careful with extractions and excisional biopsies in irradiated areas – osteoradionecrosis Extract: root tips, teeth with periodontal pockets > 6mm, teeth with acute infections, significant bone loss, involvement of the furcation, non-restorable teeth individual assessment of impacted teeth
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B. During cancer therapy and early phases of transplantation

PREVENTIVE STRATEGIES

As above

DENTAL PROCEDURES	defer all elective procedures during immunosuppression periods consult physician in cases of dental emergencies BE prophylaxis if central line is present patients on vinca alkaloid agents (vincristine, vinblastine) may present with neurotoxicity of the jaws and complain of ‘toothache’ in the absence of caries or an odontogenic infection
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ORAL TISSUES

Mucositis	aggressive oral hygiene with toothbrush, regardless of the patient’s hematological status follow the International Society of Oral Oncology guidelines for mucositis care
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Secondary infections	close monitoring of the oral cavity cultures and biopsies when appropriate Nystatin prophylaxis is ineffective
Oral bleeding	local control (pressure packs, gelatin sponges, etc) systemic therapy (platelet transfusions)
Dental sensitivity/ pain	rule out caries and odontogenic infection if negative, prescribe desensitizing toothpaste or fluoride gel
Xerostomia	use of sugar-free chewing gum and candy, special dentifrices, saliva substitutes, frequent sipping of water, oral moisturizers, bland rinses, fluoride (gel, varnish, etc)

C. After cancer therapy and transplantation

PREVENTIVE STRATEGIES

As above

DENTAL PROCEDURES

HSCT patients will be immunosuppressed for a long period of time, especially if GVHD is active
defer all elective procedures during immunosuppression periods
consult physician in cases of dental emergencies during immunosuppression
periodic evaluations should be done every 6 months (earlier if patient has or is at risk for xerostomia and GVHD), regardless of immunosuppression status
BE prophylaxis if central line is present

Oral Surgery

extreme care with extractions and excisional biopsies in irradiated areas

Orthodontics

may start or resume after at least a 2-year disease-free survival
specific guidelines for orthodontic management need to be defined

ORAL TISSUES

Oral chronic GVHD	asymptomatic: monitor tissues closely symptomatic: discuss treatment strategies with physician
close monitoring of soft tissues	malignant transformations (squamous cell carcinomas)
close monitoring of dental development	thorough assessment: rehabilitation if needed

IV. ADDITIONAL READINGS AND WEB SITES

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Chapter 19: CARDIOVASCULAR DISEASES

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_AntibioticProphylaxis.pdf

- I. CONGENITAL HEART DISEASE
- II. RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE
- III. HEART MURMUR
- IV. CARDIAC ARRHYTHMIAS
- V. HYPERTENSIVE HEART DISEASE
- VI. CONGESTIVE HEART FAILURE
- VII. INFECTIVE ENDOCARDITIS
- VIII. CARDIAC CONDITIONS ASSOCIATED WITH INFECTIVE ENDOCARDITIS
- IX. DENTAL PROCEDURES REQUIRING PROPHYLAXIS AND INFECTIVE ENDOCARDITIS REGIMENS
- X. ADDITIONAL READINGS AND WEB SITES

I. CONGENITAL HEART DISEASE

INITIAL LEFT TO RIGHT SHUNTING OF BLOOD

Examples:

- Atrial septal defect, ventricular septal defect (VSD), patent ductus arteriosus
- Transposition of great vessels – pulmonary arteries supplied by left ventricle and the aorta is supplied by the right ventricle
- Persistent truncus arteriosus – blood from both ventricles move together as it exits through a single valve exiting from the heart
- Tetralogy of Fallot - VSD, pulmonic stenosis, aorta overrides VSD, hypertrophy of the right ventricle

OBSTRUCTION OF BLOOD FLOW

- Pulmonary stenosis, coarctation of the aorta

CARDIAC DEFECTS

Associated with several of the most common congenital disorders

- Down and Turner syndromes, osteogenesis imperfecta, Marfan syndrome, Ehler-Danlos syndrome

Symptoms

Vary depending on the condition but can include:

- Dyspnea, cyanosis (late in left to right shunting and early in right to left shunting), polycythemia, clubbing of toes or fingers, syncope, coma, weakness, murmur

Complications

- Brain abscess, infective endocarditis, cerebrovascular problems, congestive heart failure, acute pulmonary edema, bleeding problems, retardation of growth

Medical Management

- Surgery to correct the defect (e.g. Tetralogy of Fallot may benefit from the Blalock-Taussig operation)
- Medication for right or left ventricular failure (e.g. digitalis and anti-coagulation therapy)
- Treating complications of congenital heart disease (e.g. bleeding problems)

Dental Evaluation/Management

- A medical consultation is recommended to determine the specific diagnosis and the possible risk of infective endocarditis

II. RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

RHEUMATIC FEVER is an acute inflammatory condition that develops in some individuals as a complication following group A *streptococcal* infections (e.g. strep throat infections).

It is thought to arise as a result of an autoimmune reaction between normal tissues that have been altered by products of the bacteria and antibodies that have been produced by the host in response to these altered tissues.

Prevalence

- Commonly occurs between the ages of 5 and 15
- Prevalent in temperate zones, high altitudes, and substandard living conditions

Symptoms

- Arthritis, carditis, chorea, erythema marginatum, subcutaneous nodules

Complications

- Inflammation reactions in the heart, larger joints, skin, lungs

Medical Management

- Penicillin g benzathine, codeine, salicylates

RHEUMATIC HEART DISEASE is the cardiac damage that can result from rheumatic fever.

- Damage most commonly occurs to the mitral or aortic valve
- Scarring and calcification in the affected valves may result in stenosis or regurgitation

Incidence

- Less than .05 per 1000 of the US population

Symptoms

- Murmur, exertional dyspnea, angina pectoris, epistaxis, blood in the sputum, congestive heart failure

Complications

- Scar tissue and deformity of the valve, mitral stenosis, incompetence of the aortic valve, aortic stenosis, acute pericarditis

Medical Management

- Asymptomatic disease requires no treatment other than for prevention of recurrent attacks of rheumatic fever.

Dental Management

- A patient with a past history of rheumatic fever is in need of medical consultation to rule out rheumatic heart disease.

III. HEART MURMURS

Heart murmurs are sounds caused by turbulence in the circulation through the valves and chambers of the heart.

- Turbulence in flow is usually the result of an increased flow rate, a change in viscosity, stenotic or narrowed valves, or a vibration of membranous structures.
- Innocent or functional murmurs are sounds caused by turbulence in the absence of any cardiac abnormality.
- Organic murmurs are sounds caused by a pathologic abnormality in the heart.

Dental Management (see sections VIII & IX)

IV. CARDIAC ARRHYTHMIAS

Arrhythmias are any variation in the normal rhythm of the heart beat. Cardiac arrhythmias may be disturbances of rhythm, rate, or conduction.

Signs

- In children, bradycardia (less than 60 beats per minute), tachycardia (more than 120 beats per minute), or irregular heart beats

Symptoms

- Patients can be asymptomatic or display symptoms of fatigue, dizziness, syncope, heart palpitations, angina, or congestive heart failure

Complications

- Ischemic heart disease, angina, myocardial infarction, congestive heart failure, cardiac arrest, blindness, cerebrovascular accident

Medical Evaluation

- Determine the presence and characteristic of the arrhythmia i.e. (atrial or ventricular)
 - Past history, pulse rate, rhythm, underlying condition causing arrhythmia, frequency of symptoms
- Provide an understanding of the status, severity, and control of arrhythmia
 - Treatment being rendered, medications and potential side effects, frequency of symptoms, and hospitalization(s),

Risk Classification

Low Risk

- Infrequent symptoms (e.g. atrial arrhythmias, premature ventricular beats, young patients with sinus bradycardia), no medications necessary

Moderate Risk

- Atrial or ventricular arrhythmias, may be asymptomatic due to chronic medications and/or pacemakers

High Risk

- Pulse greater than 100 or less than 60 and are symptomatic (e.g. irregular pulse rhythm, bradycardic patients with pacemakers, irregular pulse and bradycardia) despite being on chronic medications and/or a pacemaker

Medical Management

- Medication: digoxin, quinidine, procainamide, disopyramide, lidocaine, propanolol, verapamil
- Pacemakers
- Surgery
- Cardioversion in an emergency

Dental Management

- Thorough medical history to identify patients with arrhythmias
- Medical consult to establish risk classification, need for antibiotic prophylaxis, and management recommendations
- Minimize stressful situations

- Reduce anxiety with pre-medication, nitrous oxide, and/or sedation as indicated
- Short morning appointments
- Minimize use of epinephrine
- Avoid general anesthesia
- Avoid electrical equipment that may interfere with a pacemaker

Oral Complications

Medications that are used to control arrhythmias have potential oral side effects, which include but not limited to:

- Ulceration
- Lupus-like syndrome
- Xerostomia
- Petechiae

V. HYPERTENSIVE HEART DISEASE

Essential hypertension is defined as systolic pressure exceeding the 95 percentile for gender, age, and height in children after 3 readings in a non-stressful situation

Age	S/D
3 to 5	>116/76
6 to 9	>122/78
10 to 12	>126/82
13 to 15	>136/86
16 to 18	>140/90

Secondary hypertension is caused by an underlying disorder such as a renal or an endocrine disorder or certain types of medication such as oral contraceptives. The incidence of hypertension in newborns is 0.2-3.0% and in children ages 4-15 years is 1.5-2.0%.

Hypertension is often undiagnosed. If history and multiple, non-stressful blood pressure readings suggest hypertension, a referral for medical consultation is needed to determine status of hypertension (essential vs. secondary).

Symptoms

- Essential hypertension normally is asymptomatic. However, occipital headache, visual blurriness, changes in mental status, weakness, dizziness, and angina may occasionally occur.

Complications

- Renal failure, cerebrovascular accident, coronary insufficiency, myocardial infarction, congestive heart failure, blindness

Medical Management

- Obtain a comprehensive medical and family history
- Identify risk factors
- Rule out secondary hypertension
- Drug therapy with diuretics or beta blockers, ACE inhibitors, calcium channel blockers, vasodilators

Dental Evaluation/Management

- Measure and record blood pressure at each visit
- Proceed with dental treatment in patients with controlled to mild hypertension
- Patients with moderate hypertension schedule short morning appointments, consider ways to reduce anxiety
- Postpone elective dental procedures for patients with severe hypertension

Oral Complications

- Xerostomia secondary to diuretics and other antihypertensive medications
- Lichenoid reactions associated with thiazides, methyldopa, propranolol,
- Delayed healing and gingival healing associated with ACE inhibitors
- Gingival hyperplasia associated with calcium channel blockers
- Facial palsy associated with malignant hypertension

VI. CONGESTIVE HEART FAILURE

Congestive heart failure is the inability of the heart to deliver an adequate supply of blood to meet metabolic demands.

Etiology

- Coronary heart disease, hypertension, cardiomyopathy, infective endocarditis, congenital heart disease, endocrine disease

Symptoms

- Fatigue, weakness, dyspnea, hyperventilation, low-grade fever, cough, insomnia, weight gain, dizziness, confusion

Complications

- Compensatory responses include: increased peripheral resistance, redistribution of blood flow to the heart and brain, increased erythropoietic activity to increase oxygen carrying capacity of the blood.
- Complications include: ventricular dysfunction, congestive failure with dyspnea, pulmonary congestion, and peripheral edema

Medical Management

- Identify the causative factors (e.g. high blood pressure) and correct and stabilize
- Modify lifestyles:
 - Initiate smoking cessation, weight loss and exercise programs, restrict salt intake, and recommend techniques that promote stress reduction
- Prescribe drug therapy as needed:
 - ACE inhibitors alone or with a diuretic
 - In cases of moderate to severe congestive heart failure loop diuretics (e.g. furosemide) may be indicated

Dental Management

- Consult with patient's physician to determine ability to tolerate treatment
- Avoid procedures, which may cause a gag reflex
- Minimize use of epinephrine
- Prevent orthostatic hypotension

- Investigate potential bleeding problems from use of anticoagulants, an International Normalized Ratio (INR) of 3.5 or less is required
- Maintain the patient in an upright position during treatment if pulmonary edema is present

Oral Complications

- Infection
- Bleeding
- Petechiae
- Ecchymoses
- Drug related side effects: xerostomia, lichenoid mucosal lesions

VII. INFECTIVE ENDOCARDITIS

Infective endocarditis is a microbial infection (fungal or bacterial) of the heart valves or endocardium, occurring in multiple forms, often seen in patients with congenital defects of the heart.

- **BACTERIAL ENDOCARDITIS** - Bacterial infection of the heart valves or endocardium
 1. Acute bacterial endocarditis- sudden onset, can be fatal in less than 6 weeks, causative agent *Staphylococcus aureus* can infect normal heart valves, occurring in males more than females, median age of occurrence is 50 years
 2. Subacute bacterial endocarditis- slower onset, *Streptococcus viridans* infects damaged heart valves
- Pediatric dental patients are more likely to acquire subacute, rather than acute, bacterial endocarditis
- The incidence of all bacterial endocarditis in the US is estimated to be less than 1%

Symptoms

- Weakness, weight loss, fatigue, fever, chills, night sweats, anorexia, arthralgia

Complications

- Emboli, cerebral abscesses, myocardial abscesses, mycotic aneurysms, hemorrhage, congestive heart failure

Medical Management

- Antibiotic therapy based on culture and sensitivity findings
- Antibiotic treatment times vary depending on the type of organism causing the endocarditis.

Dental Management

- Antibiotic prophylaxis prior to bacteremia-inducing procedures (see below)

VIII. CARDIAC CONDITIONS ASSOCIATED WITH INFECTIVE ENDOCARDITIS

The following recommendations and guidelines refer only to patients with cardiac conditions. They do not refer to other types of conditions (implants, transplants, shunts, etc.) where antibiotic prophylaxis may be necessary.

Prevention of infective endocarditis- American Heart Association guidelines of April 2007 (Wilson, et al., 2007)

Patients with the following cardiac conditions are at risk for infective endocarditis therefore, antibiotic prophylaxis is recommended:

- Prosthetic cardiac valves
- Previous infective endocarditis.
- Congenital heart disease
 - Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits
 - Repaired congenital heart defect with prosthetic material or device, whether placed by surgery or catheter intervention during the first six months after the procedure
 - Repaired congenital heart disease with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

Cardiac conditions where antibiotic prophylaxis is **NOT** recommended

- Most congenital cardiac malformations (other than ones listed above).
- Acquired valvar dysfunction (e.g., rheumatic heart disease).
- Hypertrophic cardiomyopathy.
- Mitral valve prolapse with valvular regurgitation and/or thickened leaflets.
- Isolated secundum atrial septal defect.
- Surgical repair of atrial septal defect, ventricular septal defect, or patent ductus arteriosus (without residua beyond 6 months).
- Previous coronary artery bypass graft surgery.
- Mitral valve prolapse without valvular regurgitation.
- Physiologic, functional, or innocent heart murmurs.
- Previous Kawasaki disease without valvar dysfunction.
- Previous rheumatic fever without valvar dysfunction.
- Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators.

IX. DENTAL PROCEDURES REQUIRING PROPYLAXIS AND INFECTIVE ENDOCARDITIS REGIMENS

For the patients with cardiac conditions at risk for infective endocarditis antibiotic prophylaxis is recommended for the following procedures:

- All dental procedures that involve manipulation of the gingival tissue or the periapicals region of the teeth or perforation of the oral mucosal.

Endocarditis Prophylaxis is NOT recommended for:

- Routine anesthetic injections through non-infected tissue
- Placement of removable prosthodontic or orthodontic appliances
- Placement of orthodontic brackets
- Taking of oral radiographs
- Orthodontic appliance adjustment
- Shedding of primary teeth
- Bleeding from trauma to the lips or oral mucosal

Situation	Agent, Route, and Time	Regimen
Standard prophylaxis	Amoxicillin Orally 1 hour before procedure	Adult: 2.0 g Children: 50-mg/kg
Unable to take oral medications	Ampicillin IM* or IV* 30 min before procedure	Adult: 2.0 g Children: 50-mg/kg
Allergic to penicillin or ampicillin	Clindamycin Orally 1 hour before procedure	Adult: 600 mg Children: 20-mg/kg
	Cephalexin or Cefadroxil**\$ Orally ~1 hour before procedure	Adult: 2.0 g Children: 50-mg/kg
	Azithromycin or clarithromycin Orally 1 hour before procedure	Adult: 500 mg Children: 15mg/kg
Allergic to penicillin or ampicillin AND-unable to take oral medications	Clindamycin IM or IV within 30 minutes - of procedure	Adult: 600 mg Children: 20-mg/kg
	Cefazolin or ceftriaxone \$ IM or IV within 30 minutes -of procedure	Adult: 1.0 g Children: 50-mg/kg

*IM – intramuscular; IV – intravenous

**or other first or second generation oral cephalosporin in equivalent adult or pediatric dosage

\$Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin

X. ADDITIONAL READINGS AND WEB SITES

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Chapter 20: ENDOCRINE DISORDERS

- I. PANCREAS**
- II. THYROID GLAND**
- III. ADRENAL GLAND**
- IV. PARATHYROID**
- V. PITUITARY GLAND**
- VI. ADDITIONAL READINGS AND WEB SITES**

Endocrine describes the actions of hormones secreted into the bloodstream. Hormones can be defined as chemical signals secreted into the blood stream that act on distant tissue, usually in a regulatory manner.

Endocrine system is a collection of glands that produces these hormones necessary for normal bodily functions and regulates metabolism, growth and sexual development.

Major physiologic processes controlled by hormones include (1) growth and maturation, (2) intermediary metabolism, and (3) reproduction. However, endocrinology is most clearly delineated by diseases that afflict the classic glands, hypothalamus, pituitary, thyroid, parathyroid, pancreatic islets, adrenal gland, testis and ovary.

I. PANCREAS

- Produces enzymes that break down all categories of digestible foods (exocrine pancreas)
- Secretes hormones that affect carbohydrate metabolism (endocrine pancreas)

-Diabetes Mellitus

- A group of metabolic diseases characterized by hyperglycemia
- Epidemiology:
 1. Total prevalence of diabetes in the U.S (All Ages, 2005) = 7%
 2. Prevalence of diagnosed diabetes in people aged 20 or younger = 0.22%
- Diagnosis
 1. Symptoms of diabetes plus random plasma glucose concentration ≥ 200 mg/dl, or
 2. Fasting plasma glucose ≥ 126 mg/dl, or
 3. 2 hour plasma glucose ≥ 200 mg/dl during oral glucose tolerance test
- Causation/classifications: result from defects in insulin secretion, insulin action, or both which is required for normal glucose homeostasis
 1. Type I diabetes
 - Destruction of the insulin-producing beta cells in the pancreas, usually leading to absolute insulin deficiency (Immune-mediated or idiopathic)
 - Etiology involving genetic and environmental factors
 - Family history 3%-5%, but significant familial clustering
 - The most common type of diabetes in children
 - About one in every 400 to 600 children and adolescents
 - Classical symptoms (triad): polyuria, polydipsia, and weight loss
 - The classic sign of polyphagia might be absent in children
 - Ketoacidosis (DKA) is present in 15-40% of newly diagnosed children
 - Treatment requires insulin injections
 2. Type II diabetes
 - Insulin resistance with relative insulin deficiency
 - Combination of insulin resistance with a secretory failure
 - Patient frequently overweight
 - Family history 74%-100%
 - Treatment requires life style modifications with increased exercise, appropriate diet, and oral hypoglycemic agents
 3. Type III diabetes
 - Diabetes caused by other identifiable etiologies such as genetic defects of beta cell function and insulin action, diseases of the exocrine pancreas, drug or chemical induced, infections, immune-related diabetes, and genetic syndromes

- 4. Type IV diabetes: Gestational diabetes
 - 2-5% of all pregnancies
- Course of disease
 1. Glucose is not handled properly by body cells or stored appropriately in the liver and muscles.
 2. Net effect is persistent high levels of blood glucose, poor protein synthesis, and other metabolic derangements.
- Long-term complications:
 1. Retinopathy with potential loss of vision
 2. Mephropathy leading to renal failure
 3. Peripheral neuropathy with risk of foot ulcers and amputations
 4. Skeletal, joint, and skin complications
 5. Autonomic neuropathy causing gastrointestinal, genitourinary, cardiovascular symptoms, and sexual dysfunction
 6. Atherosclerotic cardiovascular, peripheral arterial and cerebrovascular disease
 7. Hypertension and abnormalities of lipoprotein metabolism
 8. Impairment of growth and susceptibility to certain infections
- Dental/Oral findings:
 1. Xerostomia
 2. Increased caries risk
 3. Oral Candidiasis
 4. Burning mouth or tongue
 5. Taste alteration
 6. Increased risk of periodontal disease
 7. Poor wound healing/surgical wound infections
 8. Acetone breath
 9. Odontalgia, percussion sensitivity, pulpitis, loss of vitality (diabetes-related microangiopathy-specially with ortho)

Dental Considerations

1. Good medical history (recent blood glucose levels, frequency of hypoglycemic episodes, medications, dosages, and times of administration)
2. Avoid hypoglycemic episode (appear weak, nervous, confused, skin is pale and moist, excessive salivary flow) by:
 - Scheduling morning appointment
 - Keep appointments short
 - Eat a usual meal and take the medication as usual
 - Minimize stress (profound local anesthesia, sedation)
 - Diet instruction post-operatively when expected difficulty with eating
3. Surgical procedures/sedations may require adjustment of insulin dosage or antibiotic
4. Consult with physician if necessary
5. In case of hypoglycemic episode
 - Stop dental treatment
 - High carbohydrate beverage (orange juice or soft drink) or IM Glucagon
 - Seek medical assist for unconscious patient
6. Good oral hygiene/routine recall/home use of topical fluoride for at risk

- 7. Aggressive periodontal care - infection management
- 8. Palliative care for dry mouth using salivary substitutes
- 9. Uncontrolled diabetes not a candidate for ortho

- Other Management Aspects
 - 1. One third of patients with longstanding type 1 diabetes may be difficult to intubate
 - 2. Modify treatment based on systemic complications - renal, cardiac, ocular, neurologic
 - 3. Pharmacologic consideration
 - Chlorpropamide/Diabinese: avoid ASA
 - Insulins: ASA leads to hypoglycemia & steroids lead to hyperglycemia

II. THYROID GLAND

- The thyroid gland secretes:
 - 1. Thyroxine (T4), triiodothyronine (T3) affecting metabolic processes throughout the body
 - 2. Calcitonin involved in regulating serum calcium and phosphorous levels and skeletal remodeling.

-Hypothyroidism

- Definition: condition resulting from insufficient production or diminished action of thyroid hormone
- Epidemiology
 - 1. 10 times more common among women than men (prevalence of 2% in adult women and 0.2% in adult men)
 - 2. Incidence increases with age
 - 3. NHANES III hypothyroidism (defined as elevated TSH levels) in 4.6%
 - 4. The annual incidence of hypothyroidism in adults is approximately 0.08% to 0.2%.
- Classifications/causes
 - 1. Primary hypothyroidism (thyroid gland dysfunction - 95% of cases)
 - Congenital hypothyroidism: Thyroid Agenesis or Dysplasia (cretinism 1 in 3500 newborns)
 - Acquired primary hypothyroidism: Hashimoto thyroiditis (an autoimmune disease, which is the most common cause of hypothyroidism after 8 years of age)
 - Drugs (including lithium, iodides, amiodarone, thiourea compounds, alpha-interferon, interleukins, tumor necrosis factor), which may be transmitted maternally to fetus
 - Iodine deficiency (rare in the US, but one of the most common causes of hypothyroidism in some other parts of the world)
 - Destruction of thyroid tissue due to external radiation therapy to the neck or radioiodine therapy
 - Surgical removal of the thyroid
 - Idiopathic
 - 2. Secondary hypothyroidism (5% of cases)
 - Pituitary or hypothalamic dysfunction
 - Congenital hypopituitarism
 - Pituitary necrosis (Sheehan syndrome)

- Course of disease
 1. Characterized by a generalized reduction in metabolic function that most often manifests as a slowing of physical and mental activity,
 2. In very young infants, hypothyroidism can result in irreversible mental retardation and slowed physical growth.

Clinical Signs and Symptoms:		
Fatigue	Cold Intolerance	Hair loss
Lethargy	Hoarseness	Generalized edema
Sleepiness	Decreased perspiration	Pitting edema of lower extremities
Mental impairment	Dry, scaly skin	Slow wound healing
Depression	Dry, brittle hair	Weight gain
Decreased appetite	Thick, brittle nails	Constipation
Infertility	Arthralgia	
Paresthesia	Menstrual irregularities	

- Prognosis: good with proper therapy
- Complications
 1. Myxedema coma: rare, life-threatening condition resulting from the progressive deterioration of thyroid function. Mental dysfunction, stupor, cardiovascular collapse, and/or coma develop after the worsening of a long-standing thyroid hormone deficiency
 2. Medical conditions associated with hypothyroidism include anemia, dilutional hyponatremia, and hyperlipidemia
 3. Thyroid hormone deficiency effect growth and development, central nervous system development and function, cardiovascular, skeletal, gastrointestinal, and reproductive system
- Diagnosis:
 1. A comprehensive history and physical examination to evaluate signs and symptoms
 2. A laboratory assay of TSH and free T4 levels confirms diagnosis. Free T3 levels may or may not be useful
- Medical treatment
 1. The treatment of routine hypothyroidism depends on the underlying cause of the disease
 2. Replacement thyroxine (levothyroxine) is the cornerstone of therapy for hypothyroidism
- Oral findings
 1. Enlarged tongue
 2. Delayed dental development and tooth eruption
 3. Malocclusion,
 4. Gingival edema
 5. Delayed skeletal development
 6. Protruding tongue, and thick lips
- Dental management
 1. Good medical history
 2. Sensitivity to stress, infection, surgery
 3. Sensitive to some drugs such as sedatives and opioid analgesics
 4. Myxedema coma signs include hypothermia, bradycardia, hypotension, seizures
 5. Consult with physician as appropriate

-Hyperthyroidism

- Definition: a hypermetabolic state that results from excess synthesis and release of thyroid hormone from the thyroid gland
 1. Thyrotoxicosis is a general term referring to all causes of excess thyroid hormone in the body, including exogenous intake of thyroid hormone preparations
 2. Clinical spectrum varies from asymptomatic, subclinical hyperthyroidism to the life-threatening thyroid storm
- Epidemiology: The overall incidence of subclinical and overt hyperthyroidism has been estimated to be 0.05 to 0.1% in the general population
- Causation
 1. Graves Disease
 - Generalized overactivity of the thyroid gland
 - Autoimmune disease
 - Most common form of hyperthyroidism
 - Up to five times more common among women than men
 - Associated with eye disease (Graves ophthalmopathy) and skin lesions (dermopathy)
 2. Excessive intake of thyroid hormones
 3. A tumor in the pituitary gland (abnormal secretion of TSH)
 4. Functioning adenoma (“hot nodule”) & Toxic Multinodular Goiter (TMNG)
 5. Thyroiditis
 6. Excessive iodine intake
 7. Metastatic thyroid cancer
- Course of disease: Thyroid hormone affects nearly every organ system in the body. Patients with elevated thyroid hormone present in a hypermetabolic state with signs of increased b-adrenergic activity
- Prognosis: Good with proper therapy
- Complications
 1. Osteoporosis, atrial fibrillation, hypertension, and congestive heart failure
 2. Thyrotoxic crisis
 - Extreme restlessness, nausea, vomiting, and abdominal pain
 - Fever, profuse sweating, marked tachycardia, cardiac arrhythmias, pulmonary edema, and congestive heart failure
 - Stupor, and coma
 - Severe hypotension and death
- Diagnosis
 1. Symptoms of rapid heart rate, intense fatigue, inability to tolerate a hot environment, and constant nervousness, jitteriness, or irritability
 2. Physical signs such as weight loss, rapid heartbeat, slight tremors of the hands, or excessive sweating
 3. Blood tests: abnormally high levels of T3 and T4 and an unusually low level of circulating thyroid stimulating hormone
 4. Diagnostic scan -radioactive iodine uptake (RAIU) testing
- Medical Treatment : Antithyroid drugs, radioactive iodine, surgery (thyroidectomy)
- Oral findings
 1. Osteoporosis may be found involving the alveolar bone
 2. Dental caries and periodontal disease appear more rapidly in these patients
 3. The teeth and jaws develop more rapidly

4. Premature loss of the deciduous teeth with early eruption of the permanent teeth is common
5. Euthyroid infants of hyperthyroid mothers have been reported with erupted teeth at birth
6. Damaged salivary gland (secondary to radioactive iodine)

Table I: Dental management of the hyperthyroid patient

Disease status	Clinical action
Detection of undiagnosed disease	<ul style="list-style-type: none"> • Symptoms • Signs • Refer for medical Dx and Rx
Diagnosed disease	<ul style="list-style-type: none"> • Determine original diagnosis and Rx • Past treatment • Current treatment • Lack of signs and symptoms • Presence of any complications • Consult MD • Avoid stress
Untreated or poorly controlled	<ul style="list-style-type: none"> • Avoid surgical procedures • Treat any acute infection • Avoid use of epinephrine or pressor amines • Thyroid storm
Well controlled	<ul style="list-style-type: none"> • Treat acute infection (avoid if possible) • Treat chronic infection • Implementation of normal procedures and management
Medical crisis (rare)	<ul style="list-style-type: none"> • Recognition and initial management of thyrotoxic crisis • Seek medical aid • Wet packs, ice packs • Hydrocortisone (100 to 300 mg) • IV glucose solution • Cardiopulmonary resuscitation • The use of salicylates is avoided

III. ADRENAL GLAND

- Produces three different classes of steroid hormones:
 1. Glucocorticoids (major - cortisol): help regulate blood sugar levels and the metabolism of protein and fat; important actions on the body's immune response, particularly important in helping the body buffer any kind of stress
 2. Mineralocorticoids (major - aldosterone): help regulate the body's sodium and potassium levels, blood volume, and blood pressure
 3. Androgens: affect secondary sex characteristics such as underarm and pubic hair in women and may be important for their libido (sex drive), not important for men, who produce most of their androgen in the testes

-Adrenal insufficiency (AI)

- Definition: characterized by a deficiency of one or more of the above classes of hormones
 1. Primary AI (Addison disease): the problem lies at the level of the adrenal gland (destruction or malfunction of more than 90% of the adrenal cortex)

Epidemiology: occurring in about eight people per million population per year, with a prevalence of about 40 to 100 per million

Causation

- Autoimmune adrenalitis
- Tuberculosis, fungal infections
- AIDS
- Congenital adrenal hypoplasia or hyperplasia
- Adrenal hemorrhage (Waterhouse-Friderichsen syndrome)
- Sepsis

Symptoms

- Fatigue, generalized weakness, loss of appetite and weight loss
- Darkening of the skin
- Gastrointestinal symptoms such as nausea and vomiting
- Hypotension
- Muscle and joint pain
- Salt cravings

2. Secondary and tertiary AI: the problem lies at the level of the pituitary gland and the hypothalamus

Causation

- Pituitary or hypothalamic tumors
- Deficiencies in one or more of the pituitary hormones (partial or panhypopituitarism).
- long-term treatment with glucocorticoid medications

Symptoms similar to those of primary insufficiency, with a few exceptions:

- Darkening of the skin and dehydration do not occur
- Gastrointestinal symptoms are less common
- Symptoms of hypoglycemia (low blood sugar) are more common and include sweating, anxiety, tremulousness, nausea, palpitations
- Other symptoms may be related to a pituitary or hypothalamic tumor, including headaches, visual field loss, and symptoms of low levels of other pituitary hormones, such as infertility, impotence, fatigue, hoarseness, constipation, or delay of puberty or skeletal growth in children

3. Adrenal crisis

Refers to overwhelming and life-threatening adrenal insufficiency

Characterized by dehydration, hypotension, and imbalances of sodium and potassium

Usually occurs in people with primary adrenal insufficiency

The main symptom is shock. In some cases, shock may be preceded by fever, nausea, vomiting, and abdominal pain; weakness and fatigue; and confusion

Usually precipitated by an infection, trauma, or some other stressor

- Diagnosis

Regular blood tests may show increased blood potassium or low blood sodium. The definitive test for the disease is called ACTH stimulation. The hormone ACTH, when injected, should make the person's blood levels of cortisol and aldosterone rise

- Medical treatment

1. Primary adrenal insufficiency — replacement of both glucocorticoids and mineralocorticoids (androgens in women)
2. Secondary or tertiary adrenal insufficiency — usually requires only

glucocorticoid replacement. Treatment may also include replacement of other deficient pituitary hormones that are unrelated to adrenal function.

3. Treatment of the underlying cause of insufficiency (i.e. infections)
4. Adrenal crisis treatment usually consists of large volumes of intravenous salt solution and an injectable form of glucocorticoid

- Prognosis
 1. Primary AI good prognosis with treatment
 2. The prognosis for patients with secondary or tertiary adrenal insufficiency will depend on the underlying cause
- Oral findings/complications
 1. Hyperpigmentation of the skin and mucous membranes
 2. Delayed healing
 3. Infection

Dental Considerations

1. Be aware of early warning signs of adrenal crisis
2. Adrenal crisis is a rare event in dentistry, and most routine dental procedures can be performed without glucocorticoid supplementation
 - Request that the patient take his or her usual steroid dose before coming to the dental office
 - Schedule the appointment in the morning when cortisol levels are highest
 - Providing stress reduction measures with appropriate postoperative analgesia
3. For dental extractions or surgery: corticosteroid dose will generally need to be increased; consult patient's MD prior to the procedure
4. Consult with patient's MD if patient stopped long-term corticosteroid therapy within the previous two weeks
5. Factors associated with the risk of adrenal crisis included the magnitude of surgery, the use of general anesthetics, the health status and stability of the patient, and the degree of pain control

-Hyperadrenalinism (Cushing syndrome)

- Definition: A metabolic disorder characterized by glucocorticoid excess
- Causation
 1. Hypercortisolism caused by excessive corticotropin (ACTH) secretion by tumors in the pituitary gland (Cushing disease)
 2. ACTH-independent cortisol secretion from adrenal tumors
 3. Chronic steroid therapy

Symptoms	
Moon face (round, red, and full)	High blood pressure
Weight gain	Weakness (often noticed when trying to stand up or to raise hand above head)
Buffalo hump (a collection of fat between the shoulders)	Backache
Central obesity with protruding abdomen and thin extremities	Headache
Skin changes	Thirst
Thinning of the skin	Increased urination
Easy bruising	Mental changes, including euphoria not linked to a life event
Acne or superficial skin infections	Cessation of menses in women
Purple striations on the skin of the abdomen, thighs, and breasts	Breast development

- Diagnosis
 1. 24-hour urine sample to measure the following:
 - Urine cortisol
 - Urine creatinine
 2. Dexamethasone suppression test
 3. Serum cortisol levels
 4. Saliva cortisol levels
 5. Plasma ACTH
- Treatment:
 1. Surgical resection of tumor is the optimal treatment for all forms of Cushing syndrome;
 2. Bilateral adrenalectomy, medical treatment, or radiotherapy is sought in inoperable or recurrent cases;
 - The medical treatment of choice is ketoconazole
- The prognosis is better for Cushing disease and benign adrenal causes of Cushing syndrome than adrenocortical cancer and malignant ACTH-producing tumors
- Complications: correlated with direct and/or indirect effects of glucocorticoid excess; cardiovascular, hypertension, impaired glucose tolerance and diabetes, obesity, hyperlipidemia, coagulopathy, osteoporosis, psychological and cognitive, alterations of other endocrine systems
- Dental findings
 1. Osteoporosis
 2. Delayed wound healing
- Dental management
 1. Susceptibility to fracture
 2. Implants contraindicated

IV. PARATHYROID GLAND

- Produces Parathyroid Hormone (PTH)
 1. Regulates serum calcium through a negative feedback mechanism
 2. A decrease in serum calcium stimulates secretion of PTH
 3. Actions
 - Activates and increases the number of osteoclasts
 - Increases renal tubular reabsorption of calcium
 - Increases conversion of Vitamin D to active form in kidneys
 - Increases urinary phosphate excretion, reducing calcium loss
 - Increases gastrointestinal calcium reabsorption
- Excess secretion of PTH-Hyperparathyroidism
 1. Primary Etiology
 - Caused by adenomas (85%) or gland hyperplasia (15%)
 - Hypercalcemia
 2. Secondary Etiology
 - Chronic renal failure
 - Hypocalcemia stimulates PTH
- Hyperparathyroidism Findings
 1. Osteoporosis
 2. Renal calcium stones
 3. Gastric distress
 4. Recklinghausen disease

5. Oral Pathology

- Generalized loss of lamina dura
- Decreased density of bony trabecula has “ground glass” appearance
- Osteitis fibrosa cystica “brown tumors” which are histologically identical to central giant cell granuloma
- Reduced Secretion of PTH-Hypoparathyroidism
 1. Primary Etiology
 - Surgery-inadvertent removal of parathyroid glands
 - Radiation
 2. Secondary Etiology
 - DiGeorge Syndrome
 - Idiopathic Atrophy
- Hypoparathyroidism Findings
 1. Tetany/Neuromuscular irritability
 2. Paresthesia of distal extremities
 3. Laryngospasm
 4. Oral Pathology
 - Circumoral paresthesia
 - Dental Developmental Findings
 - Enamel hypoplasia
 - Delayed eruption
 - Enamel Attrition
- Psuedohypoparathyroidism Findings

Organ resistance to PTH
Albright hereditary osteodystrophy has round facies, short stature, short metacarpal and metatarsal bones

V. PITUITARY GLAND

- Plays a major role in the endocrine system, linking the endocrine system and the CNS
- Hormones: Thyrotropin, Gonadotropins, Growth hormone (GH), Corticotropin, Prolactin hormone

-Hypopituitarism

- Causes of primary hypopituitarism:
 1. Genetic defects
 2. Pituitary tumors
 3. Inadequate blood supply to pituitary gland
 4. Infections and/or inflammatory diseases
 5. Sarcoidosis - a rare inflammation of the lymph nodes and other tissues throughout the body
 6. Amyloidosis - a rare disease which causes the buildup of amyloid, a protein and starch, in tissues and organs
 7. Radiation therapy
 8. Surgical removal of pituitary tissue
 9. Autoimmune diseases
- Causes of secondary hypopituitarism (affecting the hypothalamus):
 1. Tumors of the hypothalamus

- 2. Inflammatory disease
- 3. Head injuries
- 4. Surgical damage
- Symptoms
 - 1. Fatigue and weakness
 - 2. Decreased appetite and weight loss
 - 3. Cold sensitivity
 - 4. Abdominal pain and headache
 - 5. Visual disturbances - blurred vision, blindness
 - 6. Loss of body hair
 - 7. Short stature
 - 8. Infertility and loss of libido
 - 9. Cessation of menses
- Growth hormone deficiency
 - 1. Prevalence of 1 case in 3480 children
 - 2. Slow linear growth rates, normal skeletal proportions, and many have a pudgy, youthful appearance because of decreased lipolysis
- Diagnosis
 - 1. Computed tomography
 - 2. Magnetic resonance imaging (MRI)
 - 3. Blood tests to measure hormone levels
- Treatment depends on its cause: replacement hormone therapy, surgical tumor removal, radiation therapy
- Prognosis: good with therapy - increased cardiac morbidity and mortality due to growth hormone deficiency
- Clinical complications: Cushing syndrome, hyperthyroidism, adrenal crisis, diabetes insipidus
- Dental findings of Hypopituitary Dwarfism: decreased linear facial measurements, delayed tooth eruption, smaller mandible – delayed development, hypodontia
- Dental Considerations: good medical history, growth and development assessment, dental caries prevention and treatment, periodontal disease prevention and management

-Hyperpituitarism

- Growth hormone excess: Acromegaly and gigantism: disfiguring and debilitating somatic growth disorder that is characterized by bone and soft tissue overgrowth
- Common causes: benign pituitary adenoma, hypersecretion of GH, increased levels of IGF-I, tumors
- Epidemiology: Incidence: 0.4 cases per 100,000 per year, prevalence: 5-9 cases per 100,000

Signs and Symptoms	
Characteristic facies	Backache (dorsal kyphosis)
Coarse, oily skin	Barrel chest (thoracic kyphosis and rib elongation)
Prognathism	Voice changes - deepening, huskiness due to increased cartilage in larynx
Soft tissue hypertrophy	In children and adolescents - accelerated linear growth, gigantism
Spade-shaped, fleshy, moist hands and feet	Hyperhidrosis of the face, head, hands, feet (enlargement of sweat and sebaceous glands)
Increase in hat, ring, glove, and shoe size	Heel pad thickness
Skin folds (including thickening of the nasolabial folds and the scalp) and skin tags	Sleep apnea
Headaches	Sensorimotor polyneuropathy
Joint stiffness or pain	Insulin resistance and glucose intolerance
Sensation of weakness in arms and legs	Hypertriglyceridemia
Diplopia	Hypertension
Carpal tunnel syndrome	Cardiovascular abnormalities
Osteoarthritis	

- Prognosis: if a complete cure is achieved and no permanent cardiovascular or pulmonary complications, including sleep apnea and diabetes, are present, then mortality rate is the same as that for normal individuals
- Complications: diabetes mellitus, hypertension, cardiovascular and pulmonary disease, sleep apnea, and colon cancer
- Diagnosis: serum IGF-I is the best screening test for acromegaly, oral glucose tolerance test, imaging techniques
- Therapy should be directed at normalizing GH and IGF-I levels, surgery, medical therapy (dopamine agonists)
- Dental/Oral findings:
 1. Characteristic facies (frontal bossing, enlargement of nose and lips)
 2. Prognathism
 3. Malocclusion
 4. Increased spacing between the teeth (intradental separation)
 5. Macroglossia
 6. Temporomandibular arthritis
 7. Macrodontia

Dental Considerations

1. Consult with physician as appropriate
2. SBE consideration
3. Management of craniofacial abnormalities
4. Sedation considerations: sleep apnea (50% of patients), snoring due to increased soft tissue mass in the airways

VI. ADDITIONAL READINGS AND WEB SITES

1. Williams Textbook of Endocrinology. 10th ed. Philadelphia: W. B. Saunders; 2003.
2. Little, James W. Thyroid Disorders. Part I: Hyperthyroidism, Oral surgery, oral medicine, oral pathology, oral radiology and endodontics. 2006; 101(3): 276-284.
3. Vaughan, ED Jr. Diseases of the adrenal gland. Med Clin North Am. 2004; 88(2): 443-66.

http://care.diabetesjournals.org/cgi/content/full/28/suppl_1/s37

Chapter 21: HEMATOLOGIC DISORDERS

- I. ANEMIAS**
- II. BLEEDING DISORDERS**
- III. ORAL EVALUATION**
- IV. MANAGEMENT**
- V. ADDITIONAL READINGS**

I. ANEMIAS

- Anemia is a reduction of the red cell volume, hemoglobin concentration below the range or values that occur in healthy persons (12 – 18g/100ml)
- Diagnosis based upon:
 - Clinical history
 - Physical examination
 - Drug history/Family history
 - Laboratory investigation
- Anemias resulting from inadequate red cell or hemoglobin production:
 - congenital pure red cell anemia (eg. Diamond-Blackfan Syndrome)
 - anemia of chronic renal disease
 - anemia of infection, inflammation, and cancer megaloblastic anemias
 - (eg. Vit B12 deficiency or malab sorption; folic acid deficiency or malabsorption)
 - microcytic anemias (eg. iron deficiency; lead poisoning)
- Anemias resulting from increased destruction of red cells (hemolytic anemias)
 - intrinsic abnormality (eg. hereditary spherocytosis)
 - enzymatic defects (eg. G6PD deficiency)
 - defects in hemoglobin synthesis (eg. sickle cell disease, thalassemia)
- Anemias resulting from extracellular abnormalities
 - infections (eg. mononucleosis)
 - autoimmune (eg. systemic lupus erythematosus)
 - transfusion reactions

Dental Considerations

- **Low risk patient** (cause of anemia known, cause corrected, normal hematocrit or cause of anemia known, on therapy, asymptomatic, normal hematocrit): Treat as normal dental patient
- **High risk patient** (patient receiving repeated transfusions to prevent symptoms of anemia; patient with abnormal pre-op screening hemogram; patient with history of ongoing hemorrhage; patient with a bleeding disorder and anemia):
 - hematology consult; may require transfusion of RBCs
 - defer elective treatment until medical status is optimal
 - treatment goals aimed at minimizing stress
 - deep sedation, general anesthesia and invasive surgical procedures may require hospitalization

II. BLEEDING DISORDERS

1. Clotting Factor Deficiencies
 - Congenital:
 - hemophilia A (Factor VIII deficiency)
 - hemophilia B (Factor IX deficiency)
 - von Willebrand Disease (Factor VIII deficiency and platelet function abnormality)
 - others (includes hypercoagulation (eg. protein C))
 - Factor VIII and factor IX cases may develop inhibitor which is antibody to Factor and may require activated factor to bypass the inhibitor and need to be managed with the hematologist
 - Classification
 - Mild (> 5%)

Moderate (1 –5%)
Severe (<1%)

- Acquired
 - hepatic disease
 - malabsorption problems
 - heparin therapy
 - drug induced
 - vitamin K deficiency
- 2. Thrombocytopenia
 - Bone marrow failure
 - aplastic anemia
 - leukemia
 - metastatic cancer
 - folate deficiency
 - vitamin B12 deficiency
 - drug induced
 - Peripheral platelet destruction
 - thiazide diuretics
 - quinidine
 - gold salts
 - sulfonamides
 - Hypersplenism
 - partial hypertension
 - inflammatory disease
 - cancer
 - Immune Thrombocytopenic Purpura (ITP)
 - Thrombic Thrombocytopenic Purpura (TTP)
- 3. Platelet Dysfunction
 - Drug induced
 - aspirin
 - ibuprofen
 - naprosyn
 - Congenital
 - von Willenbrand Disease
 - Secondary complication
 - uremia

III. ORAL EVALUATION

- History: frequent nose bleeds; heavy menstrual flow; easy bruising; excessive bleeding following surgery; family history of bleeding disorders; known history of bleeding disorders; current medications
- Physical findings: petechiae and ecchymosis; generalized spontaneous gingival hemorrhage
- Laboratory Screening: prothrombin time (PT); partial thromboplastin time (PTT); platelet count; bleeding time
- Hematology Consult: mandatory in the presence of any significant historical, physical, or laboratory finding(s)

IV. MANAGEMENT

- Low risk patient: (patient with normal PT, PTT, platelet count and bleeding time; non significant history; no physical findings)
 - treat as normal dental patient
- Moderate risk patient: (patient with juvenile rheumatoid arthritis on chronic high dose aspirin therapy)
 - dental treatment plan developed and implemented with documented approval by physician
 - hemophilia treated with appropriate factor replacement;
 - usually bring to 40-50% of normal for restorative treatment and 80 - 100% for extractions
 - mild to moderate Factor VIII may be treated with vasopressin acetate (DDAVP), a non-blood derivative that releases factor from endothelium

Amicar™ as anti-fibrinolytic supplement following treatment

- High risk patient: (patients with known bleeding disorders; patients with abnormal coagulation tests)
 - hematology consult mandatory
 - medical management varies with the specific defect
 - hospitalization may be indicated depending on medical management and the relative invasiveness of the dental procedure
 - nasotracheal intubation may be contraindicated

V. ADDITIONAL READINGS

1. McDonald RE, Avery DR, Dean JA: Dentistry for the Child and Adolescent. Chapter 24, 8th edition 2004.
2. Rogers GP, Young NS: Bethesda Handbook of Clinical Hematology. Lippincott Williams and Wilkens 2005.
3. Gomez-Moreno G, Cutando-Soriano A et al. Hereditary blood coagulation disorders: Management and dental treatment. J Dent Res 84:978-985, 2005.
4. Buchanan GR. Thrombocytopenia during childhood: What the Pediatrician Needs to Know. Pediatrics in Review 26:401-410, 2005.

Chapter 22: INFECTIOUS DISEASES

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_AntibioticTherapy.pdf

- I. PREVENTION (T)
- II. BACTERIAL INFECTIONS
- III. VIRAL INFECTIONS (T)
- IV. FUNGAL INFECTIONS
- V. PARASITE INFECTIONS
- VI. ADDITIONAL READINGS AND WEB SITES

I. PREVENTION

DEPARTMENT OF HEALTH AND HUMAN SERVICES • CENTERS FOR DISEASE CONTROL AND PREVENTION Recommended Childhood and Adolescent Immunization Schedule UNITED STATES • 2006																		
Vaccine ▾	Age ►	Birth	1 month	2 months	4 months	6 months	12 months	15 months	18 months	24 months	4–6 years	11–12 years	13–14 years	15 years	16–18 years			
Hepatitis B ^a	HepB	HepB	HepB	HepB ^b		HepB					HepB Series							
Diphtheria, Tetanus, Pertussis ^c			DTaP	DTaP	DTaP		DTaP				DTaP	Tdap		Tdap				
<i>Haemophilus influenzae type b^d</i>			Hib	Hib	Hib ^b	Hib												
Inactivated Poliovirus			IPV	IPV		IPV				IPV								
Measles, Mumps, Rubella ^e						MMR				MMR	MMR							
Varicella ^f					Varicella					Varicella								
Meningococcal ^g											MCV4	MCV4						
Pneumococcal ^h			PCV	PCV	PCV	PCV				PCV	PPV							
Influenza ⁱ					Influenza (Yearly)					Influenza (Yearly)								
Hepatitis A ^j										HepA Series								

This schedule indicates the recommended ages for routine administration of currently licensed childhood vaccines as of December 1, 2005, for children through age 18 years. Any dose not administered at the recommended age should be administered at any subsequent visit when indicated and feasible. ■ Indicates age groups that warrant special effort to administer those vaccines not previously administered. Additional vaccines may be licensed and recommended during the year. Licensed combination vaccines may be used whenever any components of the combination are indicated and other components of the vaccine are not contraindicated or approved by the Food and Drug Administration for that dose of the series. Providers should consult the respective ACIP statement for detailed recommendations. Clinically significant adverse events that follow immunization should be reported to the Vaccine Adverse Event Reporting System (VAERS). Guidance about how to obtain and complete a VAERS form is available at www.vaers.hrsa.gov or by telephone, 800-822-7967.

■ Range of recommended ages ■ Catch-up immunization ■ 11–12 year old assessment

*Published annually (January) in *Pediatrics*, Journal of the American Academy of Pediatrics

II. BACTERIAL INFECTIONS

Impetigo contagiosa

- A superficial infection of the skin
- Most commonly seen in preschool children, endemic in populations with poor hygiene, crowding living conditions, and hot, humid climates
- Breaks in the skin (dermatologic disease, wound healing, trauma) allow infection by group A beta hemolytic streptococci and *Staphylococcus aureus*
- Tiny pustules and vesicles that later rupture without pain; Honey colored crusting; Regional lymphadenopathy and pruritis are present without systemic manifestations of infection; non-bullous and bullous forms
- Usually a self-limiting disease with appropriate topical and/or systemic antibiotics
- Rare complications uncommon; include cellulitis, lymphadenitis, acute poststreptococcal glomerulonephritis
- Diagnosis by history and clinical presentation, culture and sensitivity testing also used
- Differential diagnoses include contact dermatitis, herpes simplex virus infection, varicella, scabies, allergic stomatitis, other vesiculobullous and burns
- Treatment with topical antibiotic ointment, mupirocin, applied to the area tid for 7-10 days, treatment of choice; Widespread or complicated impetigo, oral cephalexin 25-50mg/kg/d divided into tid/qid dosing, oral erythromycin or clindamycin for children who are penicillin allergic; cleansing of wounds and improved personal hygiene
- Perioral skin involvement, intraoral lesions not seen
- Elective dental treatment should be deferred until resolution of lesions occurs

Bacterial Pharyngitis

- Diffuse erythema and inflammation of the tonsils and their pillars, petechiae of the soft palate, and pharynx with anterior cervical lymphadenopathy
- Occurs in people of all ages with children ages 5 to 15 most common
- Caused most commonly by group A beta hemolytic streptococci; other bacterial pathogens are rare
- Onset characterized by complaints of headache, abdominal pain, vomiting, fever, and malaise; throat pain and soreness is later finding
- Usually a 5 to 7 day course, symptoms commonly resolve spontaneously
- Complications for streptococcal pharyngitis include dehydration with refusal to feed, glomerulonephritis, rheumatic fever, peritonsillar abscess, otitis media, sinusitis
- Diagnosis made by culture
- Differential diagnoses include viral pharyngitis, chronic allergic rhinitis, mononucleosis, peritonsillar abscess
- Treated with oral penicillin VK, 125-250 mg/kg tid or qid for 10 days, oral azithromycin 12 mg/kg/d every day for 5 days for penicillin allergic children; antipyretics including tylenol or ibuprofen and proper hydration
- Elective dental treatment should be deferred until completion of antibiotic therapy

Gonococcal Stomatitis

- Sexually transmitted disease causing urogenital inflammation and pharyngitis in some cases
- Highest incidence is for persons aged 15-24 years; high rate of recovery in sexually abused children
- *Neisseria gonorrhoeae* infection through genital or oral contact
- Incubation is 1 day to 2 weeks
- Oral lesions resolve with proper systemic antibiotics
- Complications include further genitourinary involvement, disseminated gonococcal infection, interpersonal disease transmission, continued child abuse
- Diagnosis made via culture or gonorrhea DNA probe
- Differential diagnoses include herpes simplex virus, streptococcal pharyngitis, acute necrotizing ulcerative gingivitis
- Treated by single dose oral cefixime 400 mg for adolescents; drug of choice; ceftriaxone 25-50 mg/kg IM single dose for infants and children
- Pharyngitis in addition to involvement of gingiva, tongue, soft palate; resembles ANUG
- Dental provider must consider and rule out sexual abuse; education of sexually active adolescents on transmission via oral sexual contact

Syphilis

- Bacterial infection acquired through vertical transmission or through sexual contact
- Adolescents and young adults at highest risk, increased incidence among lower socioeconomic groups, urban, overcrowded populations, drug use and sexual activity are risk factors

- *Treponema pallidum*, is causative agent
- Four types of disease
 - Primary form: painless, slightly tender chancre, present at site of exposure, 3 week incubation
 - Secondary form: constitutional symptoms, maculopapular rash, renal, hepatic, ophthalmologic signs, meningitis
 - Tertiary form: gummatous lesions, neuronal involvement with features of stroke, aortic dilatation, changes in personality, affect, reflexes, intellect, and speech
 - Congenital form: earliest sign is syphilitic rhinitis, mucous patches, condyloma lata, hepatomegaly, neurosyphilis, dental anomalies including hutchinson's incisors (45% of patients), mulberry molars (22% of patients), and perioral rhagades (15%)
- Infection is highly sensitive to penicillins, prognosis is excellent with early diagnosis
- Diagnosis via serologic testing, treponema specific tests, and darkfield microscopy; oral specimens can not be used as other treponeme species are part of normal oral flora
- Treated with penicillin G benzathine, 50,000 U/kg IM either single dose or weekly for 3 doses depending on the duration of disease
- Oral findings include primary chancre (oral mucosa, gingiva, or lips), mucous patches, oral gummas, and congenital dental anomalies
- Dental providers should verify that antibiotic therapy has been given prior to elective dental procedures

Tuberculosis

- Chronic granulomatous infection of the lungs
- Caused by *Mycobacterium tuberculosis* after exposure to infected aerosolized droplets
- Occurs in all ages but young children and elderly at greatest risk of mortality
- Tuberculin skin test used as screening tool
- Stages of TB in children
 - Stage 1: exposed to adult with contagious TB, negative tuberculin skin test
 - Stage 2: positive tuberculin skin test, no signs or symptoms of active disease
 - Stage 3: disease present, signs and symptoms depend on location, radiographic abnormalities are present
 - Stage 4: TB with no current disease, can see abnormal yet stable radiographic findings, positive tuberculin skin test, negative bacteriologic studies
- Pulmonary and extra-pulmonary disease
- Definitive diagnosis of active disease based on isolation of organisms from secretions or biopsy specimen; difficult to obtain
- Common drug regimen for pulmonary TB: 6 month course of INH (10-20mg/kg PO every day, not to exceed 300mg/day) with rifampin (10-20mg/kg PO every day not to exceed 600mg/day) along with 2 months of pyrazinamide (15-30mg/kg PO every day, not to exceed 2g/day)
- Health care workers watch patients take medications to ensure compliance (directly observed therapy) and reduce relapse of disease and drug resistance
- Associated with chronic oral ulcers, granulomas, jaw osteomyelitis, cervical lymphadenitis, and salivary gland involvement

- Complications include military TB (low grade fever, malaise, weight loss, and fatigue common especially in young children) tubercular meningitis, pleural effusions, pneumothorax, gastrointestinal perforation, pericardial effusion, and hydronephrosis
- Consultation with patient's physician should take place before dental treatment; Treatment should be deferred when contagious TB is present

III. VIRAL INFECTIONS

Hand-Foot-Mouth Disease

- Coxsackie A16 or enterovirus 71 infection
- Incubation period of 3-6 days after exposure to infected nasal or oral secretions, fecal matter, or aerosolized droplets
- Can be sporadic or epidemic
- Mild course common; worse in infants and young children
- Prodrome including fever, anorexia, malaise, abdominal pain, sore mouth, cough
- Oral vesicles with rapid ulceration, 5-10 in number; occasional regional lymphadenopathy
- Skin lesions present for 5-10 days, usually asymptomatic
- Diagnosis by clinical presentation and distribution of lesions
- Differential diagnoses includes aphthous stomatitis, chickenpox, erythema multiforme, herpes simplex virus, herpangina
- Rare complications: dehydration, meningoencephalitis, myocarditis
- Supportive care, continued hydration, proper handwashing
- Defer elective dental treatment until oral and skin lesions resolve

Herpangina

- Coxsackie A virus infection
- Incubation time is 7-14 days after fecal-oral exposure, respiratory droplets, of fomites
- Summer and fall months; temperate climates
- Infants and young children affected
- High fever (101-104°F), malaise, sore throat, anorexia, lymphadenopathy
- Oropharyngeal vesicles and ulcerations; soft palate, uvula, tonsils, anterior pillars, posterior pharynx involved
- Diagnosed by clinical findings
- Self-limiting and mild course
- Differential diagnoses: herpes simplex virus, bacterial pharyngitis, infectious mononucleosis, hand-foot-mouth disease
- Supportive care with antipyretic agents i.e. ibuprofen, acetaminophen, topical anesthetics, and continued fluids
- Defer elective dental treatment until oral lesions resolve

Acute Nasopharyngitis

- Upper respiratory tract infection or “common cold”

- Most common infectious disease of children, months of September to April
- More than 150 various viral agents; rhinoviruses most common
- Transmitted by aerosolized droplets or direct contact
- Rhinorrhea, nasal congestion, sneezing, sore throat, malaise, headache, cough, thickened nasal secretions occur
- Clinical course 7-14 days, acute phase lasts 2-4 days, prolonged course in infants and young children
- Complications: acute otitis media, sinusitis, croup, bronchiolitis
- Medical care includes hand washing, bed rest, fluids, decongestants for children older than 6 months, humidified air, antipyretic agents i.e. ibuprofen, acetaminophen
- Defer elective dental treatment until symptoms resolve

Influenza

- Acute infection of the respiratory tract i.e nose, throat, and lungs
- Caused by 3 types of viruses (A,B,C); highly contagious
- Sporadic, epidemic, or pandemic occurrence, usually in winter months, most common in young children
- Exposure to infected respiratory secretions, airborne droplets, direct contact with fomites
- Onset of symptoms 2-3 days after exposure; nonproductive cough, headache, myalgia, chills, fatigue, sore throat (50% of cases), pharyngitis, conjunctivitis, lymphadenopathy, febrile seizures, vomiting, otitis media, diarrhea
- Diagnosis by culture; first 3 days after onset of symptoms
- Differential diagnoses: pharyngitis, respiratory syncytial virus, pneumonia (chest xrays taken for exclusion)
- Prevention by inactivated influenza viral vaccines; child at risk for complications (i.e heart disease, cystic fibrosis, asthma, diabetes mellitus)
- Supportive medical care: acetaminophen (avoid aspirin due to Reye syndrome), humidified air, fluid maintenance, bed rest, hand washing
- Elective dental treatment should be deferred until all symptoms resolve

Acute Herpetic Gingivostomatitis

- Initial clinical manifestation of herpes simplex virus (HSV-1) infection; rarely caused by HSV-2 infection
- Primarily occur in young children upon first exposure to virus; also seen in immunocompromised children (cancer, HIV infection)
- Ranges from subclinical or mild infection to severe forms
- Incubation period is usually one week before emergence of oral lesions
- Virus is present in saliva and vesicular exudates
- Oral findings are dominated by small punctuate vesicles that rupture to form shallow ulcers with smooth margins surrounded by a red halo; lesions occur in all areas of the mouth with gingiva and lips most common; gingiva shows acute inflammation
- Oral pain, dysphagia, fever, lymphadenopathy, and dehydration common
- Diagnosis typically made by clinical presentation

- Disease is self-limiting in 7-14 days
- Dehydration a concern in young children; clinical presentation with slowed capillary refill, minimized urine production, dried mucous membranes i.e. sweat, tears, saliva; tachycardia, tachypnea
- Treatment is supportive including acetaminophen for fever and pain, cold liquids and bland foods, isolation of infectious child important to reduce spread of infection
- Elective dental treatment should be deferred while child is infectious

Recurrent Herpes Simplex Virus

- Recurrent herpes simplex virus infection due to a reactivation of latent disease present in trigeminal ganglia
- Suggested to occur secondary to stress, fever, hormonal imbalance, sunlight
- Prodromal period with itching sensation precedes appearance of lesions
- Herpes labialis presents with fluid-filled vesicles on the lip and adjacent perioral skin that later rupture, coalesce, ulcerate, and heal by brownish crusted lesions
- Intraoral herpes occur on the hard palate and maxillary gingiva; clusters of symptomatic punctuate ulcers; often occurs secondary to dental treatment in the area and can be more involved with systemic features in immunocompromised patients
- Autoinoculation of hands or finger causes herpetic whitlow
- Diagnosis through clinical presentation; biopsy, cytology, culture, and serology also useful in some cases
- Supportive care is only course of action for intraoral herpes
- Use of penciclovir 1% cream (Denavir) or 10% n-docosonal cream (Abreva) for herpes labialis shortens the mean healing time if applied during the prodromal period; systemic acyclovir is not recommended for treatment of recurrent herpes labialis in immunocompetent individuals
- Elective dental treatment should be deferred until lesions have resolved to prevent autoinoculation or interpersonal spread of the virus

Chickenpox

- Infection with human herpes virus 3, varicella-zoster virus
- Primarily affects children under 10 years of age (90% of cases)
- Seen mainly from January to May; spread by direct contact, airborne or droplet transmission
- Incubation period of 13-17 days, prodromal symptoms (anorexia, malaise, low fever) appear 24 hours before rash
- Clinical course of rash: crops of small red papules develop (days 3-4) into vesicles with erythematous base, vesicles rupture within 24 hours and become crusted; lesions can be in various stages; pruritis common
- Skin lesions begin on trunk and spread to face and scalp; distal portion of extremities not usually involved
- Oral vesicles and ulcerations
- Fever during eruption of lesions (39.4-40.6°C)
- Diagnosis made by clinical presentation; differential diagnoses include contact dermatitis, herpes simplex virus, impetigo, and urticaria

- Complications include secondary bacterial infection, pneumonia, encephalitis, thrombocytopenia, glomerulonephritis
- Mild, self-limiting course for healthy, immunocompetent children; increased severity in neonates and immunocompromised children
- Disease prevention through live attenuated vaccine; efficacy of vaccine is around 80%; latent reactivation of disease can occur later in life
- Medical care is normally supportive; maintain hydration, acetaminophen for fever, diphenhydramine 5mg/kg/d PO given tid/qid to control pruritis; antiviral therapy for immunocompromised or high risk children
- Elective dental treatment should be deferred until all lesions have disappeared
- Emergency dental care should be rendered with strict adherence to universal precautions and preferably after all lesions have crusted

Infectious Mononucleosis

- An infection by human herpesvirus 4, Epstein-Barr virus
- Transmitted by oropharyngeal secretion in saliva
- Primary and latent, secondary infections
- Incubation period is 30-50 days in adolescents with shorter period in young children
- History of fatigue and malaise for 7-14 days after incubation; sore throat, fever, headache, myalgias, nausea, and abdominal pain
- Pharyngitis is most common finding in addition to generalized lymphadenopathy and hepatosplenomegaly
- Pharyngitis can be exudative; palatal petechiae at junction of hard and soft palate; tonsillar enlargement with potential for airway obstruction
- Diagnosis through 3 classical criteria in blood work: lymphocytosis, 10% atypical lymphocytes on peripheral smear, and positive serology for EBV
- Self limiting disease although complications including hepatitis, mild thrombocytopenia, hemolytic anemia, upper airway obstruction, and splenic rupture can occur
- Immunocompetent children have full recovery in several months; hematologic and hepatic complications resolve in 2-3 months; latent infection remains
- Medical care is supportive with use of NSAIDS for fever and discomfort, bed rest needed, reduce activity and heavy lifting to prevent splenic rupture
- Elective dental treatment should be deferred until resolution of acute infection
- Emergency dental care should be coordinated with the treating physician as hematologic status may be impaired

Hepatitis B

- Acute and chronic liver disease caused by infection with hepatitis B virus
- HBV is transmitted by percutaneous or permucosal exposure to infectious body fluids, sexual contact with infected person, and perinatal vertical transmission
- Primary spread is through blood exposure
- Most common in young adults however perinatal and childhood exposure leads most often to chronic disease
- Incubation ranges from 2-5 months

- Acute clinical HBV infection includes anorexia, nausea and vomiting, malaise, myalgias and arthralgias, headache, cough, jaundice with splenomegaly, abdominal pain
- Diagnosis via blood work: elevated ALT and AST levels and presence of HBsAg in serum
- Prognosis for acute hepatitis is favorable in most patients; age and immunosuppression prolong course and increase disease severity
- Complications include acute fulminating hepatitis with encephalopathy, aplastic anemia, chronic disease with liver cirrhosis and hepatocellular carcinoma
- Universal vaccination to all infants is recommended
- Medical care is primarily supportive for acute hepatitis
- Antiviral agents i.e. interferon alfa-2b aid in preventing transition to chronic disease or treating present chronic disease
- Strict adherence to universal precautions minimize the risk for disease dissemination in the dental office
- All healthcare workers should be vaccinated to develop immunity against HBV in case of accidental occupational exposure

Condyloma Acuminatum

- Multiple papillary or sessile areas of epithelial hyperplasia occurring the genital or oral mucosa
- Caused by human papillomavirus 6 and 11 (HPV6 and HPV11)
- Oral lesions commonly acquired through oral-genital sexual contact; also through autoinoculation from genitals to mouth
- Typically occur on nonkeratinized mucosa i.e. lips, floor of the mouth, lateral and ventral surfaces of the tongue, buccal mucosa, soft palate; rarely on gingiva
- Recurrence is common
- Treated by surgical excision or cryotherapy
- Lesions that are seen in children are suggestive of sexual abuse; proper documentation and reporting by dental professionals should be undertaken
- Other human papillomavirus-associated oral lesions include: squamous papilla (HPV6 and 11), verruca vulgaris (HPV2 and 4), and focal epithelial hyperplasia (HPV 13 and 32)

Human Immunodeficiency Virus (HIV)

- Caused normally by human immunodeficiency virus-1 (HIV-1); rarely HIV-2 in United States, more prevalent in West Africa and Europe
- Primary route of pediatric infection via maternal transmission; less commonly by infected blood products
- 4 to 25% of babies born to HIV infected mothers become HIV infected; anti-retroviral treatment during prenatal period reduces vertical transmission
- 7th leading cause of death in young children; African American and Hispanic children are disproportionately affected
- Major target cell for infection is CD4+ lymphocyte
- CD4+ lymphocyte depletion associated with increased risk for opportunistic infection
- CDC staging of pediatric HIV infection for children less than 13 years of age
- Clinical Categories: N: No, A: Mild, B: Moderate, C: Severe

Immunologic Categories: Absolute CD4+ Count, Level of Immunosuppression (1-3)

Category	Less than one year of age	1-5 years of age	6-12 years of age	>12 years of age
1 - No suppression	>1500 (>24)	>1000 (>24)	>500 (>24)	>500 (>29)
2 - Moderate suppression	>750 (>14)	>500 (>14)	>200 (>14)	>200 (>13)
3 - Severe suppression	<750 (<15)	<500 (<15)	<200 (<15)	<200 (<14)

- Classification by Clinical Category plus Immunologic Category (e.g. N1, A2, C3)
- Children whose HIV infection status is not confirmed are classified by using the above grid with a letter E (for perinatally exposed) placed before the appropriate classification code (e.g. EN2)
- Mildly asymptomatic HIV infection encompasses two or more of the following: dermatitis, hepatomegaly, lymphadenopathy, splenomegaly, parotitis, sinusitis, otitis, or upper respiratory infection
- Moderately symptomatic HIV infection includes invasive bacterial infection, persistent oropharyngeal candidiasis, HSV infection, disseminated varicella, and hematologic complications
- Late symptomatic HIV infection equals AIDS, manifestations include TB, PCP, recurrent pneumonia, chronic mucocutaneous HSV infection, toxoplasmosis
- Late disease: disseminated CMV retinitis, cryptococcal meningitis, histoplasmosis, wasting syndrome
- Medical treatment includes HAART therapy (highly active antiretroviral therapy); combination drug therapy using nucleoside reverse transcriptase inhibitors (i.e. zidovudine, abacavir, didanosine, lamivudine, stavudine, zalcitabine), non-nucleoside reverse transcriptase inhibitors (i.e. delavirdine, efavirenz, nevirapine), protease inhibitors (i.e indinavir, nelfinavir, ritonavir, saquinavir), and newly classified fusion inhibitors i.e enfuvirtide); also prophylaxis against pneumocystis carinii pneumonia (i.e. trimethoprim and sulfamethoxazole)
- Oral manifestations include fungal infections with candidiasis most common, viral infections (HSV infection), bacterial infections including necrotizing ulcerative gingivitis and/or periodontitis, hairy leukoplakia (rarely in children), non-Hodgkin's lymphoma, Kaposi's sarcoma (very rare in children), salivary gland enlargement with parotitis most common, oral bleeding due to thrombocytopenia, recurrent aphthous stomatitis, linear gingiva erythema

Treatment for oral lesions in pediatric HIV patients:

- Oral candidiasis: 5 to 7 day course; topical: nystatin suspension, 2-5mL, 4-6 times/day; clotrimazole troches 10mg tablet, 3-5 times/day; systemic: flucanazole 3-5mg/kg once daily
- Angular cheilitis: topical: imidazole cream (clotrimazole 1%, miconazole 2%, ketoconazole 2%), dispense 2-4 weeks supply, apply to area 4 times/day; combination creams also helpful; treatment of intraoral candidiasis needed
- Linear gingiva erythema: maintain optimal plaque control, chlorhexidine rinses, antifungal therapy in some cases
- NUG/P: plaque removal, local mechanical debridement, topical povidone-iodine, chlorhexidine rinses; systemic antibiotics metronidazole, clindamycin, and/or amoxicillin with clavulanate potassium

- Herpes simplex virus: systemic acyclovir
- Human papillomavirus: excision with biopsy
- Parotid swelling: usually no treatment necessary, anti-inflammatory medications for pain; severe cases respond to corticosteroids
- Necrotizing stomatitis: rare; debridement, topical povidone-iodine, chlorhexidine rinses, systemic metronidazole and prednisone
- Aphthous ulcers: can be drug induced; steroids (fluocinonide gel 0.05%, apply 5-6 times/day; also dexamethasone elixir 0.5 mg/5mL, oral rinse 4-6 times/day; may cause opportunistic oral candidiasis
- Hairy leukoplakia: rare; no treatment necessary; systemic acyclovir severe cases
- Xerostomia: uncommon; optimal plaque control, artificial saliva, salivary stimulation (i.e. sugarfree chewing gum), may be drug induced
- Dental caries and gingivitis more common; poor feeding behaviors in addition to highly cariogenic oral medications
- Consultation with the child's physician prior to provision of dental care as infection and bleeding can occur; dental treatment with universal precautions
- Dentist should perform more frequent oral health monitoring such as every 3 months

IV. FUNGAL INFECTIONS

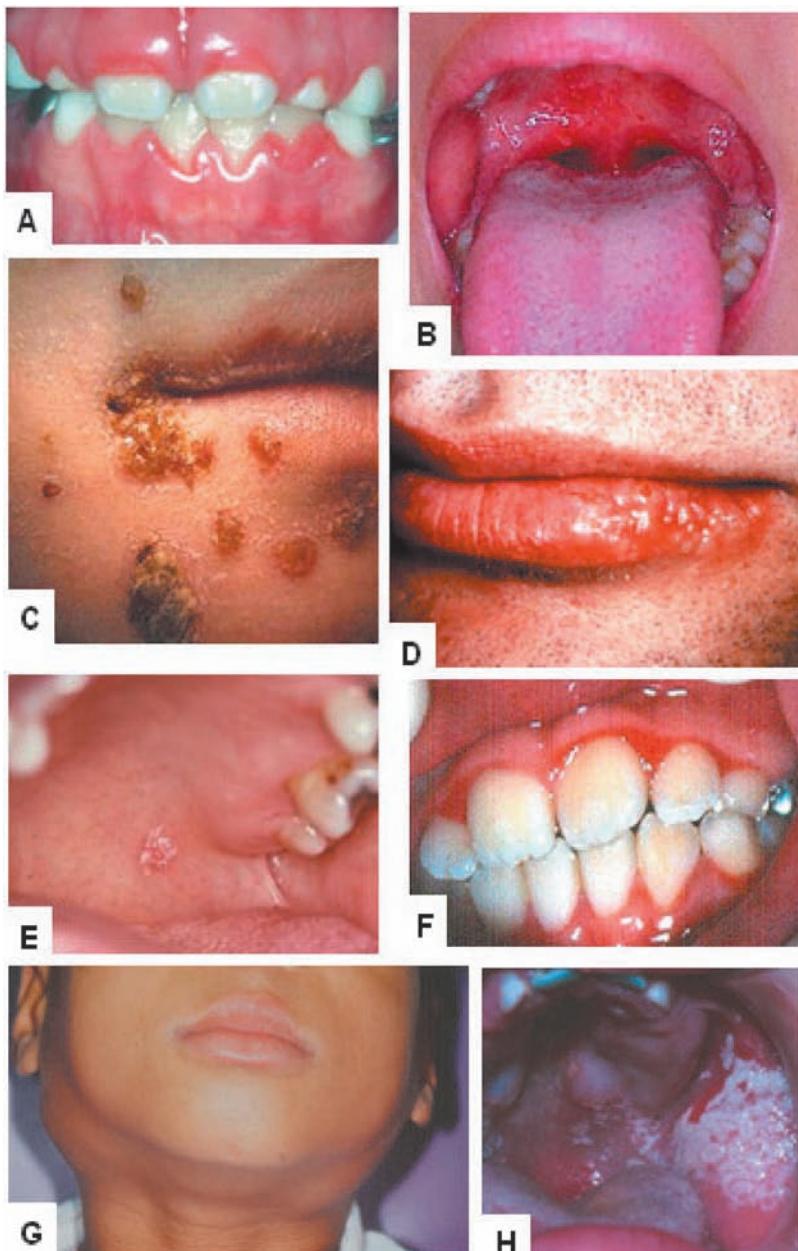
Candidiasis

- A fungal infection caused by *Candida albicans*
- Can affect skin (diaper dermatitis), nails, mucous membranes (thrush), genitals, or become systemic
- Rarely cause morbidity in the immunocompetent host
- Oral forms can be pseudomembranous (thrush) or atrophic (erythematous); thrush can involve the lips, tongue, gingiva, buccal mucosa, and palate; scraping of lesions reveals erythema and bleeding at base; glossitis also seen
- Oral burning, pain, refusal to eat with oral lesions
- Pathologic etiologies include prematurity, immunosuppression, debilitation, head and neck radiation, broad spectrum antibiotic therapy, poorly controlled diabetes mellitus, endocrinopathies, xerostomia, oral contraceptives
- Diagnosis made by clinical presentation and cytology
- Complications include pneumonia, endocarditis, hepatitis, meningitis, cystitis
- Treated with topical or systemic anti-fungal agents; nystatin oral suspension is the drug of choice for oral candidiasis: infants 1mL to each side of mouth qid until 48 hours after lesions resolve, children 2-3mL to each side of mouth as above; systemic antifungal agents should be reserved for immunocompromised individuals or systemic infection, for oropharyngeal candidiasis in children, fluconazole 6mg/kg PO on day 1 followed by 3mg/kg PO on day 2 or ketoconazole for children older than 2 years of age, 3.3-3.6 mg/kg/d PO every day until lesions resolve
- HIV infection must be ruled out when oral candidiasis presents in children
- Dental treatment should be deferred until lesions resolve

V. PARASITE INFECTIONS

Lice

- Ectoparasite infection involving the scalp (*pediculosis capitis*) and body (*pediculosis corporis*) of their host
- Lice feed on human blood, lay eggs, and become infested in the form of nits in warm portion of the body
- Transmitted through fomites including clothing, headgear, combs, and hair brushes
- Pruritis is the first sign of infestation
- Young children in daycare centers or schools common; head lice more common
- Secondary bacterial infection and regional lymphadenopathy occurs
- Medical care involves medications for patient and environmental control measures; antihelminthics i.e. permethrin 5% prescription strength cream, wash hair with nonmedicated shampoo, rinse, and then apply cream, allow to remain in place for 10 minutes, then rinse thoroughly, may require an additional treatment 7-10 days later; control measures include washing fomites in very hot water ($>131^{\circ}\text{F}$ or 55°C)
- Elective dental treatment should be deferred until second application of antihelminthic agent is used; proper control measures should be conducted if lice are found on a patient



Primary herpetic gingivostomatitis with acute gingiva inflammation (Plate A); Plate B depicts punctuate, shallow ulcerations present on the soft palate of an individual with hand-foot-and-mouth disease; Plate C shows honey colored crusts of the perioral region indicative of impetigo; Coalescence of multiple vesicles on the lower lip in an individual having a recurrence of herpes labialis (Plate D); Plate E shows a papillary, exophytic lesion of the soft palate diagnosed by histology as a squamous papilloma; Linear gingiva erythema in a child with HIV infection (Plate F); Bilateral parotitis present in a HIV infected child (Plate G); Plate H displays pseudomembranous candidiasis infection of the buccal mucosa and palate.

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Chapter 23: NEPHROLOGY

- I. DEFINITIONS**
- II. MEDICAL TREATMENT OF END STAGE RENAL DISEASE (ESRD) (T)**
- III. PROPHYLACTIC ANTIBIOTICS PRIOR TO DENTAL TREATMENT**
- IV. ORAL AND DENTAL MANAGEMENT**

V. ADDITIONAL READINGS

I. DEFINITIONS

RENAL INSUFFICIENCY

A patient's renal reserve can compensate to a point at which < 50% of renal function remains. Once the damage is past the point of compensation, its function is initially mildly to moderately diminished, resulting in an impaired ability to maintain the internal environment.

RENAL FAILURE

It is characterized by a reduction in glomerular filtration rate (GFR) which is the most valid parameter of renal function. The kidney function deteriorates to the point of chronic abnormalities in the internal environment thus normal homeostasis cannot be maintained leading, for example, to metabolic acidosis and hypocalcemia.

END-STAGE RENAL DISEASE (ESRD)

It is a chronic, irreversible, progressive disease characterized by the destruction of 50% to 75% of the nephrons which leads to the retention and accumulation of excretory products in the body and the decreased endocrine and metabolism functions of the kidney (uremic syndrome). The most common known causes of ESRD are diabetes mellitus, hypertension and chronic glomerulonephritis.

II. MEDICAL TREATMENT OF ESRD

Conservative management (e.g., dietary modification) fails to halt progression of renal damage when the GFR falls to < 10 ml/min/1.73 m² of body surface area (normal: 100–150 ml/min) and blood urea nitrogen levels rise to 100 to 150 mg% (normal: 10 to 20 mg%) or the clinical status of the child requires a more aggressive mode of treatment either in the form of dialysis or renal transplant. The most common cause of death in renal disease is cardiovascular complications, followed by infection and malignancy.

DIALYSIS

It is an artificial means of removing nitrogenous and other toxic products of metabolism from the blood and to maintain fluid and electrolyte balance. The patient's blood is separated from the dialysis fluid (dialysate) by a membrane which allows water and toxins, but not blood cells, to pass out of the blood. Dialysis replaces the normal metabolic function of the kidneys but it does not correct the endocrine abnormalities associated with renal failure.

There are two types:

- **PERITONEAL DIALYSIS (PD)**

An elastic catheter is surgically placed in the peritoneal cavity with intermittent infusion and drainage of a sterile electrolyte solution in a plastic bag that cleans the blood by osmosis. The major types of PD are continuous ambulatory (CAPD), continuous cycling (CCPD), and nighttime intermittent (NIPD). PD does not require heparinization, decreases the incidence of blood-borne disease and allows the patient greater freedom than hemodialysis.

- **HEMODIALYSIS (HD)**

The patient's blood is cleansed usually 3 times a week in a hospital setting by feeding unclotted blood through an extracorporeal artificial kidney known as dialyzer. The patient is given anticoagulants (e.g., heparin) to facilitate blood exchange and to maintain patency

of the arteriovenous shunts and fistulas created to access the patient's bloodstream through direct anastomosis of native vessels or by the use of artificial grafts.

- RENAL TRANSPLANTATION

Although patient survival is approximately equal in patients receiving a transplant and those treated by dialysis, there are major differences when considering quality of life issues such as less dietary restrictions and no time for dialysis. The 5-year survival rates in children approach 65% for a cadaveric graft and up to 80% in cases of living-related donors. Patients will be on long-term immunosuppressive drugs to prevent graft rejection which put them at risk for opportunistic infections and neoplasms. Hypertension, recurrence of the baseline disease and appearance of a new kidney disorder (transplant nephropathy) are also possible

ORAL MANIFESTATIONS OF ESRD / RENAL TRANSPLANTATION

- | | |
|--|--|
| <ul style="list-style-type: none"> • malodor • altered taste sensation (metallic taste) • xerostomia
 • glossitis • dysesthesia of the lingual nerve • parotitis • swollen submandibular glands • mucosal pallor • enamel hypoplasia / opacities • delayed or altered tooth eruption • eruption of rootless teeth
 • increased deposition of calculus • bone demineralization
 • decreased trabeculation ("ground glass") • loss of lamina dura • radiolucent giant cell or fibrocytic lesions • soft tissue calcifications • lytic areas of bone • widening of the periodontal ligament | <ul style="list-style-type: none"> • gingival overgrowth (in patients using nifedipine and/or cyclosporine) • dental pulp calcification / pulp narrowing • increased risk for development of squamous cell carcinoma, Kaposi's sarcoma and non-Hodgkins lymphoma • uremic stomatitis • oral hairy leukoplakia • malocclusion • increased risk of jaw fracture • low caries • tooth erosion (due to vomiting) • tooth mobility • extrinsic staining of teeth (due to ferrous sulfate) • abnormal bone healing after extractions • oral secondary infections due (immunosuppression) • oral metastases from renal tumors |
|--|--|

III. PROPHYLACTIC ANTIBIOTICS PRIOR TO DENTAL TREATMENT

The American Heart Association (AHA) guidelines do not address prophylactic antibiotic use prior to dental procedures in ESRD and renal transplantation. Endovascular infections may be induced by transient bacteremia such as valvular endocarditis and infections of the vascular access device, particularly with synthetic vein grafts and in the first 6 months following a fistula placement. In these cases as well as in the presence of indwelling venous catheters, antibiotic prophylaxis following the AHA recommendations may be beneficial. Peritoneal catheters are not a risk factor for endarteritis or endocarditis, but some patients on PD may have an arterio-venous access or an indwelling venous catheter which warrant the use of antibiotic prophylaxis. Other factors that have been suggested to put the HD patient at higher risk for infections include:

uremia, which is associated with immune abnormalities, chronic increase in cardiac output, which may damage cardiac valves.

The dentist should consider cases on an individual basis and discuss the management with the nephrologist, taking into consideration:

- patient's compliance,
- drug allergies,
- drug intolerance,
- recent or chronic use of antibiotics,
- length of dental care,
- renal disease status,
- drug interactions.

IV. ORAL AND DENTAL MANAGEMENT

A. DURING RENAL THERAPY AND BEFORE TRANSPLANTATION

Goals of dental exam

- Identify, stabilize or eliminate existing and potential sources of oral infection and local irritants
- Oral hygiene training
- Radiographic exam to identify pathoses and bone changes caused by renal osteodystrophy in the jaws
- Patient and caretaker education

Medical history review

- A. Review of systems:
 - a. cardiovascular history: hypertension, valvulopathy, infective endocarditis, surgeries, congestive heart failure
 - b. anemia
 - c. history of bone involvement: secondary hyperparathyroidism, osteomalacia, vit D deficiency, osteomalacia, osteopenia
 - d. immune system:
 - i. history of infections, including bloodborne pathogens (HIV, hepatitis B and C), and tuberculosis
 - ii. level of immunocompromise
 - e. history of prolonged bleeding
- B. Cause and severity of the renal disease
- C. Medical management:
 - a. physician's name and phone number
 - b. diet modifications
 - c. medications
 - i. dosage, schedule, route, allergies
 - d. dialysis type and regimen
 - i. access type
 - ii. use of anticoagulants

e. transplant	
Dental history review	Past care, symptomatic teeth, trauma hx, etc
Preventive strategies	
Toothbrushing	Regular or electric soft brush 2 to 3 times daily, regardless of the hematological status
Flossing	Once daily
Oral Rinses	Chlorhexidine rinses if the patient has poor oral hygiene or periodontal disease
Diet	Encourage a non-cariogenic diet and alert the caretakers to the high sucrose content of pediatric oral medications
Fluorides	Fluoridated paste and other agents for patients at risk for caries and xerostomia Systemic fluoride supplements are not recommended because even moderate renal impairment is likely to lead to fluoride retention
Xerostomia	Use of sugar-free chewing gum and candy, special dentifrices, saliva substitutes, frequent sipping of water, oral moisturizers, bland rinses, fluoride (gel, varnish, etc)
Education of Patients and Caretakers	Discuss the importance of optimal oral care, the oral effects of drugs, the risk of invasive dental procedures in patients using bisphosphonates
Before dental procedures	
• Consult with physician before any invasive dental care is provided. Deferral of treatment may be needed until disease is adequately controlled. In most cases when the disease is well controlled, there are no contraindications for routine dental care.	
Check: immunosuppression	
absolute neutrophil count (ANC) status	< 1,000/mm ³ : defer elective care until it rises
bleeding tendency	Platelets > 75,000/mm ³ : no additional support needed but be prepared to treat prolonged bleeding < 75,000 mm ³ : consult physician before providing dental care Coagulation tests INR, PT, PTT, APTT: for patients with liver dysfunction, coagulation problems and on HD hematocrit, hemoglobin Consult physician about supplemental corticosteroids Consult physician
anemia status	
adrenal status	
antibiotic prophylaxis	
Dental procedures	
Endodontics	
primary teeth	Failure of pulpotomies/pulpectomies during immunosuppression can have significant impact on medical therapy; if not sure about the pulpal status, extraction is indicated
permanent teeth	Symptomatic/non-vital = root canal tx at least 1 week before the transplant; If not possible, extract

	Asymptomatic/non-vital = root canal tx can be delayed until hematological status is stable
Orthodontics	<p>Smooth, well fitting appliances</p> <p>Good oral hygiene = keep appliances</p> <p>Poor oral hygiene = remove appliances</p> <p>Consider removing if patient is at risk for moderate/severe gingival overgrowth or in cases of poor oral hygiene</p> <p>Specific guidelines regarding force and pace need to be defined for patients with bone involvement</p>
Periodontics	Consider gingivectomy in moderate and severe cases
Oral Surgery	<p>Treat aggressively and consider hospitalization for severe infections and major dental procedures spontaneous dental abscesses may occur due to the formation of globular dentin with clefts and defects in the dentinal tubules in vitamin D resistant rickets</p>
full mouth braces	<p>extractions</p> <p>At least 7 – 10 days prior to the transplant</p> <p>No clear recommendations for use of antibiotics following extractions</p>
gingival hypertrophy	<p>Extract:</p> <p>root tips, teeth with periodontal pockets > 6mm, teeth with acute infections, significant bone loss, involvement of the furcation, non-restorable teeth individual assessment of impacted teeth</p>
orofacial infections	<p>bleeding disorder</p> <p>osteonecrosis risk</p> <p>drug prescription</p> <p>Currently there are no recommendations for its prevention and treatment following extractions, excisional biopsies, and preparation and placement of dental implants in patients who have used or are using bisphosphonates</p> <p>Discuss dose adjustment with physician</p> <ul style="list-style-type: none"> - avoid nephrotoxic drugs (acyclovir, aspirin, nonsteroidal antiinflammatory drugs, high doses of acetaminophen) - penicillins can be used in normal doses, except for high potassium pens which are acceptable for short-term use but not for long-term due to its high potassium levels - aminoglycosides, tetracyclines and cephalosporines should be avoided due to nephrotoxicity - most narcotics can be used safely, except for meperidine which forms a metabolite that may accumulate and cause seizures - local anesthetics are safe and well tolerated
Specific considerations	
dialysis patients	<p>Dental care should be done soon after dialysis</p> <p>Concern about the use of heparin in HD is overstated since the volume used is small and the risk of excessive bleeding is minimal</p>

	Avoid the day before dialysis because of increasing uremia and consequent failing platelet function Newly placed grafts for HD are at higher risk for direct bacterial seeding than established ones, thus no elective dental procedures should be done 4 to 6 weeks following new graft placement or surgical revision Do not measure blood pressure or use IV medications in the arm with shunt Avoid dental care during episodes of peritoneal infection
transplant patients	Ideally, all dental care should be completed before the transplant; when not feasible, prioritize procedures and place temporary restorations until the patient is stable. Prioritizing procedures: 1. infections, extractions, scaling, and sources of tissue irritation 2. carious teeth, root canal therapy and replacement of faulty restorations 3. the risk of pulpal infection and pain determines which carious lesions should be treated first
B. AFTER TRANSPLANTATION	
Preventive strategies	As above; patients may become high caries risk after the transplant
Dental procedures	Defer all elective procedures during immunosuppression periods Consult physician in cases of dental emergencies Recall every 3 – 6 months depending on the patient's caries risk Discuss need for antibiotic prophylaxis and supplemental steroids with physician Odontogenic and other oral infections should be treated aggressively during immunosuppression
Close monitoring of soft tissues	Increased risk of oral malignancy, probably related to immunosuppression Watch for secondary infections: - cultures and biopsies when appropriate - Nystatin prophylaxis is ineffective
Orthodontics	May start or resume after at least a 2-year disease-free survival Consider risk of gingival overgrowth and patient's oral hygiene status
Gingival hypertrophy	Assess need for gingivectomy, consider patient's

compliance and oral hygiene status

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Chapter 24: SPECIAL PATIENTS

AAPD GUIDELINE:

http://www.aapd.org/media/Policies_Guidelines/G_SHCN.pdf

- I. AUTISM AND AUTISM SPECTRUM DISORDER
- II. ATTENTION DEFICIT HYPERACTIVITY DISORDER
- III. MENTAL RETARDATION
- IV. SEIZURE DISORDER
- V. MITACHONDRIAL DISORDERS
- VI. NEURAL TUBE DEFECTS
- VII. HYDROCEPHALUS
- VIII. CEREBRAL PALSY
- IX. MUSCULAR DYSTROPHY
- X. DEAFNESS
- XI. ADDITIONAL READINGS

I. AUTISM AND AUTISM SPECTRUM DISORDER

Incidence

- 1 in 166 births
- 4 to 1 boys over girls

Definition—Autism is a spectrum disorder where children undergo abnormal brain development from early infancy. Children start out with slightly smaller heads than average, then undergo explosive brain growth with the more severe cases having more severe brain growth. Brain may be 10-15% larger than the norm in all areas.

Spectrum

- Autism
 1. severe language problems
 2. lack of interest in others
 3. repetitive behaviors
 4. resistance to change
 5. irrational routines
- Asperger
 1. relatively strong verbal skills
 2. trouble with social situations and sharing enjoyment
 3. obsessive interests
- PDD (Pervasive Developmental Disorders): “atypical autism” kids have less severe social impairments
- CDD (Childhood Disintegrative Disorder)
 1. normal growth and development for 2-4 years
 2. autism-like symptoms develop
- Rett Disorder: similar pattern to CDD but occurs earlier and mostly in girls

Early Signs

- does not respond to name
- acts as though were deaf
- does not smile socially
- does not point or gesture by age one
- no babbling
- talks but lacks social and communication skills

Medications

Dental Considerations

- no specific dental anomalies
- dental conditions related to behavioral patterns
- normal morphology
- occlusal attrition due to persistent bruxism
- diet modifications which may be cariogenic such as behavior modification programs and taste
- sensitivities
- may develop seizures
- some have insensitivity to pain
- oral hygiene may be poor due to child's inability to brush and accept assistance in doing so

Medications for Autism

Name	Indications	Adverse Orofacial Rxn	Systemic Side Effects
Carbamazepine <i>Tegretol</i>	Mood stabilization Antiaggression Anticonvulsant	Xerostomia Stomatitis/Glossitis Carbohydrate cravings	↓ wbc and ↑ platelets with long term use Erythromycin inhibits metabolism
Clonidine <i>Cataprus</i>	Calm hyperactivity Reduce impulsivity	Xerostomia Dysphagia, sialadenitis	Potentiates CNS depressants ♥ arrhythmias
Fluoxetine <i>Prozac</i>	Antidepressant ↓ Repetitive thoughts ↓ Compulsive behaviors Antianxiety Prevent self-mutilation	Xerostomia, Altered taste, Bruxism, Sialadenitis Stomatitis/Glossitis, Gingivitis,Jaw pain Discolored tongue	Diarrhea, Nausea, Somnolence, Dizziness ↑ bleeding time Potentiates CNS depressants Erythromycin inhibits metabolism
Methylphenidate <i>Ritalin Concerta</i>	Calm hyperactivity Enhance attention	Xerostomia	Tachycardia Nervousness Anorexia Insomnia Potentiates ♥ arrhythmogenic effects of TCAs
Olanzapine <i>Zyprexa</i>	Antipsychotic ↓ Delusions ↓ Hallucinations	Xerostomia Dysphagia Stomatitis Glossitis/Gingivitis Tongue edema Facial edema	Potentiates CNS depressants
Risperidone <i>Risperdal</i>	Antipsychotic Antiaggression ↓ Agitation ↓ Hallucinations ↓ Delusions	Xerostomia, Dysphagia, Altered taste, Stomatitis Discolored tongue, Gingivitis, Tongue edema Facial edema	Potentiates CNS depressants Thrombo-cytopenia (rare)

Used with permission from Dr. Barbara Sheller-Children's Hospital, Seattle, Wash.

Dental Treatment

- desensitization (work from least anxiety-producing to most—could take several visits)
- positive reinforcement
- physical restraint (“protective aids”) if used
- couple with desensitization and get informed consent
- sedation with the understanding that psychotropic agents could have unusual side effects on autistic patients
- general anesthesia when all else fails

Specifics in Dental Treatment

- keep sentences short and simple
- music as an aid
- use parent/caregiver as helper in communication
- when demonstrating toothbrushing techniques and use of equipment move patients limbs rather than demonstrating yourself
- keep environment familiar (most familiar procedure first)
- same people present
- use of same treatment room
- ask the parent/therapist to rehearse procedure at home/school prior to office visit
- keep visits short and repetitive with particular attention to sound distractions
- try to end visit on a positive note by ending with procedure that the patient has been able to cooperate with and then you can start with that procedure at the next visit

II. ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

- Most common neurobehavioral disorder in children
- Symptoms typically present prior to age 7 years
- Prevalence in general population: 9.2% of all males/2.9% of all females

Core Symptoms

- Inattention
- Hyperactivity
- Impulsivity

Subtypes

- Inattentive type
- Hyperactive type
- Combined type – Hyperactive/Inattentive

Child may display functional problems

- School difficulties
- Difficult interpersonal relationships-does not interact well with peers
- Low self-esteem
- Aggression

Commonly occurs with other disorders

- Oppositional defiant disorder
- Conduct disorder
- Depression
- Anxiety disorder
- Speech/language delay
- Learning disabilities

Diagnosis

- Requires that the child meet specific criteria as outlined by Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)
- Requires that the child meet behavioral symptoms of ADHD and that the child demonstrate functional impairment
- Symptoms should be present in two or more settings (e.g. reported by parent and classroom teacher)

Treatment

- Consider ADHD as a chronic condition
- Specific target outcomes should be used to guide management
- Patient may have “holidays” from drug therapy
- Requires coordination between child, family, school, and physician, with a long-term plan developed
- Stimulant medications (Methylphenidate, Dextroamphetamine)

Side effects

- Tachycardia
- Xerostomia
- Bruxism
- Decreased appetite
- Stomach ache
- Headache
- Delayed sleep onset
- Jitteriness
- Social withdrawal
- Motor tics

Symptoms of excessive dose

- Child appears dull or overly restricted

- Behavioral therapy

Home-parent training regarding structure, behavior guidance

- Increase positive behavior
- Decrease negative behavior

Classroom –provide greater structure

- Periodic systematic follow-up by physician with parent and school to determine if target outcomes are being met

Dental Considerations

- Discussion with parent/guardian and physician regarding patient's treatment for ADHD
- Careful discussion with parent/guardian regarding the dental treatment and the behavior guidance approach that will be utilized
- Review patient medications (including OTC medications) for drug interactions
- Avoid treatment, if possible, on days when patient has not taken medications
- Use positive reinforcement
- Patient may display wear of dentition from bruxism
- Stimulant medications may cause xerostomia, leading to increased risk for caries

III. MENTAL RETARDATION

Definition—a disability characterized by significant limitations both in intellectual functioning and in adaptive behavior as expressed in conceptual, social, and practical adaptive skills

Assumptions

- Limitations in present functioning must be considered with the context of community environments typical of the individual's age peers and culture
- Valid assessment considers cultural and linguistic diversity as well as differences in communication, sensory, motor, and behavioral factors
- Within an individual, limitations often coexist with strengths
- An important purpose of describing limitations is to develop a profile of needed supports
- With appropriate personalized supports over a sustained period, the life functioning of the person with mental retardation generally will improve

Etiology—manifestation of a group of disorders of CNS function

- primary causes may be considered to be an organic problem with brain metabolism or nervous system function
- cultural deprivation or from varying combinations.
 1. Organic Causes—prenatal, chromosomal, maternal age, radiation
 2. Genetic Metabolic Disorders—Hurler, Tay-Sachs, G-6-PD, Phenylketonuria, Mitochondrial
 3. Genetic Neurologic Disorders—Hydrocephaly, Spina Bifida
 4. Endocrine Disorder
 5. Perinatal Complications—prematurity, prenatal anoxia
 6. Postnatal—traumatic brain injury, encephalitis, meningitis

Signs and Symptoms—mental retardation is not a disease entity in itself but rather a symptom of a central nervous system disorder. It rarely occurs as the only symptom and can be associated with physical and psychological abnormalities such as epilepsy, cerebral palsy, orofacial deformities and emotional problems.

- Examples
 1. congenital heart disease requiring prophylactic antibiotics
 2. seizures
 3. cerebral palsy
 4. abnormal head shape
 5. other neuromuscular disorders

Classifications—The Stanford-Binet IQ Test—no longer legally used as a determiner of intelligence provided rough equivalents for the categories of mental retardation which are still in use:

- Borderline MR (IQ 68-73)
- Mild MR (IQ 52-67)
- Moderate MR (IQ=36-51)
- Severe MR (IQ=21-35)
- Profound MR (IQ=<21)
- 90% are borderline and mild

Major Oral Findings

- no difference versus normal population (except Down Syndrome)
- higher incidence of periodontal disease
- altered eruption and malocclusion
- anomalies in tooth morphology
- drooling
- macroglossia
- clenching and bruxism
- high vaulted palate

Dental Considerations

- Wide variation of behavior and level of understanding between mild and profound mental retardation
- Degree of management difficulty proportional to the level of cognitive functioning
- Informed consent
- Medical history
- Accurate and ever changing treatment plan
- Good communication—slow, simple and repetitive
- Pay attention to what the patient is trying to tell you (parent or caregiver may be able to interpret for you and of help in this phase)
- Behavior modification similar to other patients who are not mentally challenged
- Oral and IV sedation
- Physical restraints, general anaesthesia
- Strict preventive dentistry regime with patient, parents, and caregiver

IV. SEIZURE DISORDERS

- Spontaneous uncontrollable excessive discharge of cerebral neurons
- Result in suspension of motor, sensory, behavioral, or body functions
- Epilepsy- recurrent seizures-name given to seizure disorders for which no cause can be found (not a unique disease in and of itself)
- Hyperactivity and its location determines the type of seizure
- Seizures starting before age 2 are usually caused by high fevers
- Many seizures that begin between the ages of 2 and 14 years have unknown etiology
- 4% of all children have at least one seizure by 15 years of age; recurrent seizures affect 0.5% of children

Classification

- Partial-focal or local (40%)
 1. Common site of origin is within the frontal or temporal lobes
 2. Patient may experience abnormalities of taste or smell (aura)
 3. Seizure activity may have a variety of presentations (e.g.lipsmacking, picking at one's clothes, abnormal behavior, localized numbness or tingling, inappropriate

- words, hallucinations, etc.)
- 4. Amnesia, confusion, and sleepiness accompany the seizure episode
- Generalized-convulsive or nonconvulsive (40%)
 - 1. Most common form is the tonic-clonic seizure (Grand Mal)
 - 2. Involves all extremities
 - 3. Consciousness is lost abruptly
 - 4. Aura or prodromal mood change
 - 5. Post-ictal phase is characterized by atonia and incontinence
 - 6. Patient regains consciousness, is confused and then usually falls into a deep sleep
 - 7. Other forms include absence (Petit Mal), myoclonic, atonic, clonic, tonic seizures
- Absence seizures
 - Generalized but not convulsive
 - Brief eye or muscle fluttering
 - Abrupt onset (no aura) and lasts a few seconds
 - Seizure is characterized by suppression of mental function and resumes after attack
 - Onset: 4-10 years
 - Sometimes misdiagnosed as behavior or learning problem
- Unclassified (20%): Includes all seizures that cannot be classified

Seizure History for Dental Patients

- Type
- Frequency-date of last seizure
- Duration
- Triggers
- Medications and compliance
- Control-date of last hospitalization for seizure
- Diet

Medical Management of Seizure Disorders

- Anti-convulsant medications
- Ketogenic diet
- Surgery
- Complementary/alternative medicine

Oral Evaluation and Management

- Medications (many seizure medications have side effects and drug interactions of clinical relevance to dentistry-*ie* gingival hyperplasia, gingival bleeding, xerostomia)
- Determine degree of seizure control—neurology consult
- Consider general anesthesia for patients with poor seizure control
- Schedule when well rested
- Consider anxiety management
- Xylocaine decreases seizure threshold
- Be prepared to manage seizure
- Aggressive oral hygiene program

- Prosthetics may be problem for poorly controlled patient
- May be prone to anterior tooth trauma

Management of a Seizure

- Note time seizure begins
- Stay calm
- Move onlookers away
- Position patient to prevent injury
 1. Chair supine and fully down
 2. Nothing in mouth
- Post-ictal airway support
 1. Rolled towel under shoulders
 2. Suction airway
 3. Vital signs
 4. Supplemental oxygen prn
- Activate EMS in cases of continued seizures (Status Epilepticus)

V. MITOCHONDRIAL DISORDERS

- Any illness resulting from deficiency of any mitochondria-located protein
- Results from failure in mitochondrial function, leading to an energy-deficient state
- Should be considered when the following features are noted (especially in combination)
 1. Encephalopathy
 2. Neuropathy
 3. Cardiac conduction defects/Cardiomyopathy
 4. Hearing deficits
 5. Short stature
 6. Disorders of extraocular muscles
 7. Diabetes
 8. Renal tubular disease
 9. Visual Loss
 10. Lactic Acidosis
 11. Susceptibility to Malignant Hyperthermia

Mitochondrial Myopathy, Encephalopathy, Lactic Acidosis and Stroke-like episodes (MELAS)

- Classic mitochondrial encephalopathy
- Variable clinical presentation
- Majority of patients develop symptoms before age 20 years
- U.S. population frequency: 16.3 per 100,000

Genetics

- Autosomal recessive inheritance – most common in mitochondrial disorders
- Maternal inheritance, autosomal dominant, and x-linked recessive are less frequent modes of inheritance

Testing

- Several levels of testing
- Blood, Urine, Cerebrospinal Fluid, Muscle Biopsy

- Testing for inborn errors of energy metabolism, mitochondrial function

Treatment

- No known cure
- Goals of treatment are to alleviate symptoms and slow disease progression
- Supplementation with Vitamins, Co-Factors (CoQ10) to increase ATP production

Clinical Manifestations

- Variable, depending on the organ system involved
- Seizure disorder
- Developmental delay
- Movement disorders
- Complicated migraine
- Stroke

Common oral findings

- Acid erosion due to increased vomiting

Dental Management

- Consultation with physician regarding patient's specific form of disorder
- Early institution of preventive dental care
 1. Parental education regarding oral health care – hygiene, diet
 2. Increased frequency of preventive visits
 3. Appropriate use of fluoride therapy
- Avoidance of physiological stress (heat, cold, lack of sleep, lack of food, etc.)
- For General Anesthesia cases, Malignant Hyperthermia precautions should be used

VI. NEURAL TUBE DEFECTS

- Result from a failure in the neural tube development
- Type of defect depends on the site where neural tube failed to develop properly
- Typically occurs between 3-4 weeks in-utero, often before mother knows she is pregnant

Causes

- Folic acid deficiency – most common
- Maternal insulin-dependent diabetes
- Maternal medications for epilepsy
- Maternal obesity

Types

- Anencephaly
Cephalic end of neural tube fails to close
Results in absence of forebrain and cerebrum
Patient dies within days of birth, if not stillborn
- Encephalocele
Failure of neural tube to close completely
Results in groove down midline of upper part of skull

Accompanied by other craniofacial abnormalities

Clinical symptoms

- Hydrocephalus
- Spastic quadriplegia
- Microcephaly
- Ataxia

- Spina Bifida

Prevalence: 7/10,000 live births in U.S.

Three types

- Spina Bifida Occulta – most common type, most mild form
 - Outer part of some vertebrae are not completely formed
 - Spinal cord is intact
 - No long term effects
- Meningocele – least common type
 - Meninges is pushed out through cleft in vertebrae
 - Meningeal sac is usually covered by skin
 - Nerve damage is atypical
 - Few if any long-term effects
- Myelomeningocele – most severe form
 - Meninges protrudes as a sac through a cleft in vertebrae
 - Sac contains cerebrospinal fluid and nerve tissue
 - Clinical symptoms
 - Paralysis
 - Incontinence – Bladder and Bowel

Hydrocephalus – occurs in nearly 90% of patients

- Arnold-Chiari II malformation

Portion of cerebellum protudes through foramen magnum into spinal canal
Patients may only present with symptoms in adolescence or adulthood

Symptoms

- Headache in back of head
- Dizziness/double vision
- Feeling of discomfort and/or choking on reclining

Treatment - surgery to enlarge foramen magnum relieving pressure of cerebellum

- Treatment of Neural Tube Defects

Surgery to close spinal opening

- Done during infancy
- Does not correct any nerve damage

Placement of shunt for patients with hydrocephalus

- See section on Hydrocephalus
- Depending on type of shunt, SBE prophylaxis may be indicated

Dental Considerations

- Consultation with patient's physician regarding nature of defect and past medical history
- Realization that patient has had many medical interventions and may have heightened anxiety
- Some patients with neural tube defects have mental disabilities
- In adolescence, some spina bifida patients develop depression
- Latex allergy precautions
- Access for wheelchair
- Antibiotic prophylaxis for patients with VA or VV shunt for hydrocephalus
- For patients with Arnold-Chiari malformation, consider treatment with patient in a more upright position

VII. HYDROCEPHALUS

- Abnormal accumulation of cerebrospinal fluid (csf) in ventricles of brain
- Results from an imbalance in production and absorption of csf
- Incidence: 1 in 500 children
- Types
 1. Congenital – present from birth
 2. Acquired – from injury or disease
- Causes
 1. Genetic inheritance (aqueductal stenosis)
 2. Developmental disorders (neural tube defects such as spina bifida)
 3. Head trauma
 4. Meningitis
 5. Tumors
- Symptoms
 1. Rapid increase in head circumference (in infants)
 2. Vomiting
 3. Seizures
 4. Downward casting of eyes (“sunsetting”)
 5. Sleepiness
 6. Irritability
 7. Blurred vision
 8. Inability to balance
- Treatment

Surgical placement of a shunt to drain cerebrospinal fluid from brain

 - Ventriculoperitoneal (V-P) Shunt – from ventricle of brain to peritoneum
 - Ventriculoatrial (V-A) Shunt – from ventricle of brain to atrium of heart
 - Complications of Shunt – may require revision of shunt

Infection
Mechanical failure
Obstructions

Dental Considerations

- Consultation with physician regarding patient's condition (type of hydrocephalus, type of shunt, need for SBE prophylaxis)
- Recognition that patient has had many medical interventions and may be apprehensive about dental treatment
- Strong emphasis on prevention to minimize the need for restorative procedures

VIII. CEREBRAL PALSY

- Nonprogressive disorder resulting from malfunction of the motor centers and pathways of the brain
- Characterized by paralysis, weakness, incoordination or other aberrations of motor function
- Most commonly occurs during prenatal or perinatal period
- 1.5-3 cases/1000 children

- No cure, but many patients enjoy near-normal lives if their neurological problems are properly managed

Classification

- Spastic-tightness, stiff or rigid muscles, contractures and lack of control
- Dyskinetic (athetoid) –slow, writhing, involuntary movements, hypotonia
- Ataxic –tremors or uncoordinated voluntary movements
- Mixed-combination of all types

Medications

- Antiparkinsonian drugs (eg levodopa)
- Antispasticity agents (eg, baclofen)
- Anticonvulsants (including benzodiazepines such as diazepam, valproic acid, barbiturates)
- Antidopaminergic drugs
- Antidepressants
- Botox

Clinical Manifestations

- Mental retardation 60%
- Seizure disorders 30-50%
- Sensory Deficits 35%
- Speech disorders
- Joint contractures

Common dental/oral findings

- Periodontal disease
- Dental caries
- Malocclusions
- Bruxism
- ↑ erosion
- Trauma and injury
- Hyperactive bite reflex
- ↑ gag reflex
- Dysphagia
- ↑ drooling
- Mouth breathing

Dental Considerations

- Make the treatment environment calm and supportive
- Try to help your patient relax to reduce muscle movement
- Place and maintain your patient in the center of the dental chair
- Do not force limbs into unnatural positions
- Consider treating in wheel chair
- Stabilize patient's head during treatment
- Consider supports for limbs
- Use mouth props or finger splints
- Keep patient's back slightly elevated to minimize swallowing difficulties
- Forewarn patients of stimuli to minimize startle reactions
- Consider use of rubber dam for restorative procedures
- Minimize time in dental chair and take frequent breaks
- Often require premedication or GA for extensive dental work

IX. MUSCULAR DYSTROPHIES (MD)

- A group of familial disorders in which degeneration of muscle fibers occurs
- Various forms of human muscular dystrophy result from mutations in genes encoding proteins of the nuclear envelope
- No cure or effective treatment for MD

Classification

- 9 types based upon age of presentation, mode of inheritance, rate of pathogenesis, and distribution of muscles involved
- Pseudohypertrophic MD (Duchenne)
 1. Most common neuromuscular disease of childhood
 2. X-linked recessive primarily affects boys
 3. Prevalence rates ranging from 19 to 95 per million
 4. Onset: early childhood - about 2 to 6 years
 5. Ambulation usually not possible by 12 years of age
 6. Universally fatal, with death usually occurring from respiratory or cardiac complications prior to 30 years
 7. 25% demonstrate mental retardation
- Other types: Becker Muscular Dystrophy, Emery-Dreifuss Muscular Dystrophy, Limb-Girdle Muscular Dystrophy, Facioscapulohumeral Muscular Dystrophy, Myotonic Dystrophy, Oculopharyngeal Muscular Dystrophy, Distal Muscular Dystrophy

Common dental/oral findings

- Plaque/gingivitis
- Poor oral control
- Trauma and injury

Dental Considerations

- Supports (e.g., mouth props) help with muscle weakness during treatment
- May need transfer to dental chair and postural support
- Sedation and/or general anesthesia may be necessary to manage care with poor level of cooperative behavior
- May have deficits in protective airway reflexes and poor control of fluids and debris in airway
- Should not place patient in a supine position
- May require short appointments
- Rubber dam may be useful if the patient can breathe through the nose
- Treatment approach is dependent on severity and type of MD

X. DEAFNESS

Etiology

- Hereditary
- Genetics—GJB2
- Trauma
- Syndromic
- Accoustical neoplasm
- Viral
- Mitochondrial cytopathologies

Note—American Sign Language (ASL) is NOT English in sign language BUT it is a language unto itself as is French or Italian or any other language for the people it serves. In essence it is the language of deaf people.

Dental Consideration

- Maintain eye contact when using ASL
- Speak slowly but do not change your lip movements so that deaf person can read your lips
- Use head movement for communication
- Use eye movement for communication
- Other senses take over for those lost by hearing impairment
- Intelligence is normal to superior
- Hypernasality is a function of deafness
- Oral conditions are not effected in an adverse way
- Basic American Sign Language could be of assistance

XI. ADDITIONAL READINGS AND WEB SITES

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www.aap.org/policy
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<http://www.aamr.org/>
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Chapter 25: NEW MORBIDITIES

AAPD POLICIES AND GUIDELINES:

http://www.aapd.org/media/Policies_Guidelines/RS_BMICcharts.pdf

http://www.aapd.org/media/Policies_Guidelines/RS_FoodPyramid.pdf

www.aapd.org/media/Policies_Guidelines/G_childabuse.pdf

www.aapd.org/media/Policies_Guidelines/D_DentalNeglect.pdf

http://www.aapd.org/media/Policies_Guidelines/P_ChildIDPrograms.pdf

http://www.aapd.org/media/Policies_Guidelines/P_TobaccoUse.pdf

I. PREGNANCY (T)

II. OBESITY

III. ABUSED, NEGLECTED, MISSING AND EXPLOITED CHILDREN (T)

IV. SUBSTANCE ABUSE

V. BRIEF SUMMARY OF DRUGS

VI. TOBACCO USE AMONG YOUTH

VII. ADDITIONAL READINGS AND WEB SITES

I. PREGNANCY

Statistics and terminology

- Approximately 7 million pregnancies per year in the U.S. with over 4.1 million live births
- 5% to 8% of the teenage population
- Mean duration of pregnancy: 280 days (40 weeks)
- “Term”: period from 36 completed (37.0) to 42.0 weeks of gestation
- “Preterm or premature” labor: Onset of labor prior to 36 completed weeks’ gestation
- “Postterm” (prolonged): pregnancies continuing beyond 42.0 weeks’ gestation
- Low birth weight (LBW): Infants who weigh less than 2,500 g at birth, regardless of gestational age
- Sporadic pregnancy loss occurs in 10 to 15% of all clinically recognized pregnancies in the first trimester

Diagnosis of pregnancy

- Clinical history and lack of menses, Positive human beta-chorionic gonadotropin test, Detection of fetal heartbeat by Doppler (9-12 weeks)

Pregnancy Related Morbidities

- Gestational diabetes, hypertension, preeclampsia: syndrome defined by hypertension and proteinuria during pregnancy, eclampsia: the new onset of grand mal seizures in a woman with preeclampsia, hyperthyroidism, hypothyroidism, depression, premature birth (periodontal disease maybe a risk factor)

Common oral findings include pregnancy gingivitis, periodontal disease, pregnancy granuloma, dental erosion and sensitivity, dental caries and increased tooth mobility.

Oral findings associated with preterm birth: enamel hypoplasia, delayed dental eruption, reduced dental dimensions, dental caries, tooth discoloration associated with high bilirubin and palatal groove associated with intubation

Role of Oral Health Professional

- Pregnancy by itself is not a reason to postpone routine and necessary dental treatment
 1. Self-medication with over the counter medications to control pain may cause unforeseen harm to the woman and possibly to the fetus
 2. Untreated oral infection may become a systemic problem during pregnancy and may contribute to preterm and/or low birth weight deliveries
 3. Untreated cavities in mothers may increase the risk of caries in children
- First trimester comprehensive exam for diagnosis and treatment planning
 1. Needed dental x-rays can be undertaken safely to diagnose disease processes that need immediate treatment
- While routine dental x-rays are recommended, x-rays such as full mouth series, panorex and cephalograms may be postponed
- Emergency and needed treatment can be provided anytime during pregnancy; however, the time period between the 14th and 20th week is ideal time for treatment

Dental Considerations

- Delayed gastric emptying and incompetent lower esophageal sphincter; hence increased risk for aspiration
- Maintain a semi-seated position
- Treatment may be limited due to morning sickness during the first trimester
- Treatment may be impeded due to increased physical discomfort during the third trimester
- Be aware of complications which may arise during dental procedures: including syncope, enhanced gag reflex, supine hypotensive syndrome, seizures and gestational hyperglycemia
- Assess caries risk (poor OH due to morning sickness)
- Provide comprehensive gingival and periodontal examination
- Decrease maternal cariogenic bacterial load: fluoride toothpaste along with fluoride mouth rinses depending on the fluoridation status of water, restore untreated caries, chlorhexidine mouth rinses and fluoride varnish as appropriate, recommend the use of xylitol-containing chewing gum
- Complete restorations with permanent materials, if possible, during pregnancy and prior to delivery
- Provide nutritional counseling, preventive and treatment care
- Provide infant oral health anticipatory guidance
- Counsel patients concerning the harmful effects of tobacco, alcohol and recreational drugs
- Consult with the prenatal care provider when considering postponing treatment due to pregnancy, co-morbid conditions (diabetes, hypertension or heparin treated thrombophilia) exist, or when considering intravenous sedation, nitrous oxide or general anesthesia to complete the dental procedure

TABLE 1. ACCEPTABILITY OF DRUGS FOR PREGNANT WOMEN

	These drugs may be used during pregnancy	FDA Category	These drugs should NOT be used during pregnancy	FDA Category
ANTIBIOTICS	Penicillin Amoxicillin Cephalosporins Clindamycin Erythromycin (except for estolate form)	B B B B B	Tetracyclines Erythromycin in the estolate form Quinolones Clarithromycin	D B C C

ANALGESICS	Acetaminophen Acetaminophen with codeine Codeine Hydrocodone Meperidine Morphine <u>After 1st trimester and for 24-72 hrs only</u> Ibuprofen Naprosyn	B C C B B	Aspirin	C
LOCAL ANESTHESIC	2% lidocaine with 1:100,000 epinephrine	B		

II. OBESITY AND EATING DISORDERS

- In recent decades childhood overweight and obesity have reached epidemic proportions worldwide
- Obesity defined as: Body Mass Index (BMI)-for-age/gender greater than 95th percentile
- Overweight defined as BMI-for-age and gender-greater than 85th percentile
- Etiological factors:
Energy (calorie) intake (portion sizes, dietary fat/sugar)-increased
Physical activities (outdoor play, school P.E.)-reduced
Sedentary activities (TV, computer/video games)-increased
- Endocrine and metabolic factors are usually a consequence/co-morbidity rather than the cause of obesity
- Apparent correlation between increased weight and socioeconomic class such that children on the lower end of the scale tend to be overweight/obese more often than those on the upper end
- Familial (genetics and environment) factors play a role in obesity-obese parents tend to have obese children
- Obese children are more likely to become obese adults

Dietary issues:

- Primary oral/caries-related dietary concern is the type of food consumed, the frequency in which it is eaten and the level of oral hygiene
- Increased consumption of sugar-sweetened beverages and snack foods also related to obesity

Health-related concerns:

- Type 2 diabetes, periodontal disease, hypertension, cardiovascular disease, psychological stress, respiratory, orthopedic and hepatic problems
- Health risks associated with childhood overweight and obesity are strong indicators for predisposition to adult morbidity and mortality
- Obese are at increased risk for sedation/anesthesia complications

EATING DISORDERS

- Definition of ED: any of several disorders characterized by persistent disturbance of eating behavior (hypophagia, purgative hyperphagia) intended to control weight that significantly impair physical and mental health
- Over 10 million Americans are estimated to suffer from EDs
- EDs are classified as psychiatric disorders
- EDs primarily affect and originate in adolescent and young adult females—the prevalence is ~ 90% female
- EDs have the highest mortality rate of any mental illness
- EDs can have direct effects on the oral cavity
- A differential diagnosis for EDs would include many physical disorders that include hypophagia (HIV, cancer) and hyperphagia (Prader-Willi syndrome, hypothalamic tumors) AND depression and anxiety

ATTITUDES, BEHAVIORS AND CHANGE

- Weight and dieting are influenced by cultural, familial, personal and biological factors
- Pressures to remain thin may be very difficult to handle for some adolescents
- Normally children progress from ~ 8% composition of body fat to ~ 22% by the end of puberty
- These changes may lead to a dissatisfaction with body image, a fear of becoming fat and a desire to lose weight

ANOREXIA NERVOSA (AN)

- AN is characterized by thin cachectic appearance with low body weight with a BMI-for-age less than the 5th percentile
- AN individuals refuse to maintain their body weight and develop a preoccupation with food and have a distorted perception of their body image and an intense fear of gaining weight, claiming they feel fat despite the fact that they are obviously underweight
- AN often is observed in individuals pursuing artistic or athletic endeavors that emphasize weight limits (eg, ballerinas and wrestlers)
- AN individuals often are perfectionists and vehemently deny a problem even when confronted about the illness
- Most women with AN become amenorrheic
- AN includes two subtypes: up to 50% of anorexic patients engage in binge eating followed by vomiting (binge/purge type)
The other subtype do not do not binge/purge (restricting type) but rather pursue laxatives and/or diuretics
- AN signs and symptoms include hirsutism (excessive hair growth to conserve body heat), fine, brittle facial and trunk hair, brittle nails, hypothermia/ dry cool skin, constipation, insomnia, electrolyte imbalance, cardiac impairment (arrhythmias, bradycardia, mitral valve prolapse), hypotension, osteoporosis and muscular weakness
- Many patients with AN brush their teeth fastidiously and vigorously to remove any trace of caloric intake
This may cause abrasion of tooth enamel and cervical cementum, and gingival recession

- AN patients who engage in binge behavior may present with the lingual surfaces of their teeth eroded by gastric acids that decalcify the teeth because of the low pH in the range of pH 1.0-5.0
- The teeth may exhibit sharp incisal edges of eroded teeth
- Parotid gland swelling may be present and the individuals may have swallowing difficulty and redness/soreness of throat and palate
- Other signs may include the presence of abrasions and calluses on the dorsal surface of the fingers and hands related to self-induced vomiting

BULIMIA NERVOSA (BN)

- BN involves recurrent episodes of binge eating followed by self-induced vomiting, chronic use of laxatives or diuretics and/or excessive exercise
- Between eating episodes the BN patient is dieting or fasting
- BN individuals often rapidly consume/binge on average 3,400 calories in a little over an hour, eating such foods as cake, bread, pasta and ice cream
- BN individuals have a feeling of being out of control while they are binging
- BN individuals are concerned with body weight/shape and preoccupied with food
- Bulimic patients tend to be within the normal range of weight, aware that their eating pattern is abnormal and often they are depressed
- Binge eating and purging are done in secret, usually starting in adolescence (age 13-16)
- Systemic problems include dehydration, electrolyte and acid base imbalance, trauma to the esophagus/ stomach linings, and menstrual irregularity
- Dentofacial findings vary; with more frequent the episodes of vomiting, findings are more severe
- BM patients may present with the lingual surfaces of their teeth eroded as a result of the gastric acids
- Erosion is generally observed after a period of two years or approximately 2,000 episodes of vomiting
- Thermal sensitivity and margins of restorations appearing higher than the tooth surface are related to the erosion of enamel
- Parotid swelling may be cosmetically displeasing but gland is painless, soft and salivary flow appears to be normal
- Callus formation and soft palate injury caused by trauma while the patient is trying to induce vomiting
- Definitive dental treatment, such as fixed prosthetics, should be undertaken only after the eating disorder is brought under control
- Diet counseling, fluoride supplements and frequent follow-ups should be provided
- As a component of appropriate interdisciplinary team-based treatment, patients who vomit should:
 1. be told to rinse their mouth with water and sodium bicarbonate after regurgitation in attempts to neutralize gastric acids
 2. receive daily fluoride applications with custom tray (0.4% SnF or 1.1% NaF) to help prevent or reduce enamel decalcification

Eating Disorders Not Otherwise Specified (EDNOS)

- EDNOSs include conditions that meet the definition of an eating disorder but not all of the criteria for AN or BN
- EDNOS individuals may exhibit many characteristics of AN/BN including enamel erosion and electrolyte imbalance
- EDNOS includes binge eating disorder (BED) characterized by recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors such as (purgng, fasting, excessive exercise) usually characteristic of binge-purge AN or BN
- EDNOS individuals may be obese
- The prevalence is only slightly higher in females

III. ABUSED, NEGLECTED, MISSING AND EXPLOITED CHILDREN

Definition

Can include physical abuse, sexual abuse, emotional abuse, failure-to-thrive, intentional drugging/poisoning, Munchausen by proxy (faked medical illness), medical care neglect, dental neglect, safety neglect, physical neglect

- Abuse is physical or mental injury, sexual abuse, or negligent treatment of a child under 18 by a person responsible for the child's welfare under circumstances which might indicate that the child's health or welfare is harmed or threatened thereby (Federal model definition)
- Sexual Abuse is when a child is engages in a sexual situation with an adult or an older child. Sometimes this means direct sexual contact, such as intercourse, other genital contact or touching. But it can also mean that the child is made to watch sexual acts, look at an adult's genitals, look at pornography or be part of the production of pornography. Children many times are not forced into the sexual situation, but rather they are persuaded, bribed, tricked or coerced.
- Emotional/Psychological Abuse is when a child is regularly threatened, yelled at, humiliated, ignored, blamed or otherwise emotionally mistreated. For example, making fun of a child, calling a child names, and always finding fault are forms of emotional/psychological abuse
- Neglect is when a child's basic needs are not met. These needs include nutritious food, adequate shelter, clothing, cleanliness, emotional support, love and affection, education, safety, and medical and dental care.
- Dental neglect can include dental caries, periodontal diseases, and other oral conditions if left untreated can lead to pain, infection, and loss of function. These undesirable outcomes can adversely affect learning, communication, nutrition, and other activities necessary for normal growth and development. Dental neglect is willful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection

Epidemiology

- Annual incidence reported cases (est.) 3,000,000
- 1000-2000 die annually in U.S.
- Non-reported-to reported case is 100/1

- All SES involved
- 50% of children under 7 years old
- 30-65% of abusive injuries occur to the head and neck area

Causation

- family stress (economic problems, divorce, lack of support)
- single parent household
- learned behavior
- alcohol/substance abuse
- mental illness
- special needs child

Prognosis

Guarded with timely intervention

Complications

Further injury or death without intervention

Diagnosis

GENERAL CHARACTERISTICS OF ABUSED AND ABUSER

Abuser Profile

40-60% were abused as children
isolated, young, single parents
mothers more than fathers
unstable marriage
unwanted parenthood
negative/distorted self-image
substance abuse
seldom touches or looks at child
over-critical/negative of child
younger not older (sexual)
family member/friend (sexual)
not a stranger (sexual)

Abused Profile

more in special needs
hyperactive, cognitively dysfunctional
25% premature
history of prolonged family separation
behavioral extremes
shy around adults
fearful of parent
inappropriate sexual behavior
sexual remarks
preoccupation with body (sexual)
poor overall care/appearance
overreacts to parent's responses

EVENT-RELATED CHARACTERISTICS

inappropriate response to injury
delayed care seeking for injury
refuses consent for long-term care
blames other parent
implausible explanation injury
embarrassment at injury

clothes to cover injury
bruises in various healing stages
30-65% head and neck
implausible injury
tries to protect parent
bruises to protected (padded) areas
burns, bites, bruises
bruised areas not at risk

Exam, History and Documentation

- examine child without parent/with staff, ask about injury in child's terms, be sincere/comforting, open-ended questions, non-judgmental questions
- solicit history of injury from parent(s); may contact physician to help confirm

- describe injury by type, color, stage, size, characteristics; radiograph; photograph with color scale/various views; write description/use illustration of location

Differential Diagnosis

- Non-accidental trauma
- Dermatological lesions (Mongolian spots, impetigo, erythema multiforme)
- Bleeding diatheses
- Osteogenesis imperfecta
- Self-inflicted injuries
- Folk medicine (cupping, “cao gio” or coin rolling)

Medical Treatment

- all states have laws; dentists mandated reporters; most provide immunity and require report upon suspicion of abuse
- must report if suspicious
- laws aim to protect children; help families; functional level is county social service agency
- reports can always be made through local police departments
- reporting/management mechanism varies state to state
- contact agency with suspicion
- dentists may be asked to appear in court and/or file report

Dental/Oral Findings

Contusions, lacerations, scarring, bite marks, burns, fractured or displaced teeth, facial bone and jaw fractures, discolored teeth, multiple injuries in various stages of healing, oral/peri-oral gonorrhea

Dental Management

Treat oral/dental injury as definitively as possible at emergent visit since child might not return for further care in near future

IV. SUBSTANCE ABUSE

- Substance abuse refers to the overindulgence in and dependence on a stimulant, depressant, chemical substance, herb (plant) or fungus leading to effects that are detrimental to the individual's physical health or mental health, or the welfare of others
- The disorder is characterized by a pattern of continued pathological use of a medication; non-medically indicated drug or toxin, that results in repeated adverse social consequences related to drug use, such as failure to meet work, family, or school obligations, interpersonal conflicts, or legal problems
- Substance abuse may lead to addiction or substance dependence. Medically, dependence requires the development of tolerance leading to withdrawal symptoms. Both abuse and dependence are distinct from addiction which involves compulsion to continue using the substance despite the negative consequences, and may or may not involve chemical dependency.
- Substance abuse is sometimes used as a synonym for drug abuse, drug addiction and chemical dependency but actually refers to the use of substances in a manner

outside socio-cultural conventions. All use of illicit and all use of licit drugs in a manner not dictated by convention (e.g. according to physician's orders) is abuse according to this definition, however there is no universally accepted definition of substance abuse.

- Results of the 2005 Monitoring the Future Study report that approximately 50% of American students in secondary school will experiment with at least one illicit drug before graduating from high school
- Oral health professionals are in an ideal position to identify substance use disorders (SUD) and related problems in the children, adolescents and families they care for and should be able to provide preventive guidance, education and intervention
- Pediatric oral health care providers are in a unique position to screen for SUD not only in children and adolescents but also in their parents and other family members
- Some adolescents and children will experiment and stop, or continue to use occasionally, without significant problems. Others will develop a dependency, moving on to more dangerous drugs and causing significant harm to themselves and possibly others.
- Using alcohol and tobacco at a young age increases the risk of using other drugs later. Youth at risk for developing serious alcohol and drug problems include those
 - with a family history of substance abuse
 - who are depressed
 - who have low self-esteem
 - who feel like they don't fit in or are out of the mainstream
- Teenagers abuse a variety of drugs, both legal and illegal. Legally available drugs include alcohol, prescribed medications, inhalants (fumes from glues, aerosols, and solvents) and over-the-counter cough, cold, sleep, and diet medications. The most commonly used illegal drugs are marijuana (pot), stimulants (cocaine, crack, and speed), LSD, PCP, opiates, heroin, and designer or club drugs (Ecstasy)
- Drug and alcohol use is associated with a variety of negative consequences including:
 - increased risk of serious drug use later in life
 - school failure
 - poor judgment that puts youth at risk for accidents
 - violence
 - unplanned and unsafe sex
 - suicide

Warning Signs of Youth Substance Abuse

Physical—Fatigue, sleep problems, repeated health complaints, red and glazed eyes and lasting cough

Emotional—Personality change, sudden mood changes, irritability, irresponsible behavior, low self esteem, depression, withdrawal and general lack of interest

Family—Starting arguments, breaking rules, withdrawing from family

School—decreased interest, negative attitude, and drops in grades, many absences, truancy, and discipline problems

Social/behavioral—peer group involved with drugs and alcohol, problems with the law, dramatic change in dress and appearance

- Some of the warning signs can also be signs of other emotional problems. When

parents are concerned they should be referred to their child's family physician as a first step. If drug or alcohol use/abuse is suspected, then the teen should have a comprehensive evaluation by a child and adolescent psychiatrist or other qualified mental health professional

- The CAGE screening questionnaire is used to screen for alcohol abuse and dependence in adults. It is not used to diagnose the disease but only to indicate whether a problem may exist. If you answer "yes" to even 1 of the questions, you may have a problem with alcohol:
 - Have you ever felt you ought to Cut down on your drinking or drug use?
 - Do you get Annoyed at criticism of your drinking or drug use?
 - Do you ever feel Guilty about your drinking or drug use?
 - Do you ever take an Early-morning drink (eye-opener) or use drugs first thing in the morning ("a little hair of the dog that bit you") to get the day started or eliminate the "shakes"?

Characteristics of Alcohol, Tobacco and Drugs

Alcohol

- The average age when youth first try alcohol is 11 years for boys and 13 years for girls. The average age at which Americans begin drinking regularly is 15.9 years old.
- According to research by the National Institute on Alcohol Abuse and Alcoholism, adolescents who begin drinking before age 15 are four times more likely to develop alcohol dependence than those who begin drinking at age 21
- An early age of drinking onset is also associated with alcohol-related violence not only among persons under 21 but among adults as well
- It has been estimated that over three million teenagers are out-and-out alcoholics. Several million more have a serious drinking problem that they cannot manage on their own.
- Dependence on alcohol is also associated with several psychiatric problems, such as:
 - Depression
 - Anxiety
 - Oppositional defiant disorder (ODD)
 - Antisocial personality disorder

The National Drug and Alcohol Treatment Referral Routing Service provides a toll-free telephone number, (800) 662-HELP (4357), offering various resource information. Through this service you can speak directly to a representative concerning substance abuse treatment, request printed material on alcohol or other drugs, or obtain local substance abuse treatment referral information in your State.

V. BRIEF SUMMARY OF DRUGS

Club Drugs

MDMA (ecstasy), Rohypnol, GHB, and ketamine are among the drugs used by teens and young adults who are part of a nightclub, bar, rave, or trance scene. Raves and trance events are generally night-long distance, often held in warehouses.

Crack and Cocaine

- Cocaine is a powerfully addictive stimulant drug. The powdered, hydrochloride salt form of cocaine can be snorted or dissolved in water and injected. Crack is cocaine that has not been neutralized by an acid to make the hydrochloride salt. This form of cocaine comes in a rock crystal that can be heated and its vapors

smoked. The term “crack” refers to the crackling sound heard when it is heated.

- Regardless of how cocaine is used or how frequently, a user can experience acute cardiovascular or cerebrovascular emergencies, such as a heart attack or stroke, which could result in sudden death. Cocaine-related deaths are often a result of cardiac arrest or seizure followed by respiratory arrest.
- Cocaine is a strong central nervous system stimulant that interferes with the reabsorption process of dopamine, a chemical messenger associated with pleasure and movement. The buildup of dopamine causes continuous stimulation of receiving neurons, which is associated with the euphoria commonly reported by cocaine abusers.
- Physical effects of cocaine use include constricted blood vessels, dilated pupils, and increased temperature, heart rate, and blood pressure. The duration of cocaine’s immediate euphoric effects, which include hyperstimulation, reduced fatigue, and mental alertness, depends on the route of administration. Some users of cocaine report feelings of restlessness, irritability, and anxiety.

Heroin

- Heroin is an addictive drug, and its use is a serious problem in America. Heroin is processed from morphine, a naturally occurring substance extracted from the seedpod of the Asian poppy plant. Heroin usually appears as a white or brown powder. Street names for heroin include “smack,” “H,” “skag,” and “junk.” Other names may refer to types of heroin produced in a specific geographical area, such as “Mexican black tar.”
- Heroin abuse is associated with serious health conditions, including fatal overdose, spontaneous abortion, collapsed veins, and, particularly in users who inject the drug, infectious diseases, including HIV/AIDS and hepatitis.

Inhalants

- Inhalants are breathable chemical vapors that produce psychoactive (mind-altering) effects. A variety of products common in the home and in the workplace contain substances that can be inhaled. Many people do not think of these products, such as spray paints, glues, and cleaning fluids, as drugs because they were never meant to be used to achieve an intoxicating effect. Yet, young children and adolescents can easily obtain them and are among those most likely to abuse these extremely toxic substances.
- Inhalants fall into the following categories:

Volatile Solvents

- Industrial or household solvents or solvent-containing products, including paint thinners or removers, degreasers, dry-cleaning fluids, gasoline, and glue
- Art or office supply solvents, including correction fluids, felt-tip marker fluid, and electronic contact cleaners

Aerosols

- Industrial aerosol propellants and associated solvents in items such as spray paints, hair or deodorant sprays, fabric protector sprays, aerosol computer cleaning products, and vegetable oil sprays

Gases

- Gases used in household or commercial products, including butane lighters and propane tanks, whipping cream aerosols or dispensers (whippets), and refrigerant gases

- Medical anesthetic gases, such as ether, chloroform, halothane, and nitrous oxide (“laughing gas”)

Nitrites

- Organic nitrites are volatiles that include cyclohexyl, butyl, and amyl nitrites, commonly known as “poppers.” Amyl nitrite is still used in certain diagnostic medical procedures. Volatile nitrites are often sold in small brown bottles labeled as “video head cleaner,” “room odorizer,” “leather cleaner” or “liquid aroma.”
- Although they differ in makeup, nearly all abused inhalants produce short-term effects similar to anesthetics, which act to slow down the body’s functions
- Sniffing highly concentrated amounts of the chemicals in solvents or aerosol sprays can directly induce heart failure and death within minutes of a session of repeated inhalations. This syndrome, known as “sudden sniffing death,” can result from a single session of inhalant use by an otherwise healthy young person.
- High concentrations of inhalants also can cause death from suffocation by displacing oxygen in the lungs and then in the central nervous system so that breathing ceases. Chronic abuse of solvents can cause severe, long-term damage to the brain, the liver, and the kidneys.

LSD

LSD (lysergic acid diethylamide) is one of the major drugs making up the hallucinogen class of drugs. Hallucinogens cause hallucinations—profound distortions in a person’s perception of reality. Hallucinogens cause their effects by disrupting the interaction of nerve cells and the neurotransmitter serotonin. Distributed throughout the brain and spinal cord, the serotonin system is involved in the control of behavioral, perceptual, and regulatory systems, including mood, hunger, body temperature, sexual behavior, muscle control, and sensory perception.

Marijuana

- Marijuana is the most commonly abused illicit drug in the United States. A dry, shredded green/brown mix of flowers, stems, seeds, and leaves of the hemp plant *Cannabis sativa*, it usually is smoked as a cigarette (joint, nail), or in a pipe (bong). It also is smoked in blunts, which are cigars that have been emptied of tobacco and refilled with marijuana, often in combination with another drug. It might also be mixed in food or brewed as a tea.
- The short-term effects of marijuana can include problems with memory and learning; distorted perception; difficulty in thinking and problem solving; loss of coordination; and increased heart rate
- One study has indicated that a user’s risk of heart attack more than quadruples in the first hour after smoking marijuana
- Someone who smokes marijuana regularly may have many of the same respiratory problems that tobacco smokers do, such as daily cough and phlegm production, more frequent acute chest illness, a heightened risk of lung infections, and a greater tendency to obstructed airways
- Smoking marijuana possibly increases the likelihood of developing cancer of the head or neck
- Marijuana abuse also has the potential to promote cancer of the lungs and other parts of the respiratory tract because it contains irritants and carcinogens

Methamphetamine

Methamphetamine is an addictive stimulant drug that strongly activates certain systems in the brain.

- Methamphetamine releases high levels of the neurotransmitter dopamine, which stimulates brain cells, enhancing mood and body movement. It also appears to have a neurotoxic effect, damaging brain cells that contain dopamine as well as serotonin, another neurotransmitter. Over time, methamphetamine appears to cause reduced levels of dopamine, which can result in symptoms like those of Parkinson's disease, a severe movement disorder. Methamphetamine is taken orally or intranasally (snorting the powder), by intravenous injection, and by smoking.
- The central nervous system (CNS) actions that result from taking even small amounts of methamphetamine include increased wakefulness, increased physical activity, decreased appetite, increased respiration, hyperthermia, and euphoria. Other CNS effects include irritability, insomnia, confusion, tremors, convulsions, anxiety, paranoia, and aggressiveness. Hyperthermia and convulsions can result in death.

Commonly Abused Prescription Medications

- Opioids - often prescribed to treat pain.
- CNS Depressants - used to treat anxiety and sleep disorders.
- Stimulants - prescribed to treat narcolepsy and attention deficit/hyperactivity disorders
- 2003 National Survey on Drug Use and Health (NSDUH) According to the 2003 NSDUH, an estimated 6.3 million persons, or 2.7 percent of the population age 12 and older had used prescription psychotherapeutic medications nonmedically in the month prior to being surveyed. This includes 4.7 million using pain relievers, 1.8 million using tranquilizers, 1.2 million using stimulants, and 0.3 million using sedatives.

Phencyclidine

PCP (phencyclidine) was developed in the 1950s as an intravenous anesthetic. Its use in humans was discontinued in 1965, because patients often became agitated, delusional, and irrational while recovering from its anesthetic effects.

- PCP is a white crystalline powder that is readily soluble in water or alcohol
- PCP is addictive – its repeated abuse can lead to craving and compulsive PCP-seeking behavior
- At low to moderate doses, physiological effects of PCP include a slight increase in breathing rate and a pronounced rise in blood pressure and pulse rate. Breathing becomes shallow, and flushing and profuse sweating occur. Generalized numbness of the extremities and loss of muscular coordination also may occur.
- At high doses of PCP, blood pressure, pulse rate, and respiration drop. This may be accompanied by nausea, vomiting, blurred vision, flicking up and down of the eyes, drooling, loss of balance, and dizziness. High doses of PCP can also cause seizures, coma, and death (though death more often results from accidental injury or suicide during PCP intoxication). High doses can cause symptoms that mimic schizophrenia, such as delusions, hallucinations, paranoia, disordered thinking, a sensation of distance from one's environment, and catatonia. Speech is often sparse and garbled.
- People who abuse PCP for long periods report memory loss, difficulties with speech and thinking, depression, and weight loss. These symptoms can persist up to a

year after stopping PCP abuse. Mood disorders also have been reported. PCP has sedative effects, and interactions with other central nervous system depressants, such as alcohol and benzodiazepines that can lead to coma.

Methylphenidate (Ritalin)

Methylphenidate is a medication prescribed for individuals (usually children) who have attention-deficit hyperactivity disorder (ADHD).

Anabolic steroids

- “Anabolic” refers to muscle-building, and “androgenic” refers to increased masculine characteristics. “Steroids” refers to the class of drugs. These drugs are available legally only by prescription, to treat conditions that occur when the body produces abnormally low amounts of testosterone, such as delayed puberty and some types of impotence.
- They are also prescribed to treat body wasting in patients with AIDS and other diseases that result in loss of lean muscle mass
- The major side effects from abusing anabolic steroids can include liver tumors and cancer, jaundice (yellowish pigmentation of skin, tissues, and body fluids), fluid retention, high blood pressure, increases in LDL (bad cholesterol), and decreases in HDL (good cholesterol). Other side effects include kidney tumors, severe acne, and trembling. In addition, there are some gender-specific side effects:
- For men — shrinking of the testicles, reduced sperm count, infertility, baldness, development of breasts, increased risk for prostate cancer
- For women — growth of facial hair, male-pattern baldness, changes in or cessation of the menstrual cycle, enlargement of the clitoris, deepened voice
- For adolescents — growth halted prematurely through premature skeletal maturation and accelerated puberty changes. This means that adolescents risk remaining short for the remainder of their lives if they take anabolic steroids before the typical adolescent growth spurt.
- Scientific research also shows that aggression and other psychiatric side effects may result from abuse of anabolic steroids. Depression often is seen when the drugs are stopped. Users may suffer from paranoid jealousy, extreme irritability, delusions, and impaired judgment stemming from feelings of invincibility.

VI. TOBACCO USE AMONG YOUTH

- 3 million kids under the age of 18 are current smokers
- Almost one-quarter of our children are current smokers by the time they leave high school; 14.9 percent of 10th graders and 9.3 percent of 8th graders are current smokers.
- More than 6.4 million children under age 18 alive today will eventually die from smoking-related disease, unless current rates are reversed
- 21.7 percent of all high school students are current smokers. (21.6% of males and 21.8% of females)
- 30.7 percent of high school boys report past-month use of tobacco (cigarettes, spit tobacco, and/or cigars); 9.9 percent of high school boys report past-month spit (smokeless) tobacco use

- Almost 90 percent of adult smokers began at or before age 18
- More than a third of all kids who ever try smoking a cigarette become regular, daily smokers before leaving high school
- 82.8 percent of youth (12-17) smokers prefer Marlboro, Camel and Newport – three heavily advertised brands
- 20.9 percent of adults (23.4% of men and 18.5% of women) are current smokers

Spit (Smokeless) Tobacco

- Since 1970, smokeless or spit tobacco has gone from a product used primarily by older men to one used predominantly by young men and boys
- From 1970 to 1991, the regular use of moist snuff by 18-24 year old males increased almost ten-fold, from less than one percent to 6.2 percent. Conversely, use among males 65 and older decreased by almost half, from 4 to 2.2 percent.
- Among all high school seniors who have ever used spit tobacco almost three-fourths began by the ninth grade
- Despite some recent declines in youth spit tobacco use, 9.9 percent of all boys in U.S. high schools – and 1.2 percent of high-school girls – currently use spit tobacco products. In some states, spit tobacco use among high school males is particularly high, including Kentucky (23.5%), South Dakota (23.5%), West Virginia (23.3%), and Oklahoma (23.0%).
- Spit tobacco products have been marketed to youth through a number of channels, including sports events like auto racing and rodeos that are widely attended by kids
- Spit tobacco causes leukoplakia, characterized by white patches and oral lesions on the cheeks, gums, and/or tongue. Leukoplakia, which can lead to oral cancer, occurs in more than half of all users in the first three years of use. Studies have found that 60 to 78 percent of spit tobacco users have oral lesions.
- Spit tobacco contains nitrosamines, proven carcinogens, as well as 30 metals and a radioactive compound called polonium-210
- Chewing tobacco has been linked to dental caries. A study by the National Institutes of Health and the Centers for Disease Control and Prevention found chewing tobacco users were four times more likely than non-users to have decayed dental root surfaces. Spit tobacco also causes gum disease (gingivitis), which can lead to bone and tooth loss.
- High school students who use spit tobacco 20 to 30 days per month are nearly four times more likely to currently use marijuana than nonusers, almost three times more likely to ever use cocaine, and nearly three times more likely to ever use inhalants to get high. In addition, heavy users of smokeless or spit tobacco are almost 16 times more likely than nonusers are to currently consume alcohol, as well.

Types of Spit Tobacco

- Oral (moist) snuff is a finely cut, processed tobacco, which the user places between the cheek and gum, which releases nicotine which, in turn, is absorbed by the membranes of the mouth
- Looseleaf chewing tobacco is stripped and processed cigar-type tobacco leaves that are loosely packed to form small strips. It is often sold in a foil-lined pouch and usually treated with sugar or licorice.

- Plug chewing tobacco consists of small, oblong blocks of semi-soft chewing tobacco that often contain sweeteners and other flavoring agents
- Nasal snuff is a fine tobacco powder that is sniffed into the nostrils. Flavorings may be added during fermentation, and perfumes may be added after grinding.

5A's Brief Intervention to Treat Tobacco Dependence

The Public Health Service 5A's Brief Intervention to Treat Tobacco Dependence can be effective with parents and children.

For children, a 6th 'A' has been added: ANTICIPATE

Children need our forethought regarding the problems that arise for them. It is also important to consider our children's needs with respect to age group.

For the first 4 years of life, our primary concerns are with passive smoke exposure.

ANTICIPATE – Passive exposure initiation

Educate parents regarding consequences of passive smoke exposure

Acute respiratory illnesses

Hospitalization for bronchitis or pneumonia

Chronic cough

Chronic middle ear infections

ASK parents about smoking in the home, and smoking among all those who have regular contact with their children

ASSESS readiness to engage in a cessation effort

ADVISE parents to stop using tobacco. Link passive exposure to tobacco smoke to children's health problems

ASSIST parents in quitting by referral to local resources

ARRANGE for follow-up; offer regular tobacco-free messages each time

For children ages 5 -12 additional concerns become important.

ANTICIPATE by educating parents concerning the following:

- Parents who use tobacco can influence children to experiment with tobacco
- Passive smoke exposure from friends who smoke
- Poor school performance is associated with tobacco use
- Children are influenced by positive attitudes about using tobacco

ASK 5-12 year old children

- Whether they believe there is any harm trying tobacco
- Whether they have tried it

ASK parents about their tobacco use, and whether they have warned their children re: the hazards of tobacco use

ADVISE 5-12 year old children

- About the short-term consequences of tobacco use
- To be prepared to refuse tobacco
- To stop if they have started

ASSESS motivation and readiness to carry out these tasks

ASSIST children in staying away from tobacco:

- Praise nonusers
- Help users develop refusal skills
- Mention that advertisements mislead by implying tobacco use makes one important or “cool”

ASSIST parents in quitting

ARRANGE more frequent follow-up visits for children experimenting with tobacco

ARRANGE follow-up for tobacco-using parents, and provide messages each time you see the parents.

The third age group (13-20) is comprised of adolescents and young adults.

In addition to preventing environmental tobacco smoke (ETS) exposure and the onset of tobacco use, efforts to promote cessation are needed.

ANTICIPATE risk factors at this stage:

- Dropping out of school
- Negative peer influence
- Overestimating tobacco use prevalence
- Self-image concerns

Be prepared to discuss tobacco products, the addictive potential, social factors, and that adolescents are a primary marketing target.

ASK whether the teen or their friends smoke, chew, or dip. Be sure they understand spit tobacco is not a safe alternative to smoking

ADVISE the teen to stop using tobacco. Help the teen to generate personal, short-term reasons to stop, such as:

- Reduce athletic capability
- Cost
- Stains and odors
- Burns and fire hazards

ASSESS readiness to stop use

ASSIST by:

- Setting a quit date
- Providing a 5-A's based intervention
- Providing literature
- Encouraging activities incompatible with tobacco use (e.g., sports)

ARRANGE for follow-up

- Tip for working with adolescents:
- The interaction
- Have a private discussion
- Don't speak from behind a desk or while patient is reclined
- Be a partner...don't lecture
- Be truthful

Long term outcomes are not a motivator with youth such as “You will get cancer, heart disease etc”

Focus on:

- Social issues
- Reality of “choice” in decision to use
- Physical endurance and sports
- Family and friends who have had negative outcomes with tobacco
- Dispel myths (example: everyone is not using tobacco, only about 25% of population)
- Personal issues
- Quit with a friend

VI. ADDITIONAL READINGS AND WEB SITES

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4. Substance Abuse
aacap.org
5. Tobacco
<http://www.oas.samhsa.gov/nhsda.htm>
<http://www.cdc.gov/tobacco/datahighlights/index.htm>

Chapter 26: RESOURCE SECTION

- I. IMMUNE DEFICIENCIES**
- II. MISSING AND EXPLOITED CHILDREN**
- III. COMMONLY ABUSED SUBSTANCES**
- IV. MEDICATIONS FOR TOBACCO CESSATION**
- V. DENTAL GROWTH AND DEVELOPMENT**
- VI. GROWTH CHARTS**
- VII. BODY MASS INDEX (BMI) CHARTS**
- VIII. FOOD PYRAMID**
- IX. IMMUNIZATION SCHEDULE**
- X. SPEECH AND LANGUAGE MILESTONES**
- XI. RECORD TRANSFER**
- XII. COMMON LABORATORY VALUES**
- XIII. COMMON PEDIATRIC MEDICATIONS**
- XIV. MANAGEMENT OF MEDICAL EMERGENCIES**
- XV. CARDIOPULMONARY RESUSCITATION**

I. IMMUNE DEFICIENCIES

DEFECT	EPIDEMIOLOGY/ CHARACTERISTICS	LABORATORY DIAGNOSIS	MEDICAL & DENTAL COMPLICATIONS	MANAGEMENT
PRIMARY HUMORAL (B-CELL) IMMUNODEFICIENCIES (~50% of all primary immunodeficiencies):				
Agammaglobulinemia - X-linked	defect in Bruton's tyrosine kinase (Btk).	Males only. Diagnosed between 6-18 months of age. Both sexes (15%).	normal T-cells, absent or very low B-cells on flow cytometry, reduced serum immunoglobulins	Intravenous immunoglobulin (Ig)
- Autosomal recessive	mutation in one of several genes			Recurrent bacterial infections, odontogenic infections predispose to septicemia, recurrent oral aphthae
Hyper IgM Syndromes	multiple: deficiency of CD40 ligand x-linked or autosomal recessive, defect in class switching, NEMO syndrome	Primarily males (CD40 ligand). Females autosomal recessive	IgG <200 mg/ml, no IgG responsive antibodies, IgM 100-3700 mg/ml	Recurrent severe sinopulmonary infections, hepatitis, lymphoid hyperplasia, autoimmune disease. Oral candidiasis and ulceration are common, Ludwig's angina can occur.
Selected Immunoglobulin isotype deficiencies	Defect in B-cell differentiation with reduced/no production of selected isotype. IgA deficiency is most common	Both sexes. Usually diagnosed >4years old	Selective IgA deficiency	Prophylactic antibiotics, severe subclass deficiency may require Ig replacement

PRIMARY CELLULAR (T-CELL) IMMUNODEFICIENCIES:			
DiGeorge Syndrome	<p>Autosomal dominant/ spontaneous.</p> <p>3rd & 4th pharyngeal pouch maldevelopment with thymic hypoplasia / agenesis leading to deficient T-cell maturation.</p> <p>Deletion in chromosome 22q11.2. Velo-cardiofacial syndrome (VCFS) occurs in 90% of cases.</p>	<p>Both sexes. Diagnosed <6 months old. Up to 1:4000 births.</p> <p>cytometry, normal or decreased serum immunoglobulins</p>	<p>Decreased T-cells, normal B-cells on flow cytometry, normal or decreased serum immunoglobulins</p> <p>Mycobacterial, viral and fungal infections, hypocalemic tetany sec. to absent parathyroid, VCFS patients have palatal abnormalities (overt cleft palate, velopharyngeal incompetence, and submucosal cleft palate), conotruncal heart disease, prominent nose & squared nasal route, mental retardation. Oral candidiasis, herpes infection are common, enamel hypoplasia may occur.</p>

DEFECT	EPIDEMIOLOGY/ CHARACTERISTICS	LABORATORY DIAGNOSIS	MEDICAL & DENTAL COMPLICATIONS	MANAGEMENT
COMBINED IMMUNODEFICIENCIES:				
Severe combined immunodeficiency (SCID), x-linked	Mutation in gene on x-chromosome encoding the common gamma chain shared by receptors for IL-2,4,7,9 and 15	x-linked, males. Diagnosed < 6 months old	Markedly decreased T cells and NK cells, normal or increased B cells, reduced serum immunoglobulins	Recurrent, severe bacterial infection, diarrhea, failure to thrive. Usually begins in infancy. Includes candidiasis, fatal viral infections after attenuated vaccine, GVH reactions to transfusion. Fatal if not treated. Oral candidiasis, herpes, recurrent tongue and buccal mucosa ulceration, severe necrotizing ulcerative gingival stomatitis.
Adenosine deaminase (ADA) or Purine nucleoside phosphorylase (PNP) deficiencies	Absence of enzyme (ADA) leads to buildup of toxic purine metabolites in lymphocytes	ADA 1:200,000 live births (20% of SCID cases), autosomal recessive	Lymphopenia. ADA deficiency; Progressive decrease in T and B cells, reduced serum immunoglobulins	Clinical picture similar to x-linked SCID. Absent tonsils and lymph tissue. Partial ADA defect may present later, into adulthood. Lymphomas complicate partial and treated complete ADA deficiency

Ataxia-Telangiectasia Syndrome	Defective ATM gene (AT mutated) on chromosome 11q22.3 allowing a buildup of somatic mutations	Both sexes. Autosomal recessive. Usually diagnosed > 5 years old. 1:20,000-1:100,000 live births. 1.4-2% Caucasians in US are carriers.	Elevated alpha-fetoprotein in child > 8 months, immunoglobulin deficiency, poor production of antibody to bacteria containing polysaccharides in cell wall	Progressive cerebellar ataxia, oculocutaneous telangiectasia, diabetes mellitus, increased malignancies, progressive pulmonary disease secondary to infections, poor prognosis	Antibiotics, gamma globulin infusions
Wiskott-Aldrich Syndrome	Defects in WASP gene located at Xp11.22-23	Primarily males, x-linked recessive, rare autosomal. Average age at diagnosis, approx. 21 months.	Variable picture from thrombocytopenia to effect on all blood cells (rearrangement of cytoskeleton), small platelet size	90% with bleeding from thrombocytopenia, recurrent infections especially Strep. Neisseria, Hemophilus, <i>cczcema</i> , autonomic disorders	HLA-matched bone marrow transplant preferred, if not available: splenectomy, intravenous immunoglobulins, antibiotics

DISORDERS OF INNATE IMMUNITY:	DEFECT	EPIDEMIOLOGY/ CHARACTERISTICS	LABORATORY DIAGNOSIS	MEDICAL & DENTAL COMPLICATIONS	MANAGEMENT
Chronic Granulomatous Disease	loss or inactivation of one of components of NADPH oxidase (PHOX).	Males > females. Often diagnosed < 5 years, can be in 20's or 30's. 1/200,000 live births, 70% x-linked, 30% autosomal recessive.	Decreased phagocytic NADPH oxidase activity determined by one of: NBT test, cytochrome reduction assay, oxidative (respiratory) burst assay (DHR fluorescence)	Recurrent intracellular fungal and bacterial infections (including <i>Staphylococcus aureus</i> , and <i>Candida</i> spp). Oral candidiasis, gingivitis, and oral ulcers similar to that of aphthous ulcers in presentation and course but affects the attached gingiva, discoid lesions, very rarely intraoral granulomas.	Subcutaneous injections of interferon gamma. Antibiotic and antifungal prophylaxis.
Leukocyte adhesion deficiency type I (LAD-I). Also LAD-II and LAD-III reported but extremely rare	Deficiencies or defects in CD-18 common chain of the beta-2 integrin family leads to defect in migration and chemotaxis of leukocytes; also defect in adhesion and transmigration through endothelial cells.	Rare, autosomal recessive, both severe and moderate phenotypes	Marked leukocytosis with infection (5-20x normal). Absent CD18/CD11b on flow cytometry	Delayed separation of the umbilical cord, recurrent bacterial infections, (primarily skin and mucosal surfaces), leukocytosis, periodontitis, absent plus formation, impaired wound healing. Marked linear gingivitis, rapidly progressing juvenile periodontitis, oral ulcers that heal very slowly with scarring. Severe phenotype often fatal, Moderate often survive into adulthood	Severe disease: bone marrow or stem cell transplantation. Mild-moderate disease: antibiotic therapy.

Cyclic neutropenia	Mutation in neutrophil elastase gene causing defect in stem cell regulation.	Childhood form autosomal dominant, diagnosed between 6–24 months of age.	Neutrophil counts fluctuate from normal to < 500/ microL on approx. 21-day cycles, neutropenia lasting 1 week.	Spectrum of disease from asymptomatic to severe infections of skin and mucus membranes. Symptoms often decrease as gets older. Localized or generalized early onset periodontitis, may escape the primary dentition. Gingivae are inflamed and edematous with gingival recession, ulceration and desquamation.	Antibacterial mouth rinses and good plaque control. Antibiotic therapy, G-CSF (subcutaneous, daily) to increase neutrophil count
Chediak-Higashi syndrome (CHS)	Defective gene (LYST) associated with defective transport of bacteria to lysosome and inability to kill phagocytosed bacteria.	CHS includes: Recurrent pyogenic infections, partial oculocutaneous albinism, neurologic abnormalities	Mild neutropenia with giant cytoplasmic granules, decreased chemotaxis, intracellular killing, NK cell dysfunction.	Partial albinism, photophobia, nystagmus, recurrent pyogenic infections, malignant lymphomas, accompanied by neutropenia, anaemia, thrombocytopenia. Severe gingival inflammation, rapidly progressing early childhood periodontitis with premature exfoliation of teeth	Aggressive antibacterial therapy, splenectomy, bone marrow transplant. Evaluate blood parameters prior to any dental procedures, consult with the patient's physician

II. MISSING AND EXPLOITED CHILDREN

Child Safety for Communities

- Amber Alert

The AMBER Alert Plan, named for 9-year-old **Amber Hagerman**, is a voluntary partnership between law-enforcement agencies, broadcasters, and transportation agencies to activate an urgent bulletin in the most serious child-abduction cases. Broadcasters use the Emergency Alert System (EAS) to air a description of the abducted child and suspected abductor. This is the same concept used during severe weather emergencies. The goal of an AMBER Alert is to instantly galvanize the entire community to assist in the search for and safe recovery of the child.

- Code Adam

Code Adam was created and named in memory of 6-year-old **Adam Walsh**. In 1981, Adam was abducted from a Florida shopping mall and later found murdered. This incident brought national attention to the horror of child abduction. Since the beginning of the Code Adam program in 1994, it has been a powerful search tool for lost and possibly abducted children in tens of thousands of establishments across the nation, and it is one of the country's largest child-safety programs. A Code Adam decal is posted at a building's entrance alerting the public of the location's participation in the program.

Child Identification Programs

AAPD Policy on Child Identification Programs – www.aapd.org/mediaPolicies_Guidelines/P_ChildIDPrograms.pdf

Child Identification Program (CHIPS) – sponsored by the Masons gathers blood samples for DNA fingerprinting

National Child Identification Program – sponsored by the American Football Coaches Association

ID card which includes fingerprints, physical descriptions, photographs, MD's address and phone

New England Kids Identification System (K.I.D.S.) sponsored by the Massachusetts Free Masons and Mass Dental Society

bite registration

AAPD recommends a dental component documenting a child's dental home with detailed dental records updated routinely.

III. COMMONLY ABUSED SUBSTANCES

Tobacco

Alternative Tobacco Products

R.J. Reynolds - the same company that used the cartoon character, Joe Camel, to market cigarettes to kids- is now marketing flavored cigarettes, including a pineapple and coconut-flavored cigarette called "Kauai Kolada" and a citrus-flavored cigarette called "Twista Lime." In November 2004, they introduced Camel "Winter Blends" in flavors including "Winter Warm Toffee" and "Winter MochaMint"

Brown & Williamson has its own flavored versions of Kool cigarettes with names like “Caribbean Chill,” “Midnight Berry,” “Mocha Taboo” and “Mintrigue.”

The U.S. Smokeless Tobacco Company is marketing spit tobacco with flavors such as berry blend, mint, and wintergreen, apple blend, vanilla and cherry.

www.tobaccofreekids.org

Ariva tobacco lozenges (Star Scientific, Inc.) – Ariva is a mint-flavored lozenge the size of a Tic-Tac, 60 percent of which is a compressed tobacco powder that comes in the shape of a candy like lozenge and is packaged similar to a smoking cessation product.

Omni (Vector Tobacco Ltd.) and Advance (Star Scientific, Inc. and Brown & Williamson Tobacco Corp.) “low carcinogen” cigarettes – Vector recently launched national advertising for its Omni cigarette with the slogan, “Reduced carcinogens, Premium taste.”

Eclipse (R.J. Reynolds Tobacco Holdings, Inc.) – Eclipse is not similar to a traditional cigarette. Eclipse primarily heats tobacco rather than burning it. According to the Eclipse petition, “Eclipse outwardly looks like a cigarette, but is in fact a sophisticated, technologically-advanced nicotine delivery system that is completely unlike traditional cigarettes.”

Nicotine Water (S.F. Garret) – This product is sold over the Internet. Nicotine Water is regular bottled water with the addition of nicotine equal to what the manufacturer claims is contained in two cigarettes.

www.tobaccofreekids.org

Bidis (pronounced “bee-dees”) are small, thin hand-rolled cigarettes imported to the United States primarily from India and other Southeast Asian countries. They consist of tobacco wrapped in a tendu or temburni leaf (plants native to Asia), and may be tied with a colorful string at one or both ends. Bidis may be flavored (e.g., chocolate, cherry, and mango) or unflavored. They have higher concentrations of tar, nicotine, and carbon monoxide than the typical cigarettes sold in the United States. Research studies from India indicate that bidi smoking is associated with an increased risk for oral cancer, as well as an increased risk for cancer of the lung, stomach, and esophagus. Research studies in India have also shown that bidi smoking is associated with a more than three-fold increased risk for coronary heart disease and acute myocardial infarction (heart attack), and a nearly four-fold increased risk for chronic bronchitis. No US research studies have been conducted.

www.cdc.gov/tobacco

Kreteks (pronounced “cree-techs”) are referred to as clove cigarettes. Imported from Indonesia, kreteks usually contain a mixture of tobacco, cloves, and other additives. As with bidis, standardized machine-smoking analyses indicate that kreteks deliver more nicotine, carbon monoxide, and tar than conventional cigarettes. There is no evidence to indicate that bidis or kreteks are safe alternatives to conventional cigarettes. Smoking kreteks is associated with an increased risk for acute lung injury, especially among susceptible individuals with asthma or respiratory infections. Research in Indonesia has shown that regular kretek smokers have 13-20 times the risk for abnormal lung function compared with nonsmokers. No research studies on the long-term health effects of kreteks have been conducted in the United States. www.cdc.gov/tobacco

Betel nut use refers to a combination of three ingredients: the nut of the betel palm (*Areca catechu*), part of the *Piper betel* vine, and lime. Anecdotal reports have indicated that small doses generally lead to euphoria and increased flow of energy while large doses often result in sedation. Although all three ingredients may contribute to these effects, most experts attribute the psychoactive effects to the alkaloids found in betel nuts.

Adults (18 years and older)

Oral (by mouth): Betel nut can be chewed alone, but is often chewed in combination with other ingredients (called a “quid”), including calcium hydroxide, water, catechu gum, cardamom, cloves, anise seeds, cinnamon, tobacco, nutmeg, and gold or silver metal. These ingredients may be wrapped in a betel leaf, followed by sucking the combination in the side of the mouth. It is reported that ingestion of 8 to 30 grams of areca nut may be deadly. Constituents of areca are potentially carcinogenic. Long-term use has been associated with oral submucous fibrosis (OSF), pre-cancerous oral lesions and squamous cell carcinoma. Acute effects of betel chewing include asthma exacerbation, hypotension, and tachycardia.

Herbal Cigarettes contain mixtures of herbs; some combine tobacco with cloves, dried tendu leaves (a plant from India and Southeast Asia), and other unusual ingredients. Alternative cigarettes that are sold at convenience stores and over the Internet – are easy to find and buy, especially for kids who aren’t old enough to buy traditional tobacco products. These products seem exotic and come in flavors that appeal to children such as cherry or vanilla. However, their biggest selling point is that they’re supposed to be a healthy alternative to “real” cigarettes. <http://healthresources.caremark.com/topic/herbalcig>

Houkah smoking started in the Middle East and involves burning flavored tobacco in a water pipe and inhaling the smoke through a long hose. It has become popular among young people, especially on and around college campuses. It is marketed as a safe alternative to cigarettes because the percent of tobacco in the product smoked is low. This safety claim is false. The water does not filter out all of the toxins, and hookah smoke does contain varying amounts of nicotine, carbon monoxide, and other dangerous substances. Numerous cancers, as well as many other health effects, have been linked to hookah smoking.

www.cancer.org

Spit Free Tobacco products are offered as an alternative to the usual smokeless or spit tobacco that requires chewing and spitting. These products are simply tobacco in a small packet that may be held between the lip or cheek and gums. Product names are Exalt and Revel and they can be purchased in mint and wintergreen flavors. www.nih.gov

Drugs

GHB, Ketamine, and Rohypnol

GHB and Rohypnol are predominantly central nervous system depressants. Because they are often colorless, tasteless, and odorless, they can be added to beverages and ingested unknowingly.

Ketamine

Ketamine is an anesthetic that has been approved for both human and animal use in medical settings since 1970; about 90 percent of the ketamine legally sold is intended for veterinary use. It can be injected or snorted. Ketamine is also known as “special K” or “vitamin K.”

Certain doses of ketamine can cause dream-like states and hallucinations. In high doses, ketamine can cause delirium, amnesia, impaired motor function, high blood pressure, depression, and potentially fatal respiratory problems.

Rohypnol

Rohypnol, a trade name for flunitrazepam, belongs to a class of drugs known as benzodiazepines. When mixed with alcohol, Rohypnol can incapacitate victims and prevent them from resisting sexual assault. Rohypnol may be lethal when mixed with alcohol and/or other depressants.

Rohypnol is not approved for use in the United States, and its importation is banned. Illicit use of Rohypnol started appearing in the United States in the early 1990s, where it became known as “rophies,” “roofies,” “roach,” and “rope”.

IV. MEDICATIONS FOR TOBACCO CESSATION

Medication 1st Line Options	Proper Use	Advantages	Disadvantages
Nicotine Transdermal Patch (Nicoderm, Nicotrol, Habitrol)	<ul style="list-style-type: none"> Stop tobacco 1 per day, on awakening 9 - 12 weeks Tapering option 	<ul style="list-style-type: none"> Effective blood levels within 1 - 2 hours Simple to use; 3 dose levels No new drug; Eliminates "tar" and CO Concern re: use with tobacco overstated 	<ul style="list-style-type: none"> Skin-related side effects common Caution with CV disease Max dose may not be enough for some
Nicotine Polacrilex ('gum') (Nicorette)	<ul style="list-style-type: none"> Stop tobacco Chew minimally and park for 30 minutes 1 piece every 1-2 hours Up to 24 pieces per day 12 weeks 	<ul style="list-style-type: none"> 2 mg (up to 24 cigs per day) 4 mg (25 or more cigs per day) Orange / Mint / Regular Oral substitute; Use as needed Good for 'irregular' smoker 	<ul style="list-style-type: none"> Insufficient use is common Chewing too much increases side effects Taste can be unpleasant (original flavor) No food or drink before or while using
Nicotine Inhaler (Nicotrol)	<ul style="list-style-type: none"> Stop tobacco 6 - 16 cartridges per day 12 weeks; can taper over 6-12 additional weeks Stop if not quit in 4 weeks 	<ul style="list-style-type: none"> Easy to tailor Oral substitute 	<ul style="list-style-type: none"> Lower level of delivery – may not be ideal for heavier users as sole therapy Costly
Nicotine Nasal Spray (Nicotrol NS)	<ul style="list-style-type: none"> Stop tobacco 1-2 doses per hour (1 dose = 1 spray in each nostril) Max: 5 doses per hour (40 doses /day) Do not inhale while spraying 12 weeks Stop if not quit in 4 weeks 	<ul style="list-style-type: none"> May be more useful with heavier users 	<ul style="list-style-type: none"> Irritation of nasal tract Cost

Nicotine Lozenge (Commit)	<ul style="list-style-type: none"> Stop tobacco Absorbed via oral mucosal Not eating or drinking 15 minutes before use Up to 6 per 5-hour period, max of 20 per day 12 weeks 	<ul style="list-style-type: none"> 2 mg (1st cigarette after 30 min of awakening) 4 mg (1st cigarette sooner than 30 min) Oral substitute Use as needed Good for 'irregular' smoker 	<ul style="list-style-type: none"> Consuming too fast can cause side effects No food or drink before or while using
Bupropion SR (Zyban)	<ul style="list-style-type: none"> Once per day for 3 days, then twice per day for at least 7-12 weeks (up to 6 months) Tapering at end of treatment not necessary 	<ul style="list-style-type: none"> Ease of use Can initiate while still using tobacco Antidepressant effect 	<ul style="list-style-type: none"> h/o seizure or eating disorder Abrupt stopping of alcohol, sedatives No MAOI or other form of Bupropion 1-2 weeks to reach adequate blood levels
Varenicline (Chantix)	<ul style="list-style-type: none"> 0.5 mg per day for 3 days, then 0.5 mg twice per day for 4 days, then 1 mg twice per day for 11 weeks If quit at 12 weeks, consider another 12 weeks Starter Pak and Continuation Pak Eat and full glass of water 	<ul style="list-style-type: none"> Best outcomes to date No CYP450 (liver) concerns No drug-drug interactions 	<ul style="list-style-type: none"> Nausea Should not be combined with NRT
2nd Line Options			
Nortriptyline • Not FDA approved	<ul style="list-style-type: none"> Monitor closely 25 mg /day, gradual increase to 75-100 mg /day 12 weeks 	<ul style="list-style-type: none"> 3x increase in abstinence over placebo 	<ul style="list-style-type: none"> Higher level of side effects Significant risk for CV patients
Clonidine • Not FDA approved	<ul style="list-style-type: none"> Monitor closely PO: 0.10 mg /day, increase by 0.10 mg /day as needed up to 0.75 mg /day TTS: .10 mg /day, increase to .20 mg /day as needed 3 – 10 weeks 	<ul style="list-style-type: none"> 2x abstinence rates compared with placebo Transdermal or oral 	<ul style="list-style-type: none"> Higher level of side effects, especially with abrupt D/C

V. DENTAL GROWTH AND DEVELOPMENT

Primary Dentition						
	Calcification begins at	Formation complete at	Eruption Maxillary	Eruption Mandibular	Exfoliation Maxillary	Exfoliation Mandibular
Central incisors	4th fetal mo	18-24 mo	6-10 mo	5-8 mo	7-8 y	6-7 y
Lateral incisors	5th fetal mo	18-24 mo	8-12 mo	7-10 mo	8-9 y	7-8 y
Canines	6th fetal mo	30-39 mo	16-20 mo	16-20 mo	11-12 y	9-11 y
First molars	5th fetal mo	24-30 mo	11-18 mo	11-18 mo	9-11 y	10-12 y
Second molars	6th fetal mo	36 mo	20-30 mo	20-30 mo	9-12 y	11-13 y

Permanent Dentition					
	Calcification begins at	Crown (enamel) complete at	Roots complete at	Eruption* Maxillary	Eruption* Mandibular
Central incisors	3-4 mo	4-5 y	9-10 y	7-8 y (3)	6-7 y (2)
Lateral incisors	Maxilla: 10-12 mo	4-5 y	11 y	8-9 y (5)	7-8 y (4)
	Mandible: 3-4 mo	4-5 y	10 y		
Canines	4-5 mo	6-7 y	12-15 y	11-12 y (11)	9-11 y (6)
First premolars	18-24 mo	5-6 y	12-13 y	10-11 y (7)	10-12 y (8)
Second premolars	24-30 mo	6-7 y	12-14 y	10-12 y (9)	11-13 y (10)
First molars	Birth	30-36 mo	9-10 y	5.5-7 y (1)	5.5-7 (1a)
Second molars	30-36 mo	7-8 y	14-16 y	12-14 y (12)	12-13 y (12a)
Third molars	Maxilla: 7-9 y			17-30 y (13)	17-30 y (13a)
	Mandible: 8-10 y				

*Figures in parentheses indicate order of eruption. Many otherwise normal infants do not conform strictly to the stated schedule.

Logan WHG, Kronfeld R. Development of the human jaws and surrounding structures from birth to the age of fifteen years. J Am Dent Assoc 1933;20(3):379-427. Copyright © 1933 American Dental Association. All rights reserved. Adapted 2003 by permission.

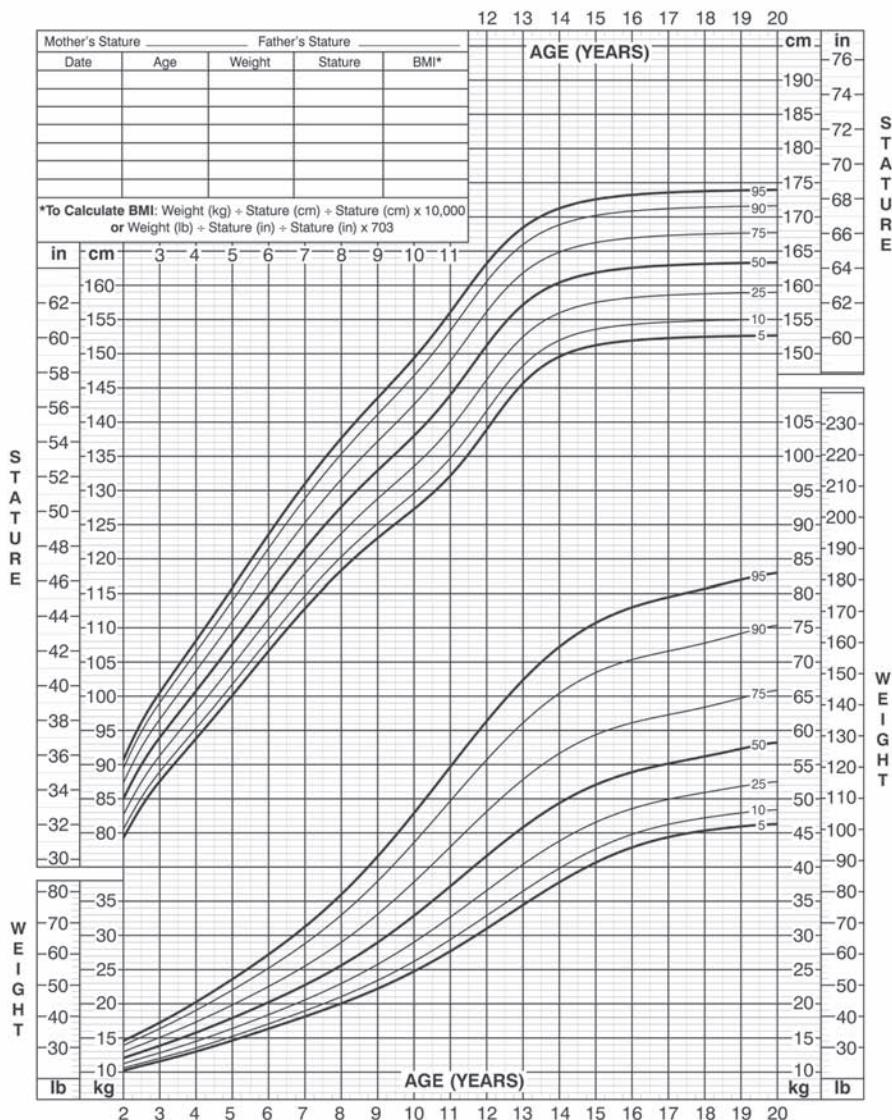
VI. GROWTH CHARTS

2 to 20 years: Girls

Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 11/21/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). <http://www.cdc.gov/growthcharts>

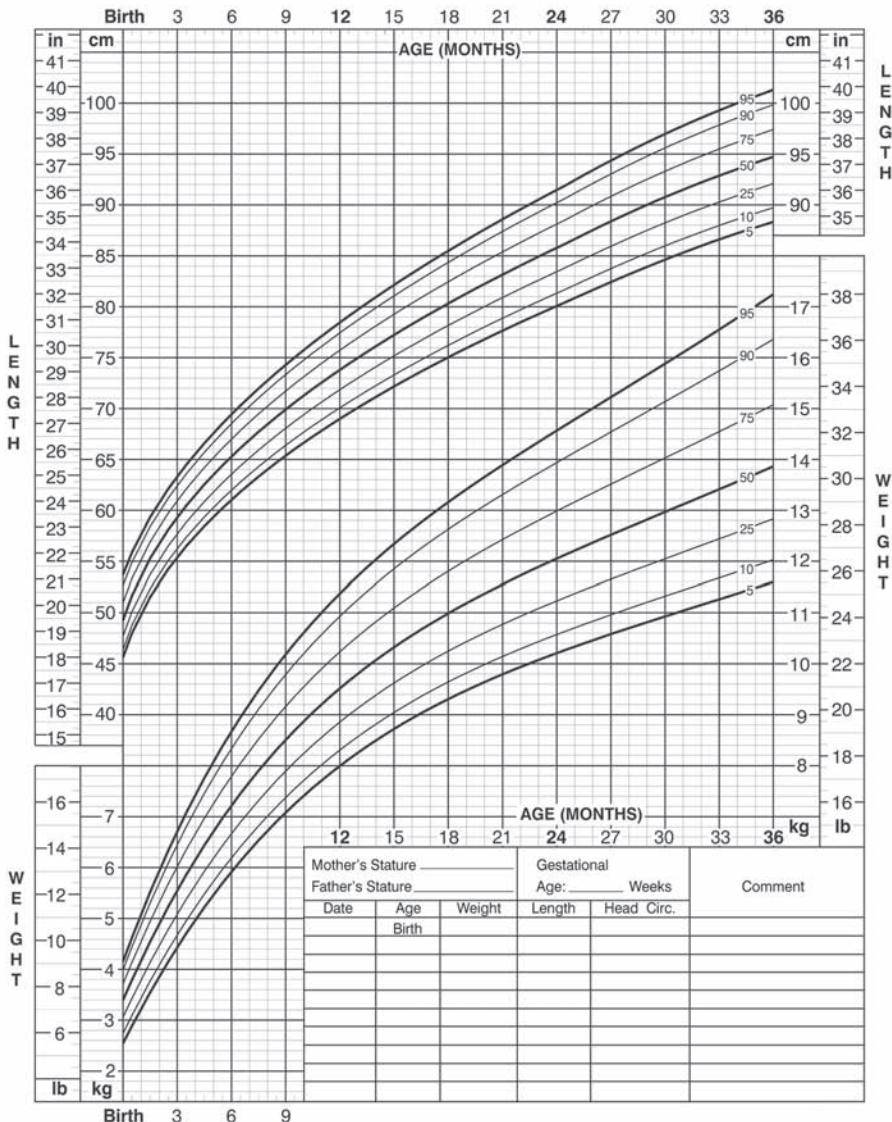


Birth to 36 months: Girls

Length-for-age and Weight-for-age percentiles

NAME _____

RECORD #



Published May 30, 2000 (modified 4/20/01)

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). <http://www.cdc.gov/growthcharts>



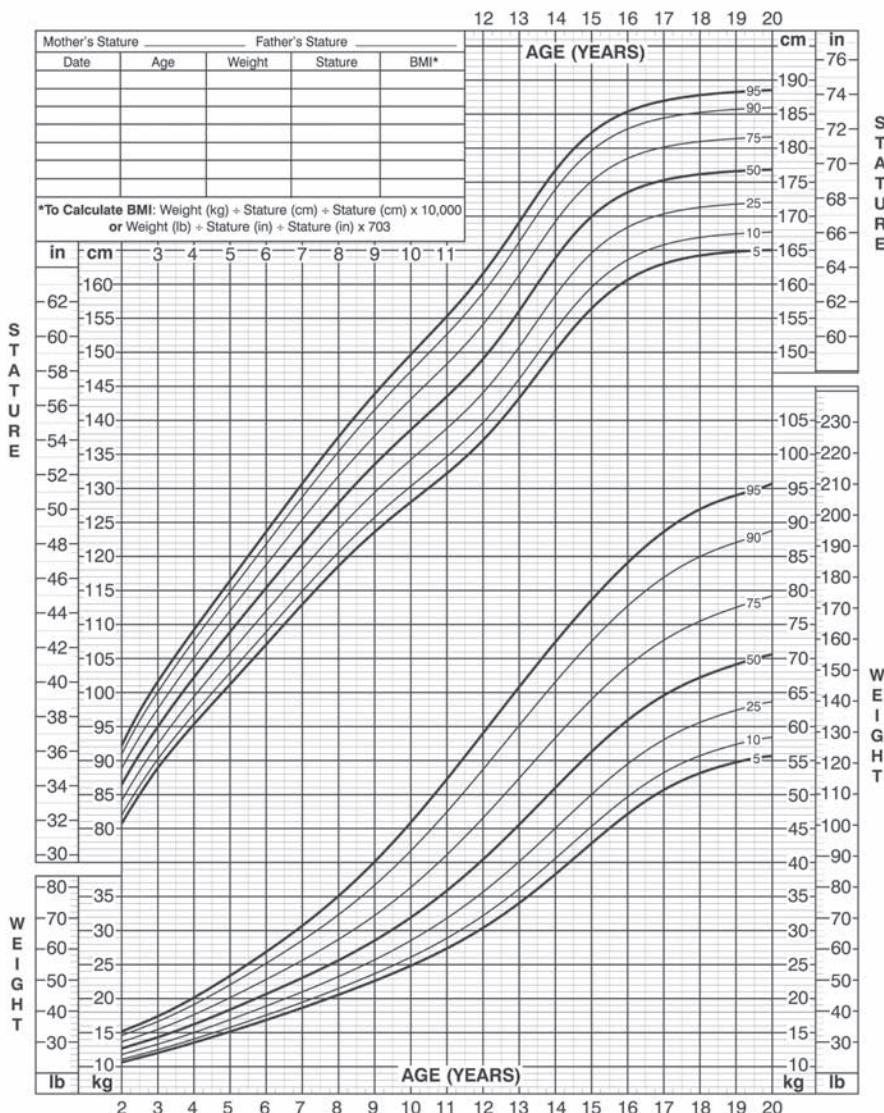
SAFER • HEALTHIER • PEOPLE™

2 to 20 years: Boys

Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 11/21/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with
the National Center for Chronic Disease Prevention and Health Promotion (2000).
<http://www.cdc.gov/growthcharts>

SAFER • HEALTHIER • PEOPLE™

2 to 20 years: Girls

Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____

Published May 30, 2000 (modified 11/21/00)

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). <http://www.cdc.gov/growthcharts>.



GOVERNMENT OF INDIA

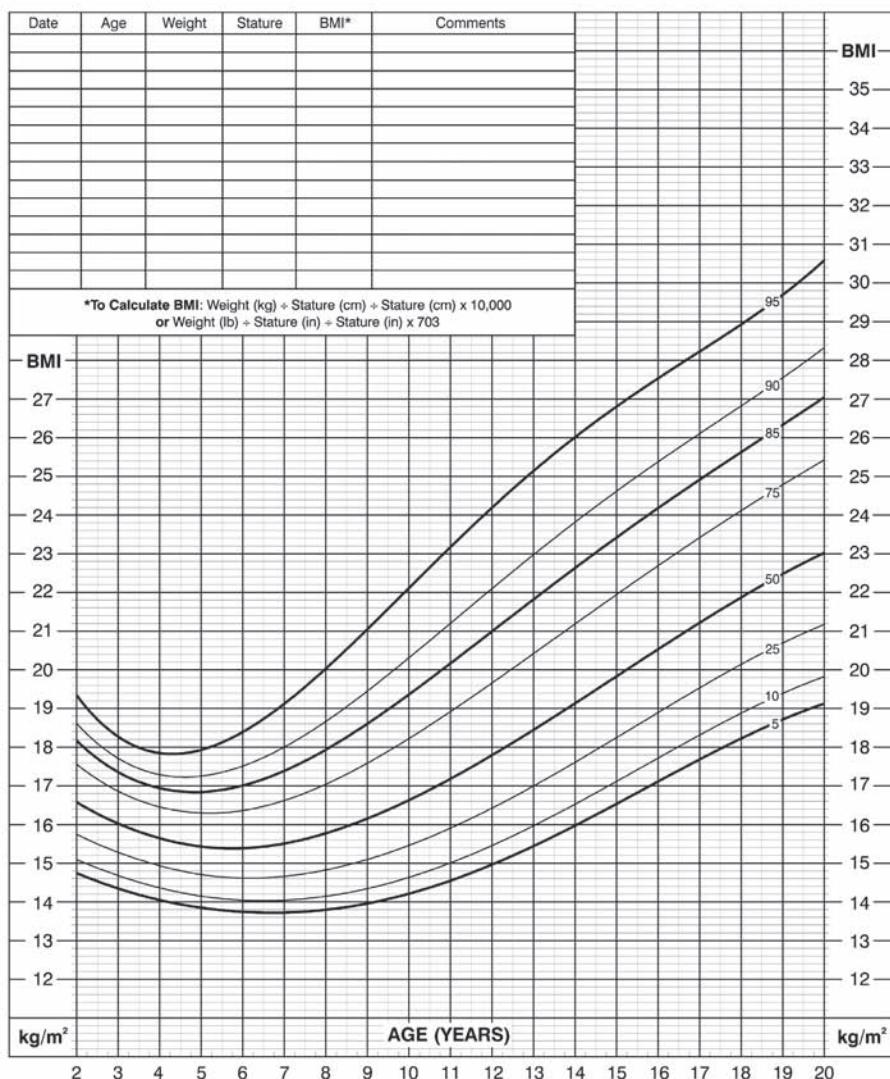
VII. BODY MASS INDEX (BMI) CHARTS

2 to 20 years: Boys

Body mass index-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 10/16/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). <http://www.cdc.gov/growthcharts>

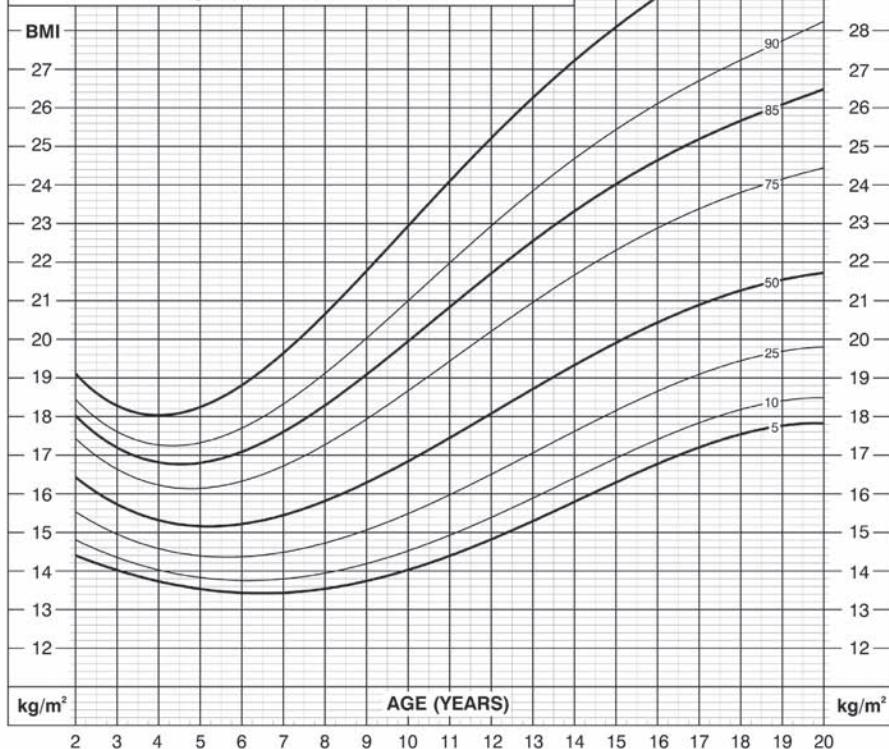
2 to 20 years: Girls

Body mass index-for-age percentiles

NAME _____

RECORD # _____

***To Calculate BMI:** Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 10/16/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). <http://www.cdc.gov/nchscharts>.

VIII. FOOD PYRAMID

Food Intake Patterns

The suggested amounts of food to consume from the basic food groups, subgroups, and oils to meet recommended nutrient intakes at 12 different calorie levels. Nutrient and energy contributions from each group are calculated according to the nutrient-dense forms of foods in each group (e.g., lean meats and fat-free milk). The table also shows the discretionary calorie allowance that can be accommodated within each calorie level, in addition to the suggested amounts of nutrient-dense forms of foods in each group.

Calorie Level ¹	1,000	1,200	1,400	1,600	1,800	2,000	2,200	2,400	2,600	2,800	3,000	3,200
Fruits ²	1 cup	1 cup	1.5 cups	1.5 cups	1.5 cups	2 cups	2 cups	2 cups	2 cups	2.5 cups	2.5 cups	2.5 cups
Vegetables ³	1 cup	1.5 cups	1.5 cups	2 cups	2.5 cups	2.5 cups	3 cups	3 cups	3.5 cups	3.5 cups	4 cups	4 cups
Grains ⁴	3 oz-eq	4 oz-eq	5 oz-eq	5 oz-eq	6 oz-eq	6 oz-eq	7 oz-eq	8 oz-eq	9 oz-eq	10 oz-eq	10 oz-eq	10 oz-eq
Meat and Beans ⁵	2 oz-eq	3 oz-eq	4 oz-eq	5 oz-eq	5 oz-eq	5.5 oz-eq	6 oz-eq	6.5 oz-eq	6.5 oz-eq	7 oz-eq	7 oz-eq	7 oz-eq
Milk ⁶	2 cups	2 cups	2 cups	3 cups	3 cups	3 cups	3 cups	3 cups	3 cups	3 cups	3 cups	3 cups
Oils ⁷	3 tsp	4 tsp	4 tsp	5 tsp	5 tsp	6 tsp	6 tsp	7 tsp	8 tsp	8 tsp	10 tsp	11 tsp
Discretionary calorie allowance ⁸	165	171	171	132	195	267	290	362	410	426	512	648

1 Calorie Levels are set across a wide range to accommodate the needs of different individuals. The attached table "Estimated Daily Calorie Needs" can be used to help assign individuals to the food intake pattern at a particular calorie level.

2 Fruit Group includes all fresh, frozen, canned, and dried fruits and fruit juices. In general, 1 cup of fruit or 100% fruit juice, or 1/2 cup of dried fruit can be considered as 1 cup from the fruit group.

3 Vegetable Group includes all fresh, frozen, canned, and dried vegetables and vegetable juices. In general, 1 cup of raw or cooked vegetables or vegetable juice, or 2 cups of raw leafy greens can be considered as 1 cup from the vegetable group.

Calorie Level	1,000	1,200	1,400	1,600	1,800	2,000	2,200	2,400	2,600	2,800	3,000	3,200
Dark green veg.	1 c/wk	1.5 c/wk	1.5 c/wk	2 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk
Orange veg.	.5 c/wk	1 c/wk	1 c/wk	1.5 c/wk	2 c/wk	2 c/wk	2 c/wk	2 c/wk	2.5 c/wk	2.5 c/wk	2.5 c/wk	2.5 c/wk
Legumes	.5 c/wk	1 c/wk	1 c/wk	2.5 c/wk	3 c/wk	3 c/wk	3 c/wk	3 c/wk	3.5 c/wk	3.5 c/wk	3.5 c/wk	3.5 c/wk
Starchy veg.	1.5 c/wk	2.5 c/wk	2.5 c/wk	2.5 c/wk	3 c/wk	3 c/wk	6 c/wk	6 c/wk	7 c/wk	7 c/wk	9 c/wk	9 c/wk
Other veg.	3.5 c/wk	4.5 c/wk	4.5 c/wk	5.5 c/wk	6.5 c/wk	6.5 c/wk	7 c/wk	7 c/wk	8.5 c/wk	8.5 c/wk	10 c/wk	10 c/wk

4 Grains Group includes all foods made from wheat, rice, oats, cornmeal, barley, such as bread, pasta, oatmeal, breakfast cereals, tortillas, and grits. In general, 1 slice of bread, 1 cup of ready-to-eat cereal, or 1/2 cup of cooked rice, pasta, or cooked cereal can be considered as 1 ounce equivalent from the grains group. **At least half of all grains consumed should be whole grains.**

5 Meat & Beans Group in general, 1 ounce of lean meat, poultry, or fish, 1 egg, 1 Tbsp. peanut butter, 1/4 cup cooked dry beans, or 1/2 ounce of nuts or seeds can be considered as 1 ounce equivalent from the meat and beans group.

6 Milk Group includes all fluid milk products and foods made from milk that retain their calcium content, such as yogurt and cheese. Foods made from milk that have little to no calcium, such as cream cheese, cream, and butter, are not part of the group. Most milk group choices should be fat-free or low-fat. In general, 1 cup of milk or yogurt, 1 1/2 ounces of natural cheese, or 2 ounces of processed cheese can be considered as 1 cup from the milk group.

7 Oils include fats from many different plants and from fish that are liquid at room temperature, such as canola, corn, olive, soybean, and sunflower oil. Some foods are naturally high in oils, like nuts, olives, some fish, and avocados. Foods that are mainly oil include mayonnaise, certain salad dressings, and soft margarine.

8 Discretionary Calorie Allowance is the remaining amount of calories in a food intake pattern after accounting for the calories needed for all food groups—using forms of foods that are fat-free or low-fat and with no added sugars.

Estimated Daily Calorie Needs

To determine which food intake pattern to use for an individual, the following chart gives an estimate of individual calorie needs. The calorie range for each age/sex group is based on physical activity level, from sedentary to active.

Calorie Range		
Children	Sedentary	Active
2–3 years	1,000	1,400
Females		
4–8 years	1,200	1,800
9–13	1,600	2,200
14–18	1,800	2,400
19–30	2,000	2,400
31–50	1,800	2,200
51+	1,600	2,200
Males		
4–8 years	1,400	2,000
9–13	1,800	2,600
14–18	2,200	3,200
19–30	2,400	3,000
31–50	2,200	3,000
51+	2,000	2,800

Sedentary means a lifestyle that includes only the light physical activity associated with typical day-to-day life.

Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3 to 4 miles per hour, in addition to the light physical activity associated with typical day-to-day life.

U.S. Department of Agriculture
Center for Nutrition Policy and Promotion
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IX. RECOMMENDED CHILDHOOD AND ADOLESCENT IMMUNIZATION SCHEDULE

Vaccine ▼	Age ►	Birth	1 month	2 months	4 months	6 months	12 months	15 months	18 months	24 months	4–6 years	11–12 years	13–14 years	15 years	16–18 years
Hepatitis B ¹	HepB	HepB	HepB ¹			HepB						HepB Series			
Diphtheria, Tetanus, Pertussis ¹			DTaP	DTaP	DTaP			DTaP			DTaP	Tdap			Tdap
Haemophilus influenzae type b ¹			Hib	Hib	Hib ²	Hib									
Inactivated Poliovirus			IPV	IPV			IPV				IPV				
Measles, Mumps, Rubella ¹						MMR					MMR			MMR	
Varicella ³						Varicella						Varicella			
Meningococcal ⁴												MCV4		MCV4	MCV4
Pneumococcal ⁵			PCV	PCV	PCV	PCV				PCV		PPV			
Influenza ⁶							Influenza (Yearly)					Influenza (Yearly)			
Hepatitis A ⁷												HepA Series			

This schedule indicates the recommended ages for routine administration of currently licensed childhood vaccines, as of December 1, 2005, for children through age 18 years. Any dose not administered at the recommended age should be administered at any subsequent visit when indicated and feasible. ■■■ Indicates age groups that warrant special effort to administer those vaccines not previously administered. Additional vaccines may be licensed and recommended during the year. Licensed combination vaccines may be used whenever

any components of the combination are indicated and other components of the vaccine are not contraindicated and if approved by the Food and Drug Administration for that dose of the series. Providers should consult the respective ACIP statement for detailed recommendations. Clinically significant adverse events that follow immunization should be reported to the Vaccine Adverse Event Reporting System (VAERS). Guidance about how to obtain and complete a VAERS form is available at www.vaers.hhs.gov or by telephone, 800-822-7967.

Range of recommended ages

catch-up immunization

■ 11–12 year old assessment

- 1. Hepatitis B vaccine (HepB). AT BIRTH:** All newborns should receive monovalent HepB soon after birth and before hospital discharge. Infants born to mothers who are HBsAg-positive should receive HepB and 0.5 mL of hepatitis B immune globulin (HBIG) within 12 hours of birth. Infants born to mothers whose HBsAg status is unknown should receive HepB within 12 hours of birth. The mother should have blood drawn as soon as possible to determine her HBsAg status; if HBsAg is positive, the infant should receive HepB as soon as possible (no later than age 1 week). **For infants born to HBsAg-negative mothers,** the birth dose can be delayed until age 1 month but still if a physician orders to withhold the vaccine and a copy of the mother's original HBsAg-negative laboratory report are documented in the infant's medical record. **FOLLOWING THE BIRTHDOSE:** The HepB series should be completed with either monovalent HepB or a combination vaccine containing HepB. The second dose should be administered at age 1–2 months. The final dose should be administered at age ≥24 weeks. It is permissible to administer 4 doses of HepB (e.g., when combination vaccines are given after the birth dose); however, if monovalent HepB is used, a dose at age 4 months is not needed. **Infants born to HBsAg-positive mothers** should be tested for HBsAg and antibody to HBsAg after completion of the HepB series, at age 9–18 months (generally at the next well-child visit after completion of the vaccine series).

2. Diphtheria and tetanus toxoids and acellular pertussis vaccine (DTaP). The fourth dose of DTaP may be administered as early as age 12 months, provided 6 months have elapsed since the third dose and the child is unlikely to return at age 15–18 months. The final dose in the series should be given at age ≥4 years.

Tetanus and diphtheria toxoids and acellular pertussis vaccine (Tdap – adolescent preparation) is recommended at age 11–12 years for those who have completed the recommended childhood DTaP/DTaP vaccination series and have not received a Td booster dose. Adolescents 10–18 years who missed the 11–12 year Td/tdap booster dose should also receive a single dose of Tdap if they have completed the recommended childhood DTaP/DTaP vaccination series. Subsequent **tetanus and diphtheria toxoids (Td)** are recommended every 10 years.

3. Haemophilus influenzae type b conjugate vaccine (Hib). Three Hib conjugate vaccines are licensed for infant use. If PRP-DOMP (PedvaxHIB® or ComVax® [Merck]) is administered at ages 2 and 4 months, a dose at age 5 months is not required. DTaP/Hib combination products should not be used for primary immunization in infants at ages 2, 4 or 6 months but can be used as boosters after any Hib vaccine. The final dose in the series should be administered at age ≥12 months.

4. Measles, mumps, and rubella vaccine (MMR). The second dose of MMR is recommended routinely at age 4–6 years but may be administered during any visit, provided at least 4 weeks have elapsed since the first dose and both doses are administered beginning at or after age 12 months. Those who have not previously received the second dose should complete the schedule by age 11–12 years.

5. Varicella vaccine. Varicella vaccine is recommended at any visit or after age 12 months for susceptible children (i.e., those who lack a reliable history of chickenpox). Susceptible persons aged ≥13 years should receive 2 doses administered at least 4 weeks apart.

6. Meningococcal vaccine (MCV4). Meningococcal conjugate vaccine (MCV4) should be given to all children at the 11–12 year old visit as well as to unvaccinated adolescents at school entry (15 years of age). Other adolescents who wish to decrease their risk for meningococcal disease may also be vaccinated. All college freshmen living in dormitories should also be vaccinated, preferably with MCV4, although **meningococcal polysaccharide vaccine (MPSV4)** is an acceptable alternative. Vaccination against invasive meningococcal disease is recommended for children and adolescents aged ≥2 years with terminal complement deficiencies or anatomic or functional asplenia and certain other high risk groups (see MMWR 2005;54: RR-7[1-2]; use MPSV4 for children aged 2–10 years and MCV4 for older children, although MPSV4 is an acceptable alternative).

7. Pneumococcal vaccine. The heptavalent **pneumococcal conjugate vaccine (PCV)** is recommended for all children aged 2–23 months and for certain children aged 24–59 months. The final dose in the series should be given at age ≥12 months. **Pneumococcal polysaccharide vaccine (PPV)** is recommended in addition to PCV for certain high-risk groups. See MMWR 2009; 49(RR-9):1–35.

8. Influenza vaccine. Influenza vaccine is recommended annually for children aged ≥6 months with certain risk factors (including, but not limited to, asthma, cardiac disease, sickle cell disease, human immunodeficiency virus [HIV], diabetes, and other conditions that can compromise respiratory function or handling of respiratory secretions that can increase the risk for aspiration), healthcare workers, and household persons (including household members) in close contact with persons at high risk (see MMWR 2005;54(RR-9):1-5). In addition, healthy children aged 6–23 months and close contacts of healthy children aged 0–5 months are recommended to receive influenza vaccine because children in this age group are at substantially increased risk for influenza-related hospitalizations. For healthy persons aged 5–49 years, the intranasally administered, live attenuated influenza vaccine (LAIV) is an acceptable alternative to the intramuscular trivalent inactivated influenza vaccine (TIV). See MMWR 2005;54(RR-8):1–55. Children receiving TIV should be administered a dosage appropriate for their age (0.25 mL if aged 6–35 months or 0.5 mL if aged ≥3 years). Children aged <8 years who are receiving influenza vaccine for the first time should receive 2 doses (separated by at least 4 weeks for TIV and at least 6 weeks for LAIV).

9. Hepatitis A vaccine (HepA). HepA is recommended for all children at 1 year of age (i.e., 12–23 months). The 2 doses in the series should be administered at least 6 months apart. States, counties, and communities with existing HepA vaccination programs for children 2–18 years of age are encouraged to maintain these programs. In these areas, new efforts focused on routine vaccination of 1-year-old children should enhance, not replace, ongoing programs directed at a broader population of children. HepA is also recommended for certain high risk groups (see MMWR 1999; 48: RR-12[1-27]).

The Childhood and Adolescent Immunization Schedule is approved by

The Childhood and Adolescent Immunization Schedule is approved by:
Advisory Committee on Immunization Practices www.cdc.gov/nip/acip • American Academy of Pediatrics www.aap.org • American Academy of Family Physicians www.aafp.org

X. SPEECH AND LANGUAGE MILESTONES

Hearing and Understanding	Talking
Birth-3 Months <ul style="list-style-type: none"> Startles to loud sounds. Quiets or smiles when spoken to. Seems to recognize your voice and quiets if crying. Increases or decreases sucking behavior in response to sound. 	Birth-3 Months <ul style="list-style-type: none"> Makes pleasure sounds (cooing, gooing). Cries differently for different needs. Smiles when sees you.
4-6 Months <ul style="list-style-type: none"> Moves eyes in direction of sounds. Responds to changes in tone of your voice. Notices toys that make sounds. Pays attention to music. 	4-6 Months <ul style="list-style-type: none"> Babbling sounds more speech-like with many different sounds, including <i>p</i>, <i>b</i>, and <i>m</i>. Vocalizes excitement and displeasure. Makes gurgling sounds when left alone and when playing with you.
7 Months-1 Year <ul style="list-style-type: none"> Enjoys games like peek-a-boo and pat-a-cake. Turns and looks in direction of sounds. Listens when spoken to. Recognizes words for common items like "cup", "shoe", "juice". Begins to respond to requests ("Come here", "Want more?"). 	7 Months-1 Year <ul style="list-style-type: none"> Babbling has both long and short groups of sounds such as "tata upup bibibibi." Uses speech or non-crying sounds to get and keep attention. Imitates different speech sounds. Has 1 or 2 words (bye-bye, dada, mama) although they may not be clear.
1-2 Years <ul style="list-style-type: none"> Points to a few body parts when asked. Follows simple commands and understands simple questions ("Roll the ball", "Kiss the baby", "Where's your shoe?"). Listens to simple stories, songs, and rhymes. Points to pictures in a book when named. 	1-2 Years <ul style="list-style-type: none"> Says more words every month. Uses some 1-2 word questions ("Where kitty?", "Go bye-bye?" "What's that?"). Puts 2 words together ("more cookie", "no juice", "mommy book"). Uses many different consonant sounds of the beginning of words.
2-3 Years <ul style="list-style-type: none"> Understands differences in meaning ("go-stop", "in-on", "big-little", "up-down"). Follows two requests ("Get the book and put it on the table."). 	2-3 Years <ul style="list-style-type: none"> Has a word for almost everything. Uses 2-3-word "sentences" to talk about and ask for things. Speech is understood by familiar listeners most of the time. Often asks for or directs attention to objects by naming them.
3-4 Years <ul style="list-style-type: none"> Hears you when call from another room. Hears television or radio at the same loudness level as other family members. Understands simple, "who?", "what?", "where?", "why?" questions. 	3-4 Years <ul style="list-style-type: none"> Talks about activities at school or at friends' homes. People outside family usually understand child's speech. Uses a lot of sentences that have 4 or more words. Usually talks easily without repeating syllables or words.
4-5 Years <ul style="list-style-type: none"> Pays attention to a short story and answers simple questions about it. Hears and understands most of what is said at home and in school. 	4-5 years <ul style="list-style-type: none"> Voice sounds clear like other children's. Uses sentences that give lots of details (e.g. "I like to read my books"). Tells stories that stick to topic. Communicates easily with other children and adults. Says most sounds correctly except a few like <i>l</i>, <i>s</i>, <i>r</i>, <i>v</i>, <i>z</i>, <i>ch</i>, <i>sh</i>, <i>th</i>. Uses the same grammar as the rest of the family.

XI. RECORD TRANSFER



To: _____

_____Re: Patient: _____ DOB: _____ Male Female

Parent/Legal guardian: _____

Special health care needs: No Yes _____

First encounter: _____ Chief complaint: _____

Last examination: _____ Planned treatment: Completed Deferred OngoingOral hygiene: Excellent Good Fair Poor Non-existentCaries history: None Low Moderate High

Remarkable clinical findings:

- Developmental anomalies
- Fluorosis
- Nonnutritive habits
- Malocclusion
- Traumatic injury
- Other _____

Radiographic history/date:

- Bitewings _____
- Panoramic _____
- Full mouth _____
- Single tooth _____
- Cephalogram _____
- Other _____

Comments _____

Professional preventive care:

- Fluoride (last tx _____)
- Sealants _____
- Prescription fluoride/chlorhexidine
- Dietary counseling

Management of developing occlusion:

- Monitored eruption/growth
- Appliances _____
- Retention _____
- Treatment completed _____

Comments _____

Behavior: Cooperative Previous difficulties Ongoing considerationsAdjunctive techniques: Nitrous Sedation GA Other _____Referral for specialty care: No Yes _____

Additional considerations: _____

Patient due for recall: _____

For additional information, please contact (_____) _____.

Signature of person completing form

Signature of attending dentist

XII. COMMON LABORATORY VALUES

CBC			
Test	Normal value	Function	Significance
Hemoglobin	12-18 g/100 mL	Measures oxygen carrying capacity of blood	Low: hemorrhage, anemia High: polycythemia
Hematocrit	35%-50%	Measures relative volume of cells and plasma in blood	Low: hemorrhage, anemia High: polycythemia, dehydration
Red blood cell	4-6 million/mm ³	Measures oxygen-carrying capacity of blood	Low: hemorrhage, anemia High: polycythemia, heart disease, pulmonary disease
White blood cell		Measures host defense against inflammatory agents	
Infant	8,000-15,000/mm ³		Low: aplastic anemia, drug toxicity, specific infections
4-7 y	4-7 y	6,000-15,000/mm ³	High: inflammation, trauma, toxicity, leukemia
8-18 y	4,500-13,500/mm ³		
Differential Count			
Test	Normal value	Significance	
Neutrophils	54%-62%	Increase in bacterial infections, hemorrhage, diabetic acidosis	
Lymphocytes	25%-30%	Viral and bacterial infection, acute and chronic lymphocytic leukemia, antigen reaction	
Eosinophils	1%-3%	Increase in parasitic and allergic conditions, blood dyscrasias, pernicious anemia	
Basophils	1%	Increase in types of blood dyscrasias	
Monocytes	0%-9%	Hodgkin's disease, lipid storage disease, recovery from severe infections, monocytic leukemia	
Absolute Neutrophil Count (ANC)			
Calculation		Normal value	Significance
(% Polymorphonuclear Leukocytes + % Bands) × Total White Cell Count 100		>1500	<1000 Patient at increased risk for infection; defer elective dental care
Bleeding Screen			
Test	Normal value	Function	Significance
Prothrombin time	1-18 sec	Measures extrinsic clotting factors	Prolonged in liver disease, impaired Vitamin K production, surgical trauma with blood loss
Partial thromboplastin time	By laboratory control	Measures intrinsic clotting of blood, congenital clotting disorders	Prolonged in hemophilia A,B, and C and Von Willebrand's disease
Platelets	140,000-340,000/mL	Measures clotting potential	Increased in polycythemia, leukemia, severe hemorrhage; decreased in thrombocytopenia purpura
Bleeding time	1-6 min	Measures quality of platelets	Prolonged in thrombocytopenia
International Normalized Ratio (INR)	Without anticoagulant therapy: 1 Anticoagulant therapy target range: 2-3	Measures extrinsic clotting function	Increased with anticoagulant therapy
Urinalysis			
Test	Normal value	Function	Significance
Volume	1,000-2,000 mL/d		Increase in diabetes mellitus, chronic nephritis
Specific gravity	1.015-1.025	Measures the degree of tubular reabsorption and dehydration	Increase in diabetes mellitus; decrease in acute nephritis, diabetes insipidus, aldosteronism
pH	6-8	Reflects acidosis and alkalosis	Acidic: diabetes, acidosis, prolonged fever Alkaline: urinary tract infection, alkalosis
Casts	1-2 per high power field		Renal tubule degeneration occurring in cardiac failure, pregnancy, and hemoglobinuric-nephrosis
Electrolytes			
Test	Normal value	Function	Significance
Sodium (Na)	135-147 mEq	Reflects acid-base balance	Increase in Cushing's syndrome
Potassium (K)	3.5-5 mEq		Increase in tissue breakdown
Bicarbonate (HCO ₃)	24-30 mEq		
Chloride (Cl)	100-106 mEq		Increase in renal disease and hypertension

XIII. COMMON PEDIATRIC MEDICATIONS

Antibiotics*	Analgesics*
Penicillin How supplied: 125 or 250 mg/5mL or 250 mg tablets Dosage: Children<12=25-50 mg/kg/d in 3-4 divided doses; max=3 g/d Children>12 and adults=1-2 g/d in 3-4 divided doses Sig: Take _____ tsp/tablet q6h for 10 d	How supplied: 20 mg/mL (120 mL) or 200, 400 mg tablets Dosage: Children <12=20 mg/kg/d in 3 divided doses Children>12 and adults=400-800 mg/d in 3 divided doses; max=1.2 g/d Sig: Take _____ tsp/tablets q8h prn pain
Amoxicillin How supplied: 125 or 250 mg/5mL or 125 or 250 chewable tablets Dosage: Children<12=20-40 mg/kg/d in 3 divided doses Children>12 and adult=250-500 mg 3 times/d; max=2-3 g/d Sig: Take _____ tsp/tablet q8h for 10 d	How supplied: drops=100 mg/mL (15 mL) or 80 mg/0.8 mL (15 mL); elixir=32 mg/mL (120 mL) Tablets=325 mg or 80 mg chewable Dosage: Children<12=65 mg/kg/d in 6 divided doses Children>12 and adults=325-650 mg/d in 6 divided doses or 1,000 mg/d in 3 or 4 divided doses; max=4 g/d Sig: Take _____ tsp/drops/tablets q4h prn pain
Clindamycin How supplied: 75 mg/5 mL or 150, 300, 450, 600, 750, 900 mg tablets Dosage: Children<12=10-25 mg/kg/d in 3 divided doses Children>12 and adults=600-1,800 mg/d in 3 divided doses; max=4-8 g/d Sig: Take _____ tsp/tablet q8h for 10 d	How supplied: elixir=120 mg acetaminophen and 12 mg codeine/5 mL or No. 2=300 mg acetaminophen and 15 mg codeine; No. 3=300 mg acetaminophen and 30 mg codeine; No. 4=300 mg acetaminophen and 60 mg codeine Dosage: Children 3-6 y=5 mL 4 times/day Children 7-12=10 mL 4 times/d Adults=15 mL or 1 tablet No. 2 or No. 3 4 times/day Sig: Take _____ tsp/tablets q6h prn pain
Cephalexin How supplied: 125 or 250 mg/5 mL Dosage: 25-50 mg/kg/d in 4 divided doses; max=4 g/d Sig: Take _____ tsp q6h for 10 d	
Augmentin How supplied: 125 or 250/5 mL or 125 or 250 mg chewable tablets Doses: 20-40 mg/kg/d in 3 divided doses; max=2 g/d Sig: Take _____ tsp/tablet q8h for 10 d	
Ibuprofen	

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XIV. MANAGEMENT OF MEDICAL EMERGENCIES

For all emergencies <ol style="list-style-type: none"> 1. Discontinue dental treatment 2. Call for assistance/someone to bring oxygen and emergency kit 3. Position patient; ensure open and unobstructed airway 4. Monitor vital signs 5. Be prepared to support respiration, support circulation, call for additional help 				
Condition	Signs and symptoms	Treatment	Drug dosage	Drug delivery
Allergic reaction (mild or delayed)	Hives; itching; edema; erythema—skin, mucosa, conjunctiva	<ol style="list-style-type: none"> 1. Discontinue all sources of allergy-causing substances 2. Administer diphenhydramine 	Diphenhydramine 1 mg/kg Child: 10-25 mg qid Adult: 25-50 mg qid	Oral
Allergic reaction (sudden onset): anaphylaxis	Urticaria—itching, flushing, hives; rhinitis; wheezing/difficulty breathing; bronchospasm; laryngeal edema; weak pulse; marked fall in blood pressure; loss of consciousness	<p>This is a true, life-threatening emergency</p> <ol style="list-style-type: none"> 1. Call for medical help 2. Administer epinephrine 3. Administer oxygen 4. Monitor vital signs 	Epinephrine 1:1000 0.01 mg/kg every 5 min until recovery or until help arrives	IM or SubQ
Acute asthmatic attack	Shortness of breath; wheezing; coughing; tightness in chest; cyanosis; tachycardia	<ol style="list-style-type: none"> 1. Sit patient upright or in a comfortable position 2. Administer oxygen 3. Administer bronchodilator 4. If bronchodilator is ineffective, administer epinephrine 	<ol style="list-style-type: none"> 1. Try patient's inhaler or one from emergency kit 2. Epinephrine 1:1000 0.01 mg/kg every 15 min as needed 	Inhale IM or SubQ
Anesthetic toxicity	Light-headedness; changes in vision and/or speech; changes in mental status—confusion; agitation; tinnitus; tremor; seizure; tachypnea; bradycardia; unconsciousness; cardiac arrest	<ol style="list-style-type: none"> 1. Assess and support airway, breathing, and circulation 2. Administer oxygen 3. Monitor vital signs 4. Transport to emergency center as indicated 	Supplemental oxygen	Mask
Anesthetic reaction: vasoconstrictor	Anxiety; tachycardia/palpitations; restlessness; headache; tachypnea; chest pain; cardiac arrest	<ol style="list-style-type: none"> 1. Reassure patient 2. Assess and support airway, breathing, and circulation 3. Administer oxygen 4. Monitor vital signs 5. Transport to emergency center as indicated 	Supplemental oxygen	Mask
Overdose: benzodiazepine	Somnolence; confusion; diminished reflexes; respiratory depression; apnea; respiratory arrest; cardiac arrest	<ol style="list-style-type: none"> 1. Assess and support airway, breathing, and circulation 2. Administer oxygen 3. Monitor vital signs 4. Establish IV access and reverse with flumazenil 5. Monitor recovery 	Flumazenil 0.01 mg/kg (not to exceed a total of 1 mg) at a rate not to exceed 0.2 mg/min	IV
Overdose: narcotic 2-3 min)	Decreased responsiveness; respiratory depression; respiratory or SubQ arrest; cardiac arrest	<ol style="list-style-type: none"> 1. Assess and support airway, breathing, and circulation 2. Administer oxygen 3. Monitor vital signs 4. Reverse with naloxone 5. Monitor recovery 	Naloxone 0.01 mg/kg	IV, IM, (may repeat after
Seizure	Warning aura—disorientation, blinking, or blank stare; uncontrolled muscle movements; muscle rigidity; unconsciousness; postictal phase—sleepiness, confusion, amnesia, slow recovery	<ol style="list-style-type: none"> 1. Recline and position to prevent injury 2. Ensure open airway and adequate ventilation 3. Monitor vital signs 4. If status is epilepticus, give diazepam 	Diazepam Child up to 5 y: 0.2-0.5 mg slowly every 2-5 min with maximum=5 mg Child 5 y and up: 1 mg every 2-5 min with maximum=10 mg	IV
Syncope (fainting)	Feeling of warmth; skin pale and moist; pulse rapid initially then gets slow and weak; dizziness; hypotension; cold extremities;	<ol style="list-style-type: none"> 1. Recline, feet up 2. Loosen clothing that may be binding 3. Ammonia inhaler 	Ammonia in vials	Inhale

XV. CARDIOPULMONARY RESUSCITATION

Comparison of Age Groups

Maneuver	Adult Lay rescuer: ≥ 8 years HCP: Adolescent and older	Child Lay rescuers: 1 to 8 years HCP: 1 year to adolescent	Infant Under 1 year of age
Airway		Head tilt–chin lift (HCP: suspected trauma, use jaw thrust)	
Breathing Initial	2 breaths at 1 second/breath	2 effective breaths at 1 second/breath	
HCP: Rescue breathing without chest compressions	10 to 12 breaths/min (approximate)	12 to 20 breaths/min (approximate)	
HCP: Rescue breaths for CPR with advanced airway		8 to 10 breaths/min (approximately)	
Foreign-body airway obstruction		Abdominal thrusts	Back slaps and chest thrusts
Circulation HCP: Pulse check (≤ 10 sec)		Carotid	Brachial or femoral
Compression landmarks		Lower half of sternum, between nipples	Just below nipple line (lower half of sternum)
Compression method Push hard and fast Allow complete recoil	Heel of one hand, other hand on top	Heel of one hand or as for adults	2 or 3 fingers HCP (2 rescuers): 2 thumb–encircling hands
Compression depth	1½ to 2 inches	Approximately one third to one half the depth of the chest	
Compression rate		Approximately 100/min	
Compression-ventilation ratio	30:2 (one or two rescuers)	30:2 (single rescuer) HCP: 15:2 (2 rescuers)	
Defibrillation AED	Use adult pads Do not use child pads	Use AED after 5 cycles of CPR (out of hospital). Use pediatric system for child 1 to 8 years if available HCP: For sudden collapse (out of hospital) or in-hospital arrest use AED as soon as available.	No recommendation for infants <1 year of age

Note: Maneuvers used by only Healthcare Providers are indicated by "HCP."

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