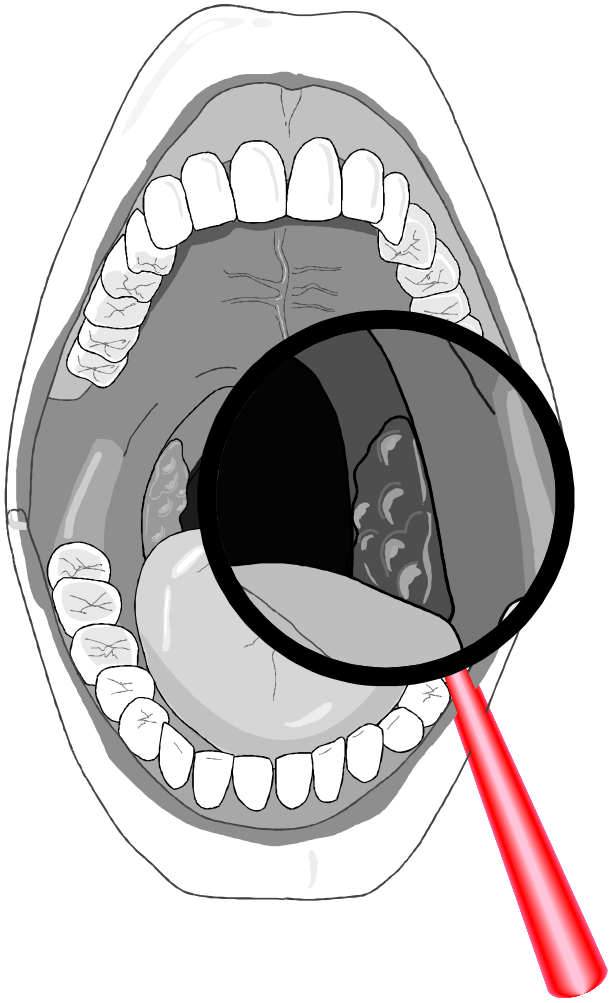
DEN 222

General & Oral Pathology



Summer Semester

2017

General and Oral Pathology Syllabus

Department: Dental Hygiene
Division: Health Sciences

Course Information

Course Number: DEN 222
Credits: 2
Pre-requisites: Enrollment in the Dental Hygiene Program

Co-requisites: None

Description: This course provides a general knowledge of oral pathological manifestations associated with selected systemic and oral diseases. Topics include developmental and degenerative diseases, selected microbial diseases, specific and nonspecific immune and inflammatory responses with emphasis on recognizing abnormalities. Upon completion, students should be able to differentiate between normal and abnormal tissues and refer unusual findings to the dentist for diagnosis.

Textbooks: Langlais, Robert P. and Miller, Craig S.
Color Atlas of Common Oral Diseases
Lea & Febiger. Fifth Edition

Supplies: Course syllabus

Instructor Information

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Office Information
Location: 119
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Office Hours: Mon. 7:45-10:00; Wed 9:45-12:15

Class Information

Beginning Date: 5/17/17 **Census Date:** 5/22/17 **Withdrawal Date:** 7/6/17
Meeting Times: Mon. 8 – 9:30 AM, Wed. 10 – 11:15 AM **Meeting Locations:** DS-213
Number of Weeks: 10 **Final Exam Date:** 7/26/17

Census Date Policy
If a student does not meet the Census Date criteria, he/she is reported as a **No Show** for the course. For a **face-to-face course or a hybrid course**, a student **must attend** one or more class sessions **prior to or on the 10% point (Census Date)** in the class, which is noted in the Instructor Information section of the course syllabus.

Withdrawal Policy
It is the student's responsibility to withdraw from a class by the course withdrawal date that is noted in the Instructor Information section of the course syllabus. Failure to follow the withdrawal procedure will result in a grade of "F" for the course.

Student Learning Outcomes
At the completion of the course, the students should be able to do the following:

1. Demonstrate the correct usage of common descriptive terminology used in oral pathology when given a simulated patient case.
2. Distinguish between normal and abnormal oral findings when giving simulated patient cases.
3. Specify the etiology associated with various disease processes when given a list of known etiologies.
4. Differentiate between oral manifestations associated with various disease processes when giving simulated patient cases.
5. Classify pathological conditions or lesions when given a list of clinical and /or radiographic findings.

Grading Policy

Grading Scale

GRADE	REQUIREMENT
A	**90-100**
B	**80-89**
C	**70-79**
D	**60-69**
F	**50-59**

Being on a 10 point grading scale, final point averages will not be rounded in any manner. If a student has a final grade point average with a .99 or lower, the final course grade will fall into the letter grade in which the .99 or lower falls in. Examples of this would include 89.99 = B, 79.99 = C, 69.99 = D.

Students in limited enrollment health programs will maintain a final grade no lower than "C" in each course with a prefix of BIO, CHM, DEN, EMS, MED, NAS, NUR, OST, PHM, PHY, PTA, RAD, and SUR.

Health program students making a grade of "D" or "F" in any health or health related course will be suspended from the program at the end of the course in which the grade occurs. In addition, health program students who are not eligible to continue at clinical sites may be suspended.

Evaluation of Performance

75%	6 lecture tests are scheduled. Case-based information will be presented on each test.
25%	Comprehensive Final Exam

Policies and Information

ADA Statement
If you have a disability that may affect your academic performance **and** are seeking accommodations, it is your responsibility to inform DisAbility Access Services, Davis Hall room 107, ext. 50157 as soon as possible. It is important to request accommodations early enough to give the disabilities staff adequate time to consider your request and recommend reasonable accommodations. Instructors will provide necessary accommodations based on the recommendations of the disabilities staff.

Title IX
Guilford Technical Community College seeks to provide an environment that is free of bias, discrimination, and harassment. If you have been the victim of sexual harassment/misconduct/assault or discrimination we encourage you to report this to our Title IX Coordinator, Michael Hughes @ 336-334-4822 x 50572, mwhughes2@gtcc.edu.

GTCC faculty are committed to supporting our students and upholding gender equity laws as outlined by Title IX. If you report an incident to a faculty member, she or he must notify the college's Title IX Coordinator about the basic facts of the incident (you may choose whether you or anyone involved is identified by name). The Title IX Coordinator will assist the student in connecting with all possible resources both on and off campus.

Title IX specifically prohibits discrimination against a student based on pregnancy, childbirth, false pregnancy, termination of pregnancy, or recovery from any of these conditions. GTCC will work with students who, as a result of pregnancy or childbirth, require accommodations. Accommodations will be offered for as long as the student's doctor deems the absences medically necessary. Should you experience any of these situations, please notify your instructor or the college Title IX Coordinator, Michael Hughes.

More information about Title IX can be found on our website: www.gtcc.edu.

Online Classroom
This course has an online classroom in *Moodle*. This classroom can be accessed by going to the GTCC Moodle site at online.gtcc.edu. Here you can access course documents, possible assignments and stay in contact with both your instructor and classmates. If you experience difficulty accessing your online classroom, please notify your instructor immediately. Contact Technical Assistance 24 hours a day/7 days a week at 1-866-826-3748.

School Closing Policy
If school is closed, the instructor will communicate the information for makeup of scheduled class time. (Post your process for notifying students here.)

The Center for Academic Engagement
The Center for Academic Engagement (CAE) provides academic and non-academic support to the GTCC community in order to help individuals succeed in education and in life.

Tutoring
Professional tutoring is offered on a walk-in basis for many general education subjects, and peer tutoring is available by request for a variety of career and technical subjects. Access the professional tutoring schedule and the peer tutor request form on the CAE's MyGTCC page by signing in to MyGTCC and clicking on Menu > Academics > Tutoring > Tutoring Home. The schedule is also located in the Tutoring block on the Moodle homepage; click on the "On Campus Tutoring" logo. For questions, email cae@gtcc.edu.

Tutor.com
Tutor.com is a 24/7 professional online tutoring service for a variety of subjects. Students have five hours of on-demand tutoring and document review per semester. To access Tutor.com, go to the Tutoring block on the Moodle homepage, read the Student Academic Integrity policy, and click "I agree."

Student Academic Integrity
Guilford Technical Community College (GTCC) is an academic community with its fundamental purpose being the pursuit of learning and student development. Consistent with this purpose and in order to uphold and support standards of personal honesty and integrity for all members of the college community, it is the policy of GTCC to enforce standards for academic integrity of our programs and courses. Conduct that violates standards of academic honesty and integrity is subject to academic disciplinary action. This conduct may include, but is not limited to, cheating, fabrication and falsification, plagiarism, abuse of academic materials and complicity in academic dishonesty. Any student who violates these standards is subject to academic disciplinary action. Please visit the Student Academic Integrity policy on our website for more information.

If a student violates any aspect of the academic integrity standards, the student will receive a 0 (zero) for the test (online or in-class) and a written warning from the instructor and 15 points will be deducted from the students' FINAL course average / grade. In addition, the student will be reported to the Disciplinary Officer and face dismissal from the program.

Student Conduct Policy
Students may not display conduct on Guilford Technical Community College premises or at GTCC sponsored events that adversely affects the college's educational objectives, is illegal, or is contrary to the rules and regulations of the college. Students who display such conduct shall be subject to disciplinary action under the college's disciplinary policy.

In addition to expectations for student behavior for this course provided above, a list of prohibited behaviors is documented in the College's Student Code of Conduct (http://supportservices.gtcc.edu/student-code-of-conduct/). Included in the Code of Conduct is reference to the authority granted to instructors to remove students from the classroom when the student's behavior becomes a significant disruption to the learning and/or teaching environment.

Class Attendance

Students will get the most benefit from their classes if they attend class regularly and are on time for all classes. Students are responsible for all content covered in a class, whether in attendance or not.

The specific attendance policy for DEN 222 is given below:

Class attendance is ***required*.** A student may be absent from this course a maximum of one class period. Upon reaching the maximum number of allowed absences, the student will be given a letter stating that the maximum number of classes within this course has been reached. The letter will also state that **3 points** will be deducted from the final course grade average for ***each*** additional absence or tardy.

Class tardiness - being late to class is a disruption for other students, as well as the instructor. Three times late to class will equal one absence.

If a class is missed, it will be the responsibility of the student to obtain and learn any material that was missed during the absence. It is not the responsibility of the instructor to take the time to catch the student up on what was missed during the missed class period.

If a student is absent on a scheduled test day, the student must notify the instructor ***PRIOR*** to the start of class for that day. Notification that the student will not be in class to test the scheduled test can be done by phone, email or text, but **must** be done ***PRIOR*** to the start of the test. Failure to notify the instructor ***PRIOR*** to the start of the scheduled class/test will result in the student receiving a **zero (0)** for the missed test. The person reporting the absence **must** either be the student or an immediate family member. If a student is tardy on the day of a test, the student must turn in their test when time is called and will not be given any additional time to complete their test.

Any student that misses a scheduled test will be required to take the test on the first day that they return to school. It is the **responsibility** of the **student** to arrange a time to take the missed test with the instructor. If the student fails to contact the instructor to take the missed test *or* fails to take the test on the first returning day, the student will receive a grade of zero (0) for that test.

If a student is late to class on a scheduled test day, the student will be allowed to take the test, but the student will only be given the time allotted to take the test. Once time is called for all students, the student that was late to class will be required to turn in their test as well. The grade for the test/exam will be based on what was submitted when the test/exam was turned in.

The course tests and final examination are given only during the specified times as noted on the course schedule and will not be given at times other than those as stated. A student may not take a test or final examination earlier than the time scheduled.

Special Note Policy

When a student misses class, the student is responsible for all work missed. Students should see the instructor outside of class for assistance in making up missed work. Asking for help on missed assignments and questions regarding missed work causes interruptions and delays starting class which is unfair to other students. Attention and priority during class with be given to the current day's lesson. See your instructor during his/her office hour or make an appointment with the instructor for assistance.

Withdrawal Policy

It is the student's responsibility to withdraw from a class by the course withdrawal date that is noted in the Instructor Information section of this syllabus. Failure to follow the withdrawal procedure will result in a grade of "F" for the course.

Time Expectation
DEN 222 meets in class for 3 ½ hours each week. A student can expect to spend an additional 3 ½ hours or more to sufficiently learn the material in this course.

Turnaround Policy
In-class tests will be graded no more than one week after the test was taken and the grade will be posted in Moodle. Tests will be available for review on dates that the instructor is available on campus.

Late Work Policy
Assessment within this course is done through in-class tests and a comprehensive final examination. In-class tests and the comprehensive final examination must be taken as noted on the schedule.

Course Expectations
Students are expected to spend adequate time preparing for class by reviewing the required text and supplemental information prior to each session. All exams will be administered on site. Class attendance is necessary for successful completion of this course.

Subject to Change
The course syllabus and class schedule are subject to change as determined by the course instructor. If changes are needed, an addendum to the syllabus and / or class schedule will be provided to each student and implementation of changes will be set forth at date that addendum is issued.

Additional Policies

Dental Department Appearance Guidelines

Classroom Attire – Monday through Thursday: ONLY approved scrubs, or monogrammed polo shirt with approved scrub pants. Closed toe shoes required. Fridays: Street clothes within the following guidelines: no bare midriffs, bare backs, plunging necklines, short skirts or short-shorts.

Cell Phone Policy

Unless working on a specific class project that requires the use of a cell phone, the use of a cell phone during class is **NOT** are permitted.

In order to control the use and access to cell phones during this class, all cell phones are to be placed in a backpack / purse away from student access. In addition, all cell phones are to be turned **off** or placed on **silent**. **NOTE:** vibrate is **NOT** silent.

If a cell phone is found in a student's direct possession (in hands, lap or coat pocket, etc.) during any aspect of this class (unless told that the cell phone is to be used for a specific class project) or should the cell phone ring or vibrate during any part of this class, the owner of the cell phone will lose **3 points** off their **FINAL** course average / grade for ***each*** infraction. If the cell phone rings or vibrates during a test, the student must turn in their test and leave the classroom. The grade on the test will be based on the answers that were completed at the time the test was turned in. In addition, the student will lose **3 points** off their **FINAL** course average / grade for the infraction.

DEN 222
GENERAL AND ORAL PATHOLOGY

2017 CLASS SCHEDULE

	May 17 - Wednesday Lecture: Introduction, Immune Reaction / Inflammation / Repair, Basic Disease Processes
May 22 - Monday Lecture: Preliminary Diagnosis Terminology Oral Path Sheet - Exercise	**May 24 - Wednesday** **Test #1- covers:** Introduction, Immune Reaction/Inflammation / Repair Basic Disease**,** Processes, Preliminary Diagnosis, Terminology Lecture: Disturbances of the Teeth (start) ***Online- Dental Caries / Apical Inflammation***
May 29 - Monday No classes - College Closed Holiday	**May 31 - Wednesday** Lecture: Disturbances of the Teeth (Finish)
June 5 - Monday **Test #2- covers:** Disturbances of the Teeth Dental Caries / Apical Inflammation Lecture: Vesiculo-bullous Lesions (Start)	**June 7 - Wednesday** Lecture: Vesiculo-bullous Lesions (Finish) Ulcerative Lesions (Start)
June 12 - Monday Lecture: Ulcerative Lesions (Finish)	**June 14 - Wednesday** Lecture: Neoplasms (Start)
June 19 - Monday **Test #3- covers:** Vesiculo-Bullous Lesions Ulcerative Lesions Lecture: Neoplasms (Finish)	**June 21 - Wednesday** Lecture: White Lesions (Start)

June 26 - Monday Lecture: White Lesions (Finish) Red-Blue Lesions (Start)	**June 28 - Wednesday** Lecture: Red-Blue Lesions (Finish)
July 3 - Monday **Test #4- covers:** Neoplasms White Lesions Lecture: Verrucal-Papillary Lesions	**July 5 - Wednesday** Lecture: Pigmentations Connective Tissue Lesions
July 10 - Monday Lecture: Misc. Lesions Salivary Gland Lesions	**July 12 - Wednesday** **Test #5- covers:** Red-Blue Lesions Pigmentations Verrucal-Papillary Lesions Lecture: As needed
July 17- Monday Lecture: Cysts	**July 19 - Wednesday** **Test #6- covers:** Salivary Gland Lesions Connective Tissue Lesions Misc. Lesions Cysts
July 24 - Monday Final Exam Review	**July 26 - Wednesday** **Comprehensive Final Examination**

Basic Disease Processes / Inflammation / Repair

Lecture Outline

a. Developmental Disorders
   - characteristics associated with developmental disorders
   - congenital
   - familial
   - hereditary

b. Inflammatory Conditions
   - characteristics associated with inflammatory conditions
   - types of injuries
   - localized vs generalized
   - acute vs chronic
   - cardinal signs of inflammation:
   - systemic inflammation

c. Infectious Diseases
   - characteristics associated with infectious diseases
   - single organism vs. multiple organisms
   - localized or systemic

4. Metabolic Diseases
   - characteristics associated with metabolic diseases
   - local or systemic

5. Neoplastic Diseases
   - characteristics associated with neoplastic diseases
   - types of neoplasms
     - benign neoplasms
       - characteristics
     - malignant neoplasms
       - characteristics
       - carcinoma
       - sarcoma

6. Other Disease Processes
   a. Reactive processes
      - definition
   b. Idiopathic
      - definition

7. Tissue Response to Injury
   - Growth of Tissue
   - Inflammation
   - Healing Methods (repair and regeneration)
   - Immunologic Response

8. Growth of Tissue
   a. Hyperplasia
      - definition
      - etiology
      - clinical examples

b. Hypertrophy
   - definition
   - etiology
   - clinical examples

9. Other terms
   a. Hyperkeratosis
      - definition
      - etiology
      - clinical examples
   b. Hypoplasia
      - definition
      - etiology
      - clinical examples
   c. Atrophy
      - definition
      - etiology
      - clinical examples
   d. Dysplasia
      - definition
      - etiology
10. Repair and Regeneration
   a. Repair
      - definition
      - types of repair:
      1) Healing by Primary Intention
         - associated characteristics
      2) Healing by Secondary Intention
         - associated characteristics
   b. Regeneration
      - definition

Basic Disease Processes

Lecture Objectives:

1. Describe how disease processes are classified with respects to their etiologies.
2. Explain the general characteristics of developmental disorders, inflammatory conditions, infectious diseases, metabolic diseases, and neoplastic diseases.
3. Discuss the term "tumor".
4. Describe the differences in the development of benign and malignant neoplasms.
5. Define the differences between a carcinoma and a sarcoma.
6. Define the following terms: etiology, idiopathic, congenital, familial, genetic, focal, systemic, induration, encapsulation, metastatic and reactive process.
7. Explain the manner by which tissues are able to respond to injury.
8. List differences and similarities between hyperplasia and hypertrophy.

9. Define the following terms: hyperkeratosis, hypoplasia, atrophy, and dysplasia
10. Recall examples for the following terms: hyperkeratosis, hypoplasia, atrophy, and dysplasia

Inflammation / Repair

Lecture Objectives:

1. Define the following terms: inflammation, regeneration, repair
2. List the cardinal signs of inflammation.
3. Discuss why each of the cardinal signs of inflammation will occur.
4. Explain how each of the cardinal signs of inflammation will appear clinically.
5. Discuss the clinical findings associated with a localized inflammatory response.
6. List the clinical and laboratory findings associated with an acute inflammatory response that has become systemic in nature.
7. Compare acute verses chronic inflammatory responses.
8. List examples of regeneration and repair.
9. List the steps that a tissue proceeds through as it undergoes repair.
10. Compare the healing by primary and secondary intention.

BASIC DISEASE PROCESSES

- Based on etiology
  - Developmental disorders
  - Inflammatory conditions
  - Infectious diseases
  - Metabolic diseases
  - Neoplastic diseases
  - Misc. diseases processes

1. Developmental Disorders

- birth defects or abnormalities that occur during growth and development of the fetus
- occur during the 1st trimester
- minor to severe
- various factors responsible for abnormalities
- often congenital
- familial or hereditary (inherited)
- Example: Clefts, tori, ankyloglossia

2. Inflammatory Conditions

- body responds to disease and injury with a process known as inflammation; the inflammatory process will allow the body to eliminate injurious agents
- body will use specific cells to remove the injurious agent(s)
- Injury- changes in the environment that causes tissue damage
  - Injury can be physical, chemical, microbial or nutritional

Inflammation:

- localized / generalized; acute / chronic
- extent and duration of the injury will determine the extent and duration of the inflammatory response

Cardinal Signs of Inflammation:

- when talking about inflammation, we see something called the five cardinal signs. These cardinal signs are found in all cases of inflammation and represent local clinical signs that an injury has occurred.

Clinical Sign	Due To
1. rubor (redness)	vasodilation
2. Calor (heat)	vasodilation
3. Tumor (swelling)	increased vessel permeability
4. Dolar (pain)	swelling and chemical mediators
5. Loss of function	associated with swelling and pain

Inflammation – systemic:

- if acute inflammation becomes more severe, it can become systemic in nature; therefore, systemic signs can present

Systemic findings include the following:

1. Fever
2. Lymphadenopathy
3. Leukocytosis- increased number of WBCs; more are made in attempt to fight off injury
4. Septicemia- bacteria in the blood

3. Infectious Diseases

- infectious diseases occur when there is an invasion by pathologic microorganisms
- can be caused by a single organism vs. multiple organisms
- localized or systemic in nature
- infectious diseases can be localized to the oral cavity (HIV, syphilis, etc…)
- Examples: Syphilis, Periodontal disease

4. Metabolic Diseases

- conditions that are related to body metabolism or process
- can present locally or involves entire body (systemic)
- initially- it may present as a local problem
- most are seen with systemic signs and symptoms

- familial or hereditary
- Example: Diabetes

5. Neoplastic Diseases

- "oma"- tumor
- neoplasm = tumor = cancer
- is a wild overgrowth of cells that arises from an existing tissue, but will grow independently of this tissue and will grow at its' own rate
- grows at the expense of healthy cells / tissue
- irreversible growth
- typically isolated, unilateral

Benign vs. Malignant

<u>Benign Neoplasms</u> – characteristics associated with them include:

⇒ slow growth

⇒ encapsulated

⇒ non-ulcerative

⇒ non-indurated

⇒ displaces structures

⇒ not metastatic

⇒ regular cell arrangement, normal appearance

⇒ not fatal

- Example: Ameloblastoma

Malignant Neoplasms – characteristics associated with them include:

⇒ fast growth rate

⇒ become fixed to tissues

⇒ indurated

⇒ surface ulceration

⇒ invasive and destructive

⇒ can metastasize

⇒ irregular cell arrangement

⇒ cells of varying sizes

- 2 types:
  - carcinoma
  - sarcoma
- Example: Squamous cell carcinoma, Kaposi's sarcoma

6. Other Disease Processes

- some lesions or conditions do not neatly fall into a perfect category. This is where they are placed.

1. Reactive processes
   - those lesions that are created by trauma

   Example: Hematoma

2. Idiopathic
   - those lesions that have an unknown etiology or cause

   Example: Geographic tongue

Tissue Response to Injury

If a tissue is subjected to an injury, the tissue can respond or fix itself by several different methods.

The method that will be used will vary on the type of injury, the extent of the injury and the tissue itself. Tissues can deal with an injury in the following ways:

- Growth of Tissue
- Inflammation
- Healing Methods (repair and regeneration)
- Immunologic Response

Growth of Tissue

- cell proliferation (growth) occurs
- growth of a tissue can occur with or without the presence of inflammation

Methods by which the growth of tissue occurs includes the following processes:

a. Hyperplasia

⇒ an increase in the number of normal cells in a tissue or organ that results in the increase in the size of the tissue or organ

- protective mechanism that the tissue uses to protect itself against chronic trauma being placed on it
- clinical picture: the tissue will appear white or pale in color
- if the cause is removed, the tissue returns back to normal
- Example: Papillary hyperplasia

b. Hypertrophy

- an increase in the size of the cells within the tissue or organ that results in an increase in the size of the tissue or organ
- tissue's response to meet increased need or demands being placed on it
- Example: Muscle cells

Other terms dealing with tissue response to injury, development or change:

Hyperkeratosis

⇒ thickening of the outer layer of the skin; increase in squamous epithelium

⇒ protective mechanism against trauma- protects by changing character of tissue surface

- Example: Callus

Hypoplasia

- failure to develop fully (of an tissue or organ)
- Example: Hypoplastic mandible (Class II)

Atrophy

- opposite of hypertrophy
- defined as an decrease in the size of the cells within the tissue or organ and results in a decrease in the size of the tissue or organ
- Example: Disuse atrophy

Dysplasia

⇒ abnormal growth pattern (nucleus, cell division) within a given tissue

⇒ reflects a change of one cell type to less mature cell type

Repair and Regeneration

- processes represent the body's final defense mechanism to restore injured tissue to its original state
- occurs only if the injury is removed

Repair

- generic term that includes processes in which the body restores / replaces damaged cells by dissimilar cells

Types of Repair:

- Healing by Primary Intention
- Healing by Secondary Intention

Healing by Primary Intention

- refers to healing of an injury that has a little loss of tissue
- clean cuts or incisions; wound edges are close approximated
- little granulation tissue is formed
- minimal scarring will occur

Healing by Secondary Intention

- wound edges not closely approximated
- have a tissue loss
- increased formation of granulation tissue will occur
- increased scar formation
- increased loss of tissue function can be present

Regeneration

- replacement of damaged tissue by cells that are similar or identical to those destroyed
- Examples of tissues that can regenerate:
  - surface epithelium, oral epithelium

Preliminary Diagnosis Of Oral Lesions

Lecture Outline

1. Lesion evaluation and diagnosis
2. Components for diagnosing
    a. Clinical diagnosis
        – components of a clinical evaluation
    b. Radiographic diagnosis
        – components of a radiographic evaluation
        – radiographic terminology
    c. Historic diagnosis
        – components of a historic evaluation
    d. Laboratory diagnosis
        – blood tests, urinalysis and cultures
    e. Microscopic diagnosis
        – main component of definitive diagnosis
        – incisional biopsy
        – excisional biopsy
    f. Surgical diagnosis
    g. Therapeutic diagnosis
3. Differential diagnosis
    – definition
4. Final (Definitive) diagnosis
    – definition

Diagnostic and Descriptive Terminology

Lecture Outline

1. Why use of correct terminology is important
2. Oral pathology terminology
    a. Macule
        – definition
    b. Erosion / ulcer
        – definition
    c. Fissure
        – definition
    d. Papule / nodule
        – definition
    e. Vesicle
        – definition
    f. Bulla
        – definition
    g. Pedunculated
        – definition
    h. Exophytic
        – definition
    i. Sessile
        – definition

j. Indurated
   – definition
k. Fluctuant
   – definition

Lecture Objectives:

1. Select appropriate descriptive terminology for various clinical lesions.
2. List criteria or procedures that are used to aid in the diagnosis of a lesion.
3. List how the clinical diagnosis is used in the evaluation of a given lesion.
4. List how the radiographic diagnosis is used in the evaluation of a given lesion.
5. List how the historic diagnosis is used in the evaluation of a given lesion.
6. List how the laboratory diagnosis is used in the evaluation of a given lesion.
7. List how the microscopic diagnosis is used in the evaluation of a given lesion.
8. List how the surgical diagnosis is used in the evaluation of a given lesion.
9. List how the therapeutic diagnosis is used in the evaluation of a given lesion.
10. Define the term differential diagnosis.
11. List procedures that are used when starting with a differential diagnosis and ending with a final diagnosis.
12. Define the following: macule, erosion, ulcer, fissure, papule, vesicle, bulla, plaque, nodule, tumor, cyst, sessile, peduculated, exophytic, indurated, fluctuant

Preliminary Diagnosis	**Color Atlas**
Descriptive Terminology	18 - 30

PRELIMINARY DIAGNOSIS OF ORAL LESIONS

So, let's say that we are looking at something kinda funky on a patient.  We are thinking to ourselves "I wonder what the heck that thing is?"  To determine what the lesion or condition is may involve the use of several things. So, how we diagnose what something is becomes like working on a puzzle. There are 8 areas that we can use to come up with the diagnosis of the lesion or condition that we are looking at. 

How are lesions diagnosed? The practitioner can use a number of the following areas to evaluate a given condition or lesion:

1. Clinical Diagnosis

- assesses clinical aspects of the lesion- this will involve the clinical appearance of the lesion
  - what the lesion looks like clinically- sessile, pedunculated, etc…
  - consistency (how the lesion feels)- is done by palpation; soft, firm, indurated, etc…
  - color- red, white, blue, etc…
  - size (in mm or cm)
  - texture (how the surface appears)- smooth, fissured, etc…
- some lesions have a distinct clinical appearance

2. Radiographic Diagnosis

- radiographs provide an additional view of the area. They can help to determine whether something is limited to the soft tissues or whether it extends into the underlying bone.
- some lesions can be distinguished by radiographs

Radiographic terminology:

- radiolucent, radiopaque
- unilocular, multilocular

3. Historic Diagnosis

- this can provide a lot of information regarding the lesion or condition. This will include the history of the lesion (how long it has been present, what may have started it, etc…), does anyone else in the family have a similar finding, medical / dental / social history, any personal habits done and any medications taken.

4. Laboratory Diagnosis

- if needed, additional lab tests can be done to add in the diagnosis of a lesion
- lab tests can include blood tests, urinalysis and cultures

5. Microscopic Diagnosis

- the main component of the definitive diagnosis
- a biopsy is the removal of a small amount of the tissue surgically. There are two types of biopsies, an incisional biopsy and an excisional biopsy.
- incisional biopsy- removes a small section of the area being evaluated. It is like removing a piece from a cake.
- excisional biopsy- will remove the entire area being evaluated. This is like taking the entire pie!
- in either case, the area taken during the biopsy will be evaluated microscopically. Everything has a specific appearance microscopically and a microscopic evaluation of the biopsied area will give a very definite result.

6. Surgical Diagnosis

- in some cases, surgery may be needed to help with the diagnosis. This may give the practitioner specifics regarding the lesion that may not have been known otherwise. Some lesions will have a specific appearance surgically. This may be used in conjunction with the microscopic diagnosis.

7. Therapeutic Diagnosis

- the use some type of therapy to make a diagnosis
- some lesions can be distinguished by radiographs

8. Differential Diagnosis

- is when the practitioner comes up with few lesions or conditions that he/she thinks that it could be
- out of the list of suspected lesions / conditions, the practitioner will systematically compare and contrast the suspected lesions

Final (Definitive) Diagnosis

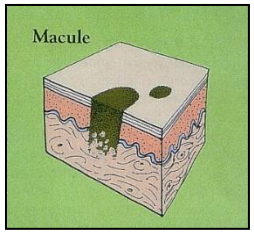
- evaluation of all the suspected lesions ----> narrow down the list to come up with one lesion or condition

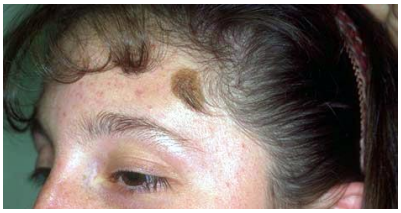
DIAGNOSTIC & DESCRIPTIVE TERMINOLOGY

This section will take a look at some of the terms that are used to describe oral lesions. These terms can be used when writing progress notes, lesion descriptions and referral letters. The object is to use correct terminology, but not to over use them by being too wordy.

Macule

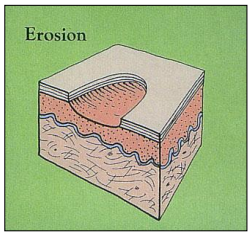
* term used to describe a flattened area that is of a different coloration than that of the surrounding area
* Example: nevi (mole)

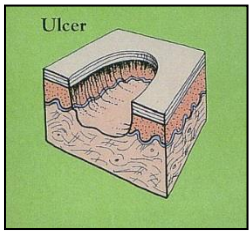


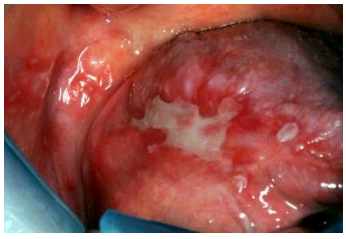


Erosion / Ulcer

* term used to describe an area that exhibits a loss of surface tissue
* depth of the lesion distinguishes between the two terms- erosion is shallow, whereas an ulcer is deeper (extends into the underlying connective tissue)
* Example: traumatize / scrape the surface of a tissue

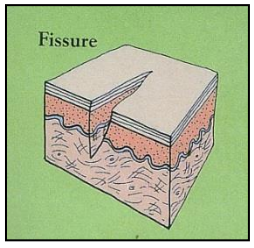


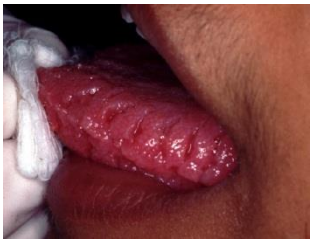




Fissure

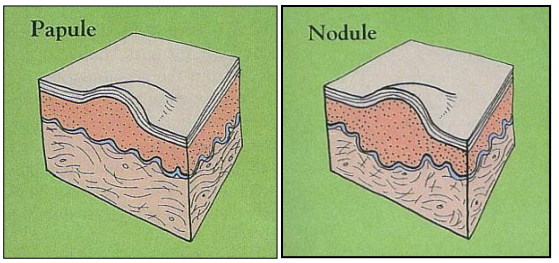
* term used to describe a clefting or cracking within the epidermis
* Example: dry, cracking tissue on the lower lip or corners of the mouth (angular cheilitis)

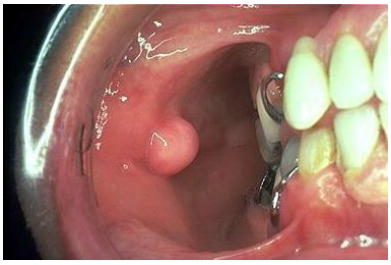




Papule / Nodule

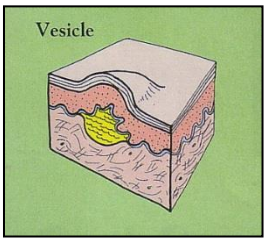
* terms used to describe a solid mass of tissue (lesion)
* difference between the terms is related to depth- a papule is superficial, whereas a nodule is deep
* Example: if you had something like a grape (is the lesion) that is under the tissue surface. The tissue around the grape has attached into the grape. The grape can be felt when you feel the tissue, but the grape does not move much when you are feeling it.

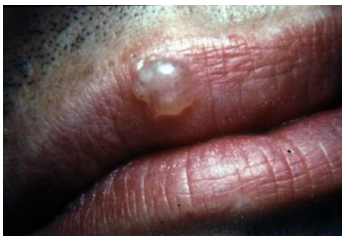




Vesicle

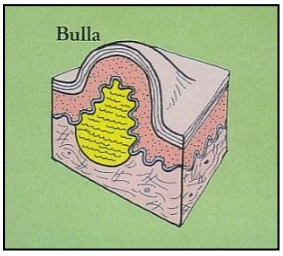
* term used to describe a fluid-filled lesion that is on the tissue surface;
* less than 1 cm in size
* Example: small raised blister

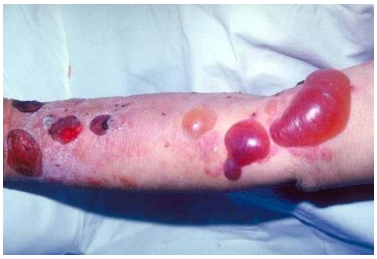




Bulla

* term used to describe a fluid-filled lesion that is on the tissue surface
* is a vesicle that is larger than 1 cm in size
* Example: large blister



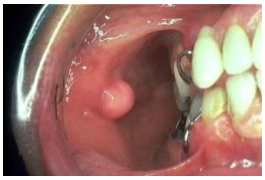


Pedunculated

* term used to describe when a tissue mass grows on a stalk
* Example: lesion that grows like a mushroom or flower on top of the tissue surface

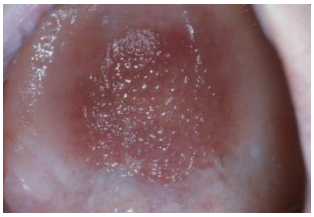
Exophytic

* term used to describe a lesion that projects above the tissue surface
* Example: lesion growing on the top of the tissue and has the appearance of sticking “out”



Sessile

* term used to describe a lesion with a broad base (i.e.- something that is wide)
* Example: lesion that covers the entire hard palate



Indurated

* term used to describe something that is hard when palpated (felt)
* Example: feel or palpate a pebble

Fluctuant

* term used to describe something that is moveable when palpated
* Example: palpate or feel the Pillsbury Dough Boy!

Dental Caries, Pulpitis and Apical Inflammation

Lecture Outline

1. Dental caries
   - definition
   - etiology
   - facts associated with dental caries

   a. Development of dental caries
      - required ingredients

   b. Cariogenic bacteria
      - *Streptococcus mutans*
      - other bacteria involved with the decay process

   c. Locations
      - fissural caries
        - detection techniques
      - smooth surface caries
        - detection techniques

   d. Types of caries
      1) Slowly developing
      2) Rampant caries
      3) Radiation caries
      4) Root caries
      5) Nursing bottle caries
      6) Recurrent decay
      7) Incipient decay
      8) Arrested caries

2. Cavity formation and progression
   a. Formation of incipient lesion
      - characteristics

   b. Untreated incipient lesion
      - characteristics

3. Classic clinical features
   - demineralization
   - color change
   - cavitation
   - enamel softness

4. Classic symptoms associated with caries
5. Pulpitis
   - definition

   a. Reversible pulpitis
      - clinical findings

   b. Irreversible pulpitis
      - clinical findings

6. Pulpal necrosis
   - definition
   - clinical findings

7. Periapical abscess
   - definition
   - clinical and radiographic findings

8. Granuloma / periapical cyst
   - definition
   - clinical and radiographic findings
9. Cellulitis
   - definition
   - clinical findings
   - other findings / terminology
     - sinus tract
     - fistula
     - Ludwig's angina
     - pericoronitis
     - Cavernous sinus thrombosis

Lecture Objectives:

1. Explain the steps involved in the development and progression of dental caries.
2. List characteristics associated with the progression of dental caries.
3. Identify the major bacteria involved in the development of dental caries.
4. List factors that play a role in the development of dental caries.
5. Identify the tooth surfaces that can develop dental caries.
6. Describe the etiology for nursing bottle caries.
7. Discuss how the dentition is involved when affected by nursing bottle caries.
8. Define the following terms: root caries, pulpitis, pulpal necrosis, periapical abscess, periapical cyst, cellulitis, Ludwig's angina, Cavernous sinus thrombosis

Dental Caries	**Color Atlas**
Dental caries	67 - 76
Ludwig's angina	144 - 145
Cavernous sinus thrombosis	144 - 145

Notes on dental caries are presented during the online presentation.

Disturbances of the Teeth

Lecture Outline

1. Gemination
   - etiology
   - clinical findings
2. Fusion
   - etiology
   - clinical findings
   - distinguishing between gemination and fusion
3. Concrescence
   - etiology
   - findings associated with concrescence
4. Dilaceration
   - definition
   - etiology
   - radiographic appearance
5. Dens invaginatus
   - etiology
   - clinical and radiographic appearance
6. Enamel pearls
   - etiology
   - clinical / radiographic findings
7. Attrition
   - definition
   - etiology
   - clinical appearance
8. Abrasion
   - definition
   - etiology
   - clinical appearance
9. Erosion
   - definition
   - etiology
   - clinical appearance
10. Hypodontia
    - definition
    - etiology
11. Anodontia
    - definition
    - etiology
    - hereditary ectodermal dysplasia
12. Supernumerary teeth
    - definition
    - etiology
    - common locations
    - Cleidocranial dysostosis
    - Gardner's syndrome

- mesiodens
- natal teeth

13. Enamel hypoplasia
    - definition
    - etiology
    - clinical appearance
14. Enamel hypocalcification
    - definition
    - clinical appearance
15. Hutchinson’s incisors
    - definition
    - clinical appearance
16. Mulberry molars
    - definition
    - clinical appearance
17. Mottled enamel
    - etiology
    - clinical appearances
    - resistance to decay
18. Amelogenesis imperfecta
    - etiology
    - types of amelogenesis imperfecta
    - clinical and radiographic appearances
19. Dentinogenesis imperfecta
    - etiology
    - clinical and radiographic appearances
20. Pulp calcifications
    - radiographic appearance
21. Internal resorption
    - definition
    - etiology
    - radiographic and clinical appearance
22. External resorption
    - definition
    - etiology
    - radiographic appearance
23. Tetracycline staining
    - etiology
    - clinical appearances

Lecture Objectives:

1. Describe the following developmental entities:
2. List the etiology for the following entities:

   Gemination, fusion, concrescence, dilacerations, dens in dente, enamel pearls, attrition, erosion, erosion, hypodontia, anodontia, ectodermal dysplasia, supernumerary teeth, mesiodens, cleidocranial dysostosis, natal teeth, enamel hypoplasia, enamel hypocalcification, hutchinson's incisors, mulberry molars, mottled enamel, amelogenesis imperfecta, dentinogenesis imperfecta, pulp calcifications, internal resorption, external resorption, tetracycline staining

3. List clinical findings that are associated with the following entities:

   Gemination, fusion, concrescence, dilacerations, dens in dente, enamel pearls, attrition, erosion, erosion, hypodontia, anodontia, ectodermal dysplasia, supernumerary teeth, mesiodens, cleidocranial dysostosis, natal teeth, enamel hypoplasia, enamel hypocalcification, hutchinson's incisors, mulberry molars, mottled enamel, amelogenesis imperfecta, dentinogenesis imperfecta, pulp calcifications, internal resorption, external resorption, tetracycline staining

4. List radiographic findings for the following entities:

   Gemination, fusion, dilacerations, dens in dente, enamel pearls, ectodermal dysplasia, supernumerary teeth, mesiodens, mottled enamel, amelogenesis imperfecta, dentinogenesis imperfecta, pulp calcifications, internal resorption, external resorption, tetracycline staining

5. Compare between gemination and fusion.

Tooth Disturbances	Color Atlas
Gemination	50
Fusion	50
Concrescence	50
Dilaceration	52 - 53, 62 - 63
Dens in Dente	48 - 49
Enamel Pearls	52 - 53
Attrition	60 - 61
Abrasion	60 - 61
Erosion	60 - 61
Hypodontia	42 - 45
Anodontia	42
Ectodermal Dysplasia	44 - 45
Supernumerary Teeth	47, 52 - 53
Mesiodens	46
Cleidocranial Dysostosis	46 - 47
Natal Teeth	34 - 35
Enamel Hypoplasia	54 - 55
Enamel Hypocalcification	-
Hutchinson's Incisors	54
Mulberry Molars	54
Mottled Enamel	54 - 55
Amelogenesis Imperfecta	54 - 55
Dentinogenesis Imperfecta	56 - 57
Pulp Calcifications	62 - 63
Internal Resorption	64 - 65
External Resorption	64 - 65
Tetracycline Staining	58 - 59

DISTURBANCES OF THE TEETH

Gemination

- "twinning"
- developmental disturbance that occurs during tooth formation
- have one tooth attempting to make two teeth; a single tooth germ splits into two
- macrodont
- typically see 2 crowns with shared root
- most commonly involves the mandibular primary incisors

Fusion

- a developmental disturbance that occurs during tooth formation
- have the union or joining of two tooth buds at the dentin
- results in the formation of 1 tooth from 2 separate tooth germs
- macrodont
- root canal can be separate or shared
- most commonly involves the mandibular primary incisors

Gemination vs. Fusion- how to distinguish these two conditions apart:

- count the number of teeth present and in fusion, there will be one tooth missing

Concrescence

- type of fusion that occurs during tooth development
- results in adjacent teeth being joined by cementum
- occurs when teeth develop close to one another, trauma
- most commonly involves the molars

Dilaceration

- term denotes an abnormally curved or angled root
- most likely due to trauma to the tooth germ (Hertwig's) during development

Dens Invaginatus

- "tooth within a tooth" / "dens in dente"
- developmental anomaly in which enamel and dentin of the crown invaginate inward
- clinically, a pit or crevice may be noted on the crown of the tooth

- radiograph will show invagination of the hard tissue
- most commonly involves the MX laterals

Enamel Pearls

- "enameloma"
- reflects an abnormal displacement of some ameloblasts during tooth formation
- results in a small nodule of ectopic enamel that is located on a root surface
- found on roots at the bi- or trifurcation

Attrition

- physiologic wearing of tooth structure
- an age related process that is mastication related
- normal process that can be carried to an extreme
- amount of attrition will be influenced by diet, jaw muscularity and chewing habits

Abrasion

- pathologic wearing of tooth structure that results from a repetitive mechanical habit
- can be due to an abnormal habit or an abnormal use of an abrasive substance
- most commonly noted on exposed root surfaces
- slow process; secondary dentin will be laid down

Erosion

- loss of tooth structure that results from a non-bacterial, chemical process
- can be seen on labial or lingual surfaces of the teeth
- clinically, smooth polished surfaces and / or extruded restorations will be noted

Hypodontia

- refers to the congenital absence of one or a few teeth
- most commonly involves the 3rd molars and the MX lateral incisors

Anodontia

- refers to a rare condition in which no teeth develop
- may be part of a finding in a syndrome called hereditary ectodermal dysplasia

Supernumerary Teeth

- "hyperdontia"
- relates to an overproliferation (over production) of the dental lamina
- can see isolated tooth involvement
- MX incisors, MD premolars
- or can be a part of a syndrome
  - Cleidocranial dysostosis
  - Gardner's syndrome

Mesiodens: supernumerary teeth that present at the MX or MD midline

Natal teeth: supernumerary teeth present at birth

Enamel Hypoplasia

- refers to an incomplete or defective formation of the enamel
- etiology- trauma, diseases (measles, etc...), high fever, excessive fluoride
- results in a reduction in the amount of enamel that is formed
- clinically, pits, grooves and / or missing enamel will be seen
- enamel is of normal hardness, but see defects in the quantity and shape of the enamel

Enamel Hypocalcification

- developmental anomaly that results in defective mineralization of the enamel
- clinically, chalky white spots on the enamel are seen
- tooth has a normal shape and form

Hutchinson's Incisors

- malformed teeth that are caused by the presence of congenital syphilis during tooth development
- type of hypoplasia that produces a screwdriver-shaped incisor (crowns are narrowed at the incisals)

Mulberry Molars

- malformed teeth that are caused by the presence of congenital syphilis during tooth development
- type of hypoplasia that produces a permanent 1^{st} molar with a "raspberry" appearance on the occlusal surface

Mottled Enamel

- "fluorosis"
- enamel hypoplasia that results from the ingestion of high concentrations of fluoride during
- tooth development
- also hypocalcification due to the presence of fluoride- it replaces some of the hydroxy groups found in the enamel
- enamel exhibits mottling (irregular areas of discoloration)
- in mild cases, the teeth will exhibit white flecks / chalky appearance
- in severe cases, the teeth will exhibit brown to black coloration and pitting
- increased amounts of fluoride in the enamel will make it more resistant to decay

Amelogenesis Imperfecta

- "brown teeth"
- refers to a group of inherited conditions that affect the enamel
- unknown etiology
- can involve an isolated tooth or the entire dentition
- histologically, there is normal dentin and pulp
- Radiograph:
  - see decreased enamel thickness with normal dentin and pulp

Various types of Amelogenesis Imperfecta exist:

- Hypoplastic type
- Hypocalcified type
- Hypomaturation type - not commonly noted
- Hypoplastic - hypomaturation type - not commonly noted

Hypoplastic Type:

- most common type form of AI
- enamel does not develop to a normal thickness
- teeth will exhibit a generalized pitted appearance

Hypocalcification Type:

- have a normal quantity of enamel, but the enamel is poorly calcified
- enamel is soft, will wear or fracture and easily stains

Dentinogenesis Imperfecta

- "hereditary opalescent dentin"
- group of inherited conditions that affect the dentin
- more common than Amelogenesis Imperfecta
- affects both primary and permanent dentitions
- creates teeth with bulbous crowns, a constricted CEJ, and short roots with no pulp
- dentin is soft and will wear or fracture
- color variations are possible
  - opalescent brown to brownish blue

Pulp Calcifications

- also called pulp stones or denticles
- small, rounded radioopaque masses (calcifications) that are found within the pulp
- diffuse (dust-like) or nodular (mass)
- asymptomatic; are seen with increasing age

Internal Resorption

- resorption of tooth structure (dentin) from the pulpal side
- unknown cause, but trauma is suspected
- asymptomatic and tend to be found on radiographs
- can result in the formation of a "pink tooth"

External Resorption

- resorption of tooth structure from the outside surface

Causes include:

- exfoliation of primary teeth
- trauma
- chronic inflammation
- cysts
- malignant neoplasms

Tetracycline Staining

- condition in which tetracycline incorporates within the developing dentin and enamel
- teeth become discolored following eruption / exposure to sunlight
- represents an intrinsic / endogenous stain
- generalized discoloration that creates a band-like pattern
- varies in severity and a range of colors are noted- yellow, green, gray-brown

Vesiculo-Bullous Diseases

Lecture Outline

1. Terminology
   - vesicle
   - bulla
   - Nikolsky sign
2. Herpes simplex
   - etiology
   - transmission
   - Type 1 HSV 2 forms
   a. Primary herpetic gingivostomatitis
      - clinical findings
      - complications
      - treatment
   b. Secondary or recurrent HSV infections
      - HSV reactivation
      - precipitating factors
      - clinical findings
      - prodromal symptoms
      - herpes labialis
      - treatment
   c. Herpetic whitlow
3. Varicella - Zoster
   a. Varicella
      - etiology
      - transmission
      - clinical findings
   b. Herpes zoster
      - etiology
      - reactivation
      - clinical findings
      - treatment
4. Measles (rubeola)
   - etiology
   - transmission
   - clinical findings
   - koplik spots
5. Infectious mononucleosis
   - etiology
   - transmission
   - clinical findings
   - oral lesion
   - complications

6. Erythema multiforme
   - etiology
   - clinical findings
   - target lesions
   - Stephens - Johnson syndrome
7. Pemphigus
   a. Pemphigus vulgaris
      - etiology
      - clinical findings
8. Benign mucous membrane pemphigoid
   - etiology
   - clinical findings

Lecture Objectives:

1. Define the following terms: Vesiculo-bullous lesion, Nikolsky sign, vesicle, bulla, prodromal signs.
2. List types of diseases that will exhibit vesicles or bulla as a clinical finding.
3. Illustrate the order of progression for the herpes simplex virus from the initial infection (contact) to its' reoccurrence.
4. List the etiology for the following diseases:
   Herpes simplex (primary herpetic gingivostomatitis, recurrent HSV infections, herpes labialis, etc...), varicella-zoster, measles, infectious mononucleosis, erythema multiforme, including variants (Stephens - Johnson syndrome), pemphigus vulgaris, benign mucous membrane pemphigoid
5. List the methods of transmission for the following diseases:
   Herpes simplex (primary herpetic gingivostomatitis, recurrent HSV infections, herpes labialis, etc...), varicella-zoster, measles, infectious mononucleosis
6. List the clinical findings associated with the following diseases:
   Herpes simplex (primary herpetic gingivostomatitis, recurrent HSV infections, herpes labialis, etc...), varicella-zoster, measles, infectious mononucleosis, erythema multiforme, including variants (Stephens - Johnson syndrome), pemphigus vulgaris, benign mucous membrane pemphigoid
7. List the management for primary herpetic gingivostomatitis, recurrent herpes simplex and herpes zoster.
8. Identify vesiculo-bullous diseases when clinical information and / or slides are given.

Vesiculo-bullous Diseases	**Color Atlas**
Herpes Simplex Virus	184 -185
Primary Herpetic Gingivostomatitis	184 - 185
Herpetic Whitlow	184 - 185
Varicella	186 - 187
Herpes Zoster	186 - 187
Measles (Rubeola)	-
Infectious Mononucleosis	202 - 203
Erythema Multiforme	190 - 191
Stephens - Johnson Syndrome	190 - 191
Pemphigus Vulgaris	192 - 193
Benign Mucous Membrane Pemphigoid	-

VESICULO-BULLOUS DISEASES

⇒ those lesions which produce a "blister-like" lesion; lesion is fluid - filled

Vesicle

- is a small, fluid - filled blister; < 1 cm in size
- fluid is lymph or blood

⇒ subepithelial or intraepithelial

Bulla

- is a larger, fluid - filled blister; > 1 cm in size
- may form when several vesicles coalesce

⇒ subepithelial or intraepithelial

- history of vesicles or bulla limits disease to viral disease or oral findings of dermatologic diseases

⇒ V-B diseases can exhibit a positive *Nikolsky* sign (diagnostic sign whereas normal epithelial tissue can be rubbed off with finger pressure)

Viral Diseases

- uncommon to see vesicles or bulla clinically
- oral findings by viruses is common
- Viral infections presenting in this classification of disease processes include:

Viruses and Associated Conditions of Significance to Clinical Dentistry		
Virus Family	**Virus**	**Disease**
Herpes virus	HSV type 1	Primary herpetic gingivostomatitis Secondary herpes Herpetic whitlow Occasionally, genital herpes
	HSV type 2	Genital herpes Occasionally, oral herpes
	Varicella – zoster virus	Varicella, herpes zoster
	Epstein – Barr virus	Mononucleosis Hairy leukoplakia
	Cytomegalovirus	Salivary gland disease
Papovavirus	Papilloma virus	Oral warts Oral papilloma Carcinoma?
Paramyxovirus	Measles virus Mumps virus	Measles Mumps

Herpes Simplex

Etiology:

⇒ viral – HSV type I / II

⇒ Transmission:

- typically by direct physical contact with infected person

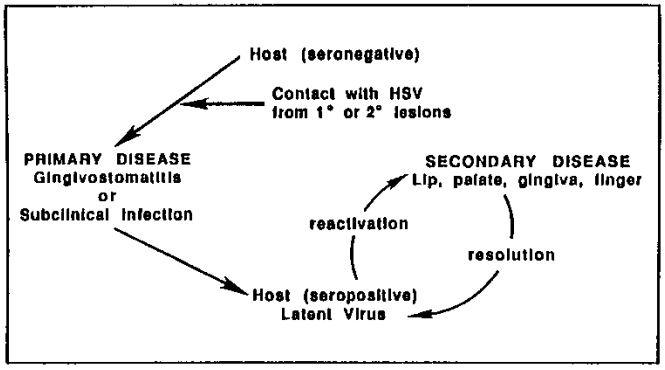


Figure 1–2. Pathogenesis of HSV-I infections.

Type 1 HSV appears in 2 forms:

- Primary herpetic gingivostomatitis and secondary (recurrent) HSV

Primary Herpetic Gingivostomatitis

- oral disease caused by the initial infection with HSV
- following exposure, 10% show clinical signs & symptoms of primary infection; 90% are sub-clinical (go un-noticed)

⇒ Clinical Findings:

- children (typical ages are between 3 – 10), young adults
- child will initially exhibit fever, malaise, headache, irritability, pain in mouth
- initial signs and symptoms are followed in 1-3 days by vesicular eruptive eruption
- on gingiva, vermilion border, mucous membranes
- vesicles rupture and leave painful ulcers
- initial infection infects non-keratinized tissues
- red, edematous gingival tissues are noted
- bilateral, painful lymphadenopathy is present
- dehydration can result in a patient with primary herpetic gingivostomatitis

Treatment:

- this is a self-limiting disease and will run it's course
- fluids; no acids, ice cream, yogurt
- antipyretics (no ASA), antibiotics prn
- clean teeth with cotton tips

Secondary or Recurrent HSV Infections

- reactivation of latent HSV in pre-infected persons
- up to 90% of the population has antibodies to HSV (has been infected with HSV); 40% of these will develop secondary HSV clinically
- reactivation of latent virus in the trigeminal nerve ganglion; reactivated virus travels by way of the trigeminal nerve to the originally infected epithelial surface, where replication occurs

⇒ Precipitating factors (those factors that predispose a patient to having an outbreak) include:

- emotional stress, sunlight ("fever blisters"), common cold, heat, trauma, illness,
- dehydration, immunosuppression, scaling, tooth extraction, menstruation
- common manifestation of AIDS

Clinical Findings:

- rapid onset
- prodromal symptoms occur a few hours before vesicular eruption (25% do not have these)
- multiple clusters of vesicles; vesicles rupture to leave small ulcerations
- ulcerations can coalesce

Herpes Labialis

- "cold sores"
- reactivation of HSV on the lips and the adjacent perioral tissues
- edema and redness on areas and is followed by clusters of small vesicles
- 90% of secondary HSV will occur as herpes labialis
- reoccurrence will occur at or near the same site

⇒ as for intraoral lesions (10% of secondary HSV lesions), the outbreaks will occur on keratinized tissues – will present on the hard palate or attached gingiva

- heal in 7 - 14 days without scarring
- watch for exaggerated cases- may be indicative of an immunocompromised patient

Treatment:

- goal is to control and not cure; the sooner the treatment is started, the better (start no less than 48 hours after onset of symptoms)
- antivirals- acyclovir, Valtrex
- lysine, ice, Motrin
- sunscreen on lips to prevent outbreaks
- maintain fluid /caloric intake

Herpetic Whitlow

⇒ refers to either a primary or secondary HSV infection involving the finger(s)

- pain, redness and swelling; up to 4-6 weeks in duration

Varicella - Zoster

- virus that causes both chickenpox and shingles; creates a maculopapular rash

Varicella

⇒ primary H-Z outbreak in a seronegative individual is known as varicella

Etiology:

- primary infection with varicella-zoster virus

Transmission:

- airborne- mainly through the inhalation of contaminated droplets, but can also be
- through direct contact (a minor transmission method)
- increased prevalence in winter and spring

Clinical Findings:

- contagious childhood disease, but due to vaccinations, it is uncommon to see in developed countries
- presents with headaches, fever, chills, malaise
- red rash initially presents on the trunk and will spread outwardly
- rash → vesicles → pustular → crusting
- as lesions are healing, there will be new lesions forming; will see lesions in varied stages
- if oral lesions are noted, vesicles → ulcers
- palate, lips- more common sites
- heals in 7 - 10 days

Treatment:

- palliative
- heals on its own
- may scar

Herpes Zoster

⇒ shingles

Etiology:

- in adults, the V-Z virus usually causes a different form of the disease; this secondary or reactivated disease is known as Herpes Zoster or shingles
- unknown factors associated with reactivation, but this is considered a condition of the
- older adult population and of individuals who have compromised immune responses
- characteristically follows (triggers) such occurrences as immunosuppressive states resulting from malignancy (leukemia), drug administration, HIV, seen in elderly, immunocompromised patients, those receiving steroids or radiation, surgery and
- radiation of the spinal cord, local trauma

Clinical Findings:

⇒ characterized by unilateral, painful vesicular eruption along sensory nerve distribution

⇒ typically affects sensory nerves of the trunk and head/neck

Areas of Involvement:
- trunk (T3 - L2)
- head and neck- (V1 / V2)

- prodromal symptoms → rash → vesicles / ulcers → new lesions
- accompanied by pain, fever, malaise, headache

Treatment:
- antiviral agents- high doses of oral acyclovir
- palliative- analgesics, antibiotics prn
- heals on its own

Measles (Rubeola)

- acute, contagious infection of childhood

⇒ Etiology:

- paramyxovirus

Transmission:

- in saliva, secretions, bedding, dust

Clinical Findings:

- child exhibits fever, malaise and sore throat

⇒ the initial finding is an oral lesion called Koplik spots - bluish-white specks with red areola (bilaterally on the buccal mucosa)

- maculopapular skin rash presents next on the patient
- rash begins on the head and neck and will progresses in an outward manner

Treatment:

- palliative treatment- analgesics, antibiotics prn
- bed rest, fluids, diet

Infectious Mononucleosis

- an acute, self-limiting disease process
- low contagiousness is associated with this condition

Etiology:

⇒ Epstein – Barr Virus

Transmission:

- unclear, but is frequently transmitted through salivary transfer

Clinical Findings:

- typically noted in young adults
- fever, malaise, headache, fatigue, tonsillitis are present
- accompanied by painful generalized lymphadenopathy
- oral lesions are often the earliest manifestation noted; multiple red palatal petechiae found at the junction of the hard and soft palate
- additional complications are possible (liver / spleen damage)

Treatment:

- palliative treatment, bed rest, diet, analgesic, antipyretics, disease runs course in few weeks

Erythema Multiforme

- is an acute, self-limiting hypersensitivity reaction that is characterized by the appearance of skin and mucous membranes lesions
- symptom complex that is associated with vesicles / bulla and a characteristically appearing skin rash

Etiology:

⇒ unknown (obscure), but hypersensitivity is suspected

⇒ may be an autoimmune response and has been associated with a reaction to medications,

⇒ viral, bacterial, fungal involvement, previous HSV infections, pregnancy, and sunlight

⇒ in half of the cases, trigger factors can be identified and these fall into two categories: infections (HSV, TB) and drugs (barbiturates, sulfonamides, phenytoin)

Clinical Findings:

- can affect any age, but more commonly noted in young adult males
- acute onset is typical
- low grade fever, malaise, headache- precedes the appearance of lesions by a few days

⇒ classic lesion of EM is the target / iris / bulls-eye lesion (this skin lesion presents as red macules arranged in a symmetrical pattern)

⇒ oral lesions are possible- see painful vesicles / ulcerations that typically encircle the mouth (oral lesions are seen in 25-50% of those with cutaneous lesion)

Treatment:

- no real treatment is done; try to determine the etiology
- bed rest, diet, hydration
- systemic steroids, antibiotics prn

⇒ Stephens - Johnson Syndrome

- represents a severe variant of erythema multiforme
- presents with bulla / erosions on multiple sites
- eye / genital / oral involvement

Pemphigus

⇒ pemphigus is a general term for a group of chronic autoimmune, mucocutaneous diseases that are characterized by the formation of intraepithelial blisters

- various types of pemphigus exist; p. vulgaris represents the most frequently encountered type

Pemphigus Vulgaris

Etiology:

⇒ believed to be an autoimmune disorder

Clinical Findings:

⇒ it is characterized by the development of vesiculobullous lesions on the mucous membranes and skin

- 68% will present with intraoral lesions prior to cutaneous lesions
- vesicles / bulla → ulcers
- condition exhibits a + Nikosky sign
- oral lesions are most commonly noted on the lips, buccal mucosa, gingiva, and palate

Treatment:

- medical referral, adequate diet, systemic steroids, oral topical steroids

Benign Mucous Membrane Pemphigoid

Etiology:

- believed to be a chronic autoimmune disease process
- it occurs primarily on the mucous membranes with occasional skin involvement

Clinical Findings:

- the formation of bulla are most commonly noted on the palate, gingiva, and buccal mucosa
- skin lesions will begin as a rash → bulla form → rupture
- lesions may also appear on conjunctiva, genitalia

Treatment:

- medical referral, systemic steroids prn, oral topical steroids

Ulcerative Conditions

Lecture Outline

1. Reactive lesions
   - etiology
   - clinical findings
   - treatment
2. Syphilis
   - etiology
   - transmission
   a. Primary syphilis
      - chancre
      - clinical findings
   b. Secondary syphilis
      - mucocutaneous lesions
      - mucous patch
      - clinical findings
   c. Tertiary syphilis
      - gumma
      - additional oral findings
   d. Treatment
   e. Congenital syphilis
      - etiology
      - congenital malformations
      - Hutchinson's triad
3. Tuberculosis
   - etiology
   - transmission
   - clinical findings
   - oral lesions
   - treatment
4. Aphthous ulcers
   - variations
   a. Minor aphthous ulcers
      - etiology
      - clinical findings
      - locations
      - comparison of secondary oral HSV infections to minor aphthous
      - treatment options
   b. Major aphthous stomatitis
      - clinical findings
   c. Bechet's syndrome
      - clinical findings
   d. Herpetiform ulceration
      - clinical findings

5. Lupus erythematosis
   - etiology
   a. Discoid lupus erythematosis (DLE)
      - clinical findings
      - butterfly rash
   b. Systemic lupus erythematosis (SLE)
      - multi-system organ involvement
      - butterfly rash

Lecture Objectives:

1. Define the following: ulcer, iatrogenic, reactive lesions
2. List the etiology for the following diseases:

   Syphilis, congenital syphilis, tuberculosis, aphthous ulcers and variants (minor, major, Behcet's syndrome, herpetiform), lupus erythromatosis
3. List methods of transmission for syphilis and tuberculosis.
4. Compare clinical findings in the three stages of syphilis.
5. List clinical findings that are associated with the following diseases:

   Syphilis, congenital syphilis, tuberculosis, aphthous ulcers and variants (minor, major, Behcet's syndrome, herpetiform), lupus erythromatosis
6. Discuss the clinical management for reactive lesions, aphthous ulcerations, and allergic conditions.
7. Identify ulcerative conditions when clinical information and / or slides are given.

Ulcerative Conditions	**Color Atlas**
Reactive Lesions	202 - 203
Syphilis	202 - 203
Congenital Syphilis	-
Tuberculosis	-
Recurrent Aphthous	194 - 195
Major Aphthous Stomatitis	196 - 197
Behcet's Syndrome	196 - 197
Lupus Erythematosus	166 - 167

Ulcerative Conditions

Ulcers ⇐

- defined as a loss of epithelium due to any cause
- these may be initially preceded by vesicles / bulla
- varied clinical appearance and etiology
- can be either an acute or chronic process

Reactive Lesions

Etiology:

⇒ trauma; acute or chronic in nature

- include iatrogenic, self-induced, use of chemicals on tissue, burns and radiation

Clinical Findings:

- shape and size of a traumatic ulcer will vary
- whether pain is present or not, will depend on whether the lesion is associated with an
- acute or chronic situation
- need to obtain a thorough history on the lesion

Treatment:

- observe
- remove cause
- symptomatic treatment prn

Syphilis

- is a systemic, communicable disease / venereal disease

Etiology:

⇒ bacterial origin

⇒ caused by a spirochete- *Treponema pallidum*

Transmission:

1. sexual contact (contact with an active lesion)- main method of transmission
2. transplacental
3. transfusion of infected blood

⇒ There are 3 stages associated with syphilis

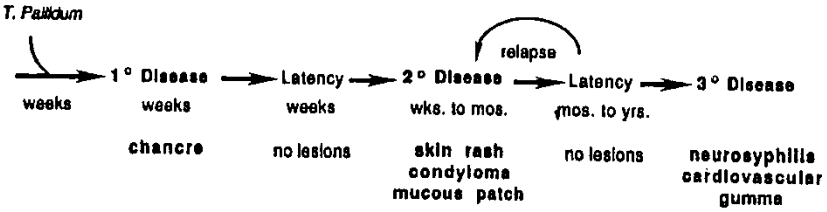


Figure 2–10. Pathogenesis of syphilis.

⇒ Clinical Findings- Primary Syphilis:

- chancre – this is the lesion associated with primary syphilis
  - lesion is found at the site of spirochete entry or site of initial contact
  - presents as a solitary, indurated, painless ulcer
  - ulcer will exhibit rolled margins
  - surface of the chancre contains spirocetes
  - majority of chancres are found on the genitalia, but can be found on the oral tissues
- patient will also exhibit unilateral, regional lymphadenopathy

Clinical Findings- Secondary Syphilis:

- patient with secondary syphilis will exhibit a skin rash, fever, generalized
- lymphadenopathy, and sore throat
- mucocutaneous lesions (skin rash)- presents as painless, red-brown macules/papules

⇒ mucous patch – this is the oral lesion associated with secondary syphilis

⇒ mucous patch is a highly contagious, painless ulcer with red halo that is covered by a mucous exudate

⇒ the mucous patch will mimic cancer

Clinical Findings - Tertiary Syphilis:

⇒ gumma – this is the oral lesion associated with tertiary syphilis

⇒ gumma is a painless necrotic tumor

⇒ additional findings associated with tertiary syphilis include palatal perforations and atrophic glossitis

- above mentioned lesions are not contagious in this stage
- patients with tertiary syphilis will also exhibit changes in the nervous system and heart

Treatment: education, Penicillin in earlier stages

Congenital Syphilis

Etiology:

- refers to syphilis that is transmitted to the fetus from an infected, pregnant mother

Findings Associated with Congenital Syphilis:

⇒ congenital malformations are noted in newborn

⇒ the presence of Hutchinson's triad:
1. Interstitual keratitis
2. 8th nerve deafness
3. Dental abnormalities- Hutchinson's Incisors, Mulberry Molars

Tuberculosis

- typically found in areas of low socioeconomics, poor living conditions and in the immunocompromised

Etiology:

⇒ bacterial origin- mycobacterium tuberculosis

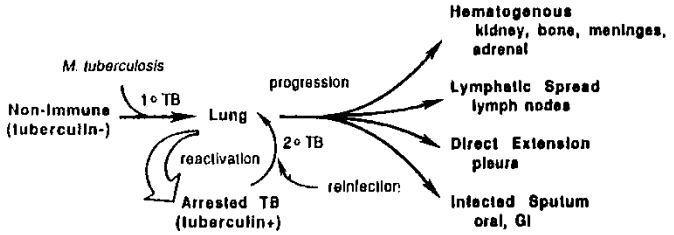


Figure 2–15. Pathogenesis of tuberculosis.

Transmission:

- by airborne droplets

Clinical Findings:

⇒ can infect any tissue, but the main tissue is the lungs

- patient will also exhibit weight loss, cough, lymphadenopathy, possible chest pain
- also associated with a low grade fever, night sweats and malaise

⇒ if found orally, the presence of a granulomatous ulcer is noted

- lesion is a non-healing (chronic) ulcer that is indurated and painless
- reflects secondary infections⇐
- favored sites include tongue, palate ⇐
- lesion is contagious ⇐

Treatment:

- antituberculosis agent- INH, rifampin, streptomycin

Aphthous Ulcers

4 Variations of Aphthous Ulcers:

⇒ Minor- most common form

- Major
- Herpetiform
- Aphthous associated with Bechet's syndrome

Minor Aphthous Ulcers

- "canker sores"

⇒ "aphthous stomatitis" / recurrent aphthous ulcers (stomatitis)

- most common lesion of the oral mucosa; studies find that 15-20 % to 20-60% of the population are affected

Etiology:

- unknown

⇒ many theories exist- immune dysfunction is the most likely cause, possible stress, trauma, nutritional deficiencies (B_{12}, iron), hormonal imbalance, food hypersensitivity, smoking cessation can be exaggerated in immunocompromised cases

⇒ Clinical Findings:

- patient may exhibit prodromal symptoms
- lesion appears on the oral tissues as an ulcer; no history of vesicles
- presents as a small, painful, recurrent ulcer covered by a yellow fibrinous
- membrane that is surrounded by an erythematous halo
- lesion is oval to round, single or multiple (1-5 in number), found on moveable tissues
- lesion will typically recur on a regular basis
- more commonly found in professionals, upper socio-economic groups and in
- those who do not smoke

Comparison of Secondary Oral Herpes Simplex Infections to Minor Aphthous

Parameter	Herpes	Minor Aphthous
Cause	HSV-1	Focal immunodysregulation
Precipitating factors	Stress, trauma, UV light, change in immune status	Stress, trauma, hormonal changes, diet, immunologic alterations
Prodromal symptoms	Usually	Occasionally
Vesicular stage	Yes	No
Number of ulcers	Multiple, confluent	Usually one, oval
Pain	Yes	Yes
Location	Hard palate, gingival, vermillion	Moveable intraoral tissues
Duration	1-2 weeks	1-2 weeks
Recurrent	Yes	Yes

Treatment: Try and identify any predisposing factors. Can use mouth rinses that contain sodium bicarbonate / water, chlorohexidine, or tetracycline suspensions; lasers, silver nitrate.

Major Aphthous Stomatitis

- more severe form of aphthous stomatitis; affects 10% of those with RAS
- presents with larger, more extensive ulcers (> 1 cm) last longer and reoccur more frequently
- lesions can scar

Bechet's Syndrome

- multi-system disease in which recurrent oral aphthous are a consistent finding
- of unknown etiology, but is believed to involve aspects of the immune system
- presents with a range of manifestations- oral, ocular and genital ulcerations, arthritis, CV disease, etc….

Herpetiform Ulceration

- recurrent focal ulceration that resembles herpes (ulceration that is left)
- no vesicles
- multiple, small shallow ulcerations
- unknown etiology

Lupus Erythematosis

- represents an acute / chronic inflammatory, autoimmune disease process
- considered a syndrome because this disease can include a wide spectrum of disease activity

⇒ Etiology:

- believed to be a chronic inflammatory autoimmune disease that can have acute exacerbations
- disease may be influenced by genetic or viral factors and may be triggered by medications, sunlight, stress and infections

2 major forms / types of lupus exist:

1. Discoid Lupus Erythematosis (DLE)
    - most mild form of lupus
    - less aggressive form that affects only the skin
    - skin lesions are most commonly noted on the face and scalp (see erythematous plaques with hyperpigmented margins)

    ⇒ classic lesion of DLE is the butterfly rash – this rash presents as a reddish rash on bridge of nose

    - characteristically seen in middle age women (F>M, 8:1); blacks > whites (3:1)

    ⇒ 20 - 40% of the patients with DLE will also exhibit oral findings and present as ulcerations, erosions, white plaques

2. Systemic lupus Erythematosis (SLE)
    - most common form of lupus
    - have multi-system organ involvement that results in kidney, cardiac, lung and joint disease
    - skin and mucosal lesions are mild; most complaints deal with systemic involvement (fever, weight loss, malaise, joint pain, heart and lung involvement)
    - butterfly rash can also be seen
    - oral lesions associated with SLE are similar to those found in DLE

Treatment:

- topical steroids on skin lesions and systemic steroids for systemic involvement

Neoplastic Conditions

Lecture Outline

1. Cancer statistics
2. Basal cell carcinoma
   - etiology
   - secondary factors
   - general clinical findings
   - treatment options
3. Squamous cell carcinoma
   - statistics
   - role of the dental team
   - etiology
   - risk factors
     - tobacco usage
     - alcohol
     - additional risk factors
   - ethnicity

   a. Tobacco and oral cancer
   b. Things not associated with oral cancer
   c. Clinical findings
   d. Locations
   e. Verrucous carcinoma
   f. Carcinoma in-situ
   g. Lesion evaluation
      - clinical examination
      - biopsy
      - OralCDx
      - toluidine blue staining

   g. Metastatic lesions
   h. Prognosis
   i. TNM staging
   j. Treatment options
   k. Radiation side effects
      - reversible side effects
      - irreversible side effects

Lecture Objectives:

1. List the etiology and secondary factors associated with basal cell carcinoma.
2. Recall clinical findings associated with basal cell carcinoma.
3. List the etiology and risk factors associated with squamous cell carcinoma.
4. Explain reasons why there is a poor survival rate associated with squamous cell carcinomas.
5. List ethnic findings that are associated with squamous cell carcinoma.
6. Recall clinical findings associated with squamous cell carcinoma.
7. List common presenting signs of oral cancer.
8. Identify the common clinical locations for squamous cell carcinoma to occur.
9. Define the term carcinoma in-situ.
10. List clinical findings associated with verrucous carcinoma.
11. Compare the methods of treatment for squamous cell carcinoma.
12. List side effects associated with radiation therapy.
13. Identify neoplastic conditions when clinical information and / or slides are given.

Neoplasms	**Color Atlas**
Basal Cell Carcinoma	-
Squamous Cell Carcinoma	118 - 119, 162 - 163, 197 - 198
Verrucous Carcinoma	156 - 157

NEOPLASTIC CONDITIONS

Neoplasm

- tumor = cancer = neoplasia
- benign vs. malignant
- group of diseases characterized by uncontrolled growth and spread of abnormal cells

Cancer Facts:

- anyone is at risk for developing cancer; occurrence increases with increasing age
- family history, at risk behaviors can come into play
- about 1,443630 new cancers to be diagnosed in 2009 (does not include basal / squamous cell skin cancers)

2010 - Seven Leading disease causes of death in US:

Heart disease
Cancers- (1 out of every 4)
Pulmonary disease
Stroke
Accidents
Alzheimer's
Diabetes

Leading Sites of New Cancer Cases:	Cancer Death by Site:
Males: ▪ Prostate (26 %) ▪ Lung (16 %) ▪ Bladder (7 %)	Males: ▪ Lung / bronchus (31 %) ▪ Prostate (10 %) ▪ Bladder (4 %)
Females: ▪ Breast (27 %) ▪ Lung (15 %) ▪ Colon / rectum (8 %) ▪ Uterine (7 %)	Females: ▪ Lung (26 %) ▪ Breast (15 %) ▪ Colon / rectum (9 %) ▪ Uterine (4 %)

Basal Cell Carcinoma

- "rodent ulcer"

⇒ most common malignant neoplasm of the skin (800,000 – 900,000 new cases per year)

Etiology

- unknown

Secondary factors associated with it:

  - fair skin
  - chronic sun exposure
  - increased age
  - history of skin burns

General Clinical Findings

⇒ found in areas that are exposed to the sun

⇒ has a predilection for mid- to upper face

⇒ is not found on mucous membranes

- more common in males vs. females
- more commonly found in those over the age of 40; those that are found in "younger" patients are typically more aggressive in nature
- are a locally invasive, slowly spreading cancer
- rarely metastasizes

Clinical Findings Associated with Early Lesions: (variations exist)

- indurated, elevated nodule with a smooth center
- hyperkeratotic
- ulceration with a crusted center

Clinical Findings Associated with Mature Lesions:

- large nodule with or without ulceration
- non-healing ulcer
- pigmented tumor

Treatment

- surgical removal, radiation

Squamous Cell Carcinoma

- account for 3 – 5 % of all cancers that will be diagnosed
- 90 % of oral cancers are squamous cell carcinomas

2009 Statistics for Oral Cancer (estimated) in the US

	Total	Male	Female
New Cases	35,720	25,240	10,480
Deaths	7,600	5,240	2,360

2011 Statistics for Oral Cancer in North Carolina

	Total
New Cases	1,342
Deaths	235

Role of the dental team:

1. Identification
2. Management and referral
3. Management of oral complications

Etiology:

- unknown, but several risks factors have been identified as to having a role with the development of oral cancer; oral cancer is a multi-factorial disease

⇒ Risk factors:

- tobacco plays a significant role
  - tobacco is believed to be the important factor regarding the development of oral cancer
  - all forms of tobacco have been linked to causing oral cancer
  - cigar and pipe smoking have a greater risk than that of cigarette smoking
  - reverse smoking significantly increases the risk
  - time and dose relationship
  - use of smokeless tobacco is associated with a low risk for developing oral cancer

- alcohol
  - adds to the risk of developing oral cancer, but determining how much is hard to prove
  - it irritates the mucosa and acts as a solvent for carcinogens that are found in alcohol and tobacco

- age
  - is an important risk factor
  - 95 % of the oral cancers occur in those over the age of 40

- sun (UV light) exposure has involvement with the development of basal cell of the skin and squamous cell of the lower lip and skin
- decreased immune responses will put a patient at risk for developing oral cancer
- dietary deficiencies- this area is not well-documented
- viruses (EBV, papillomaviruses (genital cancers), Human herpesvirus-8 (Kaposi's sarcoma)
  - some microorganisms have been implicated in the development of oral cancer
  - HPV subtypes 16 and 18 have been implicated in the development of tonsillar squamous cell and possibly in the development of verrucous carcinoma

Ethnicity:

- ethnic background is known to influence many types of cancers (Chinese: nasopharyngeal cancer)
- African Americans develop oral cancer more frequently than other ethnic groups
- there is no genetic influence for developing an oral cancer

Tobacco and Oral Cancer:

- oral cancer is associated with all forms of tobacco, as well as the use of betel nuts and reverse smoking
- "tars" (aromatic hydrocarbons) are the most potent carcinogens in tobacco
- smokers are twice as likely to get oral cancer than non-smokers
- smoking increases the risk for developing a second oral cancer
- snuff, dip or chewing tobacco are related to oral cancer, but it takes longer for the cancer to develop (average of 20 years); it is suggested that chewing tobacco is a significant cause of oral cancer, but epidemiologic studies have shown that the risk is small and less than that associated with smoking tobacco

Things Not Associated with Oral Cancer:

- chronic irritation
- dental x-rays
- fluoridated water

Clinical Findings Associated with Oral Cancer:

- more common to find in males (M > F, 2:1)

⇒ typically found in those over the age of 40

⇒ presents with varied clinical appearances and will make it hard to diagnose

⇒ chronicity and induration are important clues related to oral cancers

⇒ a combination of colors and surface patterns are possible

  - a lesion that is red or white with a red component will carry a greater risk for malignant transformation
  - if there is a red component and discomfort, it is more likely to be malignant
  - these lesions are more dangerous if they have an ulcerated area associated with them

- pain, soreness and irritation are the most common oral complaints when a patient does come into the dental office for an evaluation of a given area
- the more posterior and inferior the cancer is located, the worse the prognosis becomes
- patients may report numbness or burning sensation in the area of the lesion
- as the lesion ages, there will be an increase in size, the margins of the lesion become irregular, the lesion becomes fixed and indurated
- field cancerization is possible- refers to the development of separate, individual foci of cancer

⇒ <u>Sites of Occurrence:</u>

Tongue	30%
Oropharynx	27 %
Floor of mouth	23 %
Gingiva	8 %
Buccal mucosa	5 %
Lips	4 %
Hard palate	3 %

<u>Verrucous Carcinoma</u>

⇒ a variant or sub-type of squamous cell carcinoma

- lesion is associated with tobacco usage and may be associated with smokeless tobacco
- typically presents as an exophytic, pebbly / wart-like, slow growing and superficially spreading mass
- 80 – 90 % are found on the buccal mucosa, gingival and alveolar mucosa
- rarely metastasizes; favorable prognosis
- treatment by surgical excision

Carcinoma In-situ

⇒ represents severe dysplasia

⇒ refers to a carcinoma is not invasive and is still within the epithelium

Oral Cancer - Lesion Evaluation

- clinical examination
- biopsy
- exfoliative cytology; brush biopsy (OralCDx)
- toluidine blue staining (93% accurate)

Spread of Tumor

- oral cancers spread mainly by local extension of the lesion and by the lymphatic system
- any involved lymph nodes will enlarge, become indurated, become fixed and are asymptomatic
- metastatic spread from an intraoral lesion is most common to the lungs, bone and liver
- spread of cancers to the mouth from other sites is primarily from breast cancers and most commonly involves the mandible

Prognosis of Squamous Cell Carcinoma

- half of all patients with oral cancer will survive for at least 5 years following treatment
- advancements in treatments have not improved survival; early diagnosis is the key factor
- person with oral cancer is 2-3 times more likely to develop additional malignancies

Reasons as to why there is a poor survival rate associated with oral cancer:

- overall survival rate is about 50 %, with the survival rate for blacks being significantly lower

1. ½ will metastasize to regional lymph nodes prior to detection
2. the cells in the cancer are not well-organized
3. they are asymptomatic in earlier stages

Prognosis will depend on:

- lesion's histology
- site of the lesion
- TNM staging
  - site of primary Tumor (1-3)
  - Node involvement (0-3)
  - Distant Metastasis (0,1)

Treatment for Squamous Cell Carcinoma

- treatment used depends on many factors such as the size and site of the lesion, the health of the patient and whether the tumor has spread
- surgery
- radiation
- combination of these treatments
- chemotherapy can be added to improve the cure rates

Radiation Therapy Side Effects

Reversible Side Effects (those effects that will dissipate after the therapy is completed):

- ulcerations
- pain
- candidiasis
- erythema

Permanent Side Effects (those effects that will NOT dissipate after the therapy is completed):

- xerostomia
- radiation caries
- osteoradionecrosis

White Lesions

Lecture Outline

1. Leukoplakia
   - definition
2. Idiopathic leukoplakia
   - definition
   - etiology
   - clinical findings
   - treatment
   - biopsy evaluation
3. Soft tissue changes associated with white lesions
4. Lesion diagnosis
   - wipe off
   - lesion history
   - family history
5. Hereditary conditions
   a. Leukoedema
      - associated findings
      - clinical findings
      - lesion diagnosis
      - treatment
   b. White sponge nevus
      - clinical findings
      - treatment
6. Reactive lesions
   a. Frictional hyperkeratosis
      - etiology
      - clinical findings
      - treatment
   b. Snuff keratosis
      - etiology
      - clinical findings
      - treatment
      - pre-malignant
      - other findings associated with long term use
   c. Nicotine stomatitis
      - etiology
      - clinical findings
      - treatment
   d. Actinic (solar) cheilitis
      - etiology
      - clinical findings
      - treatment
        - pre-malignant lesion

7. Other white lesions
    a. Hairy leukoplakia
        - etiology
        - association with AIDS
        - clinical findings
        - treatment
    b. Hairy tongue
        - etiology
        - predisposing factors
        - clinical findings
        - variations in color
        - treatment
    c. Geographic tongue
        - etiology
        - associations
        - clinical findings
        - stomatitis areata migrans
        - treatment
    d. Lichen planus
        - etiology
        - clinical findings
        - mucocutaneous disease
        - varied forms
            - reticular
                - Wickham striae
            - plaque-like
            - atrophic
            - erosive
            - bullous
        - treatment
    e. Candidiasis
        - etiology
        - opportunistic infection
        - predisposing factors
        - clinical findings
        - various forms
            - acute pseudomembraneous
            - chronic atrophic form
            - chronic hyperplastic form
            - angular cheilitis form
        - treatment
    f. Angular cheilitis
        - etiology
        - additional causes
        - clinical findings
        - treatment

Lecture Objectives:

1. Define the following terms: leukoplakia, idiopathic
2. List the 3 soft tissue changes that will produce a white lesion.
3. List examples for each of the soft tissue changes that will produce a white lesion.
4. List the etiology for the following lesions:

   Leukoedema, white sponge nevus, frictional hyperkeratosis, white lesions associated with smokeless tobacco, nicotine stomatitis, actinic cheilitis, idiopathic leukoplakia, hairy leukoplakia, hairy tongue, geographic tongue, lichen planus, candidiasis, angular cheilitis

5. List the clinical findings that are associated with the following diseases:

   Leukoedema, white sponge nevus, frictional hyperkeratosis, white lesions associated with smokeless tobacco, nicotine stomatitis, actinic cheilitis, idiopathic leukoplakia, hairy leukoplakia, hairy tongue, geographic tongue, lichen planus, candidiasis, angular cheilitis

6. List clinical findings that will distinguish white lesions apart from one another.
7. Describe the clinical management for unexplained leukoplakic lesions.
8. Identify various white lesions when clinical information and / or slides are given.

White Lesions	**Color Atlas**
Leukoedema	152 - 153
White Sponge Nevus	154 - 155
Frictional Hyperkeratosis	156 - 157
Snuff Keratosis (Dipper's Patch)	156 - 157
Nicotine Stomatitis	156 - 157
Actinic (Solar) Cheilitis	132 - 133
Idiopathic Leukoplakia	154 - 155
Hairy Leukoplakia	126 - 127
Hairy Tongue	126 - 127
Geographic Tongue	128, 129
Lichen Planus	164 - 165
Candidiasis	168 - 169, 204 - 205
Angular Cheilitis	132 - 133, 168 - 169

WHITE LESIONS

Leukoplakia

- this is a generic clinical term that is used to identify a white lesion

⇒ localized white patches suggest trauma or neoplastic etiology

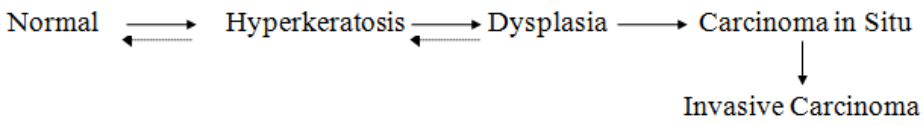
⇒ widespread involvement suggests a systemic, immunologic or hereditary condition

Idiopathic Leukoplakia

⇒ defined as a white patch or plaque that cannot be rubbed off and cannot be characterized clinically or pathologically as any other disease

Etiology

- most are associated with tobacco use
- other factors include protective reactions against chronic trauma, candida albicans, nutritional factors



Clinical findings

- associated with middle to older aged; most common in 40 - 80 year olds
- M > F (slightly)
- appears in any location, but tends to be seen in the buccal vestibule, palate, alveolar ridge, lip and tongue (more favored to least)

Treatment

⇒ identify etiology, remove cause, biopsy prn

Upon biopsy and evaluation, the following is noted:

- 80 % are benign
- 12 % exhibit dysplasia
- 4 % are carcinoma in-situ
- 4 % are carcinoma

⇒ **White Lesions- these lesions are produced by 3 soft tissue changes:**

1. increase in thickness of surface epithelium
2. accumulation of surface debris
3. reduced blood supply

Additional diagnostic procedures to help evaluate a white lesion:

- obtain thorough history on the lesion
- ask if something similar is present in other family members
- identify any oral habits that the patient may have

Hereditary Conditions

Leukoedema

- represents a normal anatomic variant of the oral mucosa that is due to thickened epithelium and edema

⇒ found in 92% of black adults and in 45% of white adults

⇒ Clinical findings

- found bilaterally on the buccal mucosa
- appears milky or as a diffuse opalescence
- can be wrinkled
- to help diagnosis this condition, stretch the patients' cheek → the lesion will
- disappear or dissipate
- no treatment needed

White Sponge Nevus

- rare, familial disorder that appears early in life

⇒ Clinical findings

- presents as a bilateral, deeply folded or spongy white lesion
- most commonly noted in the buccal / labial mucosa, floor of mouth, alveolar mucosa

Treatment

- none

Reactive Lesions

Frictional Hyperkeratosis

⇒ Etiology

- represents a protective measure against a low-grade, long term trauma; analogous to a callus

Clinical findings

- presents as a rough textured, grayish-white, slightly raised lesion
- has an irregular outline and cannot be rubbed off
- found in those areas that are more accessible to trauma

Treatment

- look for any identifiable causes
- obtain a thorough history
- biopsy prn

Snuff Keratosis

- "snuff dipper's patch"

⇒ Etiology

- chewing tobacco or snuff placed directly onto the oral tissues and causes hyperkeratosis
- all forms of smokeless tobacco may cause this tissue damage, but snuff is more
- likely to cause the lesion (as compared to chewing tobacco)
- is a response to tobacco constituents and possibly to the flavoring and moisture retention agents

⇒ Clinical findings

- presents as an asymptomatic lesion that is located at the site where the tobacco is placed on the tissues
- most commonly noted in the buccal mucosa of the mandibular molar and incisor regions
- early lesion will appear pale pink and wrinkled
- as the lesion "ages", the lesion becomes whitish in color, has a wrinkled appearance; it can be folded in some cases
- lesion does not wipe off

Treatment

- discontinue use of smokeless tobacco
- possible malignant transformation? It has been suggested that the use of smokeless tobacco is a significant cause of squamous cell carcinoma, but epidemiologic studies have shown that the risk is small and less than that associated with smoking tobacco
- long-term smokeless tobacco usage is also associated with an acceleration of periodontal disease, an increase in dental caries, alterations in taste

Nicotine Stomatitis

⇒ "smokers' palate"

- represents a tobacco-related form of keratosis

Etiology

- most commonly associated with pipe and cigar smoking; can also see with cigarette and reverse smoking
- thermal and chemical agents of the tobacco act locally on the tissues

⇒ Clinical findings

- presents as an asymptomatic, grayish-white lesion with red dots on palate that is posterior to the palatal rugae
- grayish-white part of the lesion is due to increased keratinization
- red dots represent ductal inflammation of the minor salivary glands
- presence of this lesion is an indicator that the patient is a heavy smoker

Treatment

- discontinue smoking → lesion regresses
- rare to develop into palatal cancer

Actinic (Solar) Cheilitis

- represents accelerated tissue degeneration of the lower lip

⇒ Etiology

- condition is caused by chronic sun exposure

⇒ Clinical findings

- mainly noted on the lower lip
- lip will appear atrophic and pale; it can appear fissured, wrinkled, dry and scaly

- most commonly noted in the elderly and fair-skinned males with outdoor occupations

Treatment
- none needed
- continue to observe for any changes
- considered a pre-malignant lesion

Other White Lesions

Hairy Leukoplakia

⇒ Etiology

- represents an opportunistic infection that is caused by EBV with a possible candida albicans
- found predominately in those with HIV (80% of those with HL will progress into AIDS), but can be found in other forms of immunosuppression (particularly those associated with organ transplants)

⇒ Clinical findings

- presents as an asymptomatic, white lesion on the lateral borders of tongue
- lesion can also be seen to involve ventral surface
- lesion can have a plaque-like or vertical corrugated appearance
- lesion is typically bilateral, but can also be unilateral
- lesion cannot be rubbed off

Treatment
- none
- may resolve while taking Acyclovir

Hairy Tongue

- clinical term that refers to a condition of filiform papillae overgrowth on the dorsal surface of the tongue

⇒ Etiology

- unknown, but is most commonly seen in those patients that do not clean their tongue; the patient does not naturally exfoliate the epithelial cells on the dorsal surface
- other predisposing factors or conditions that this condition is seen in include those taking broad spectrum antibiotics, systemic steroids, using oxidizing agents, heavy smokers, those on radiation therapy, those with candida albicans, those with febrile illnesses, xerostomia

Clinical findings

⇒ presents as an abnormal elongation (hypertrophy) of the filiform papillae (anterior 2/3s of dorsal surface of tongue)

- clinically, the dorsal surface will have a "furred" appearance, be asymptomatic, with varying colors being possible

⇒ variations in color are the result of extrinsic (diet, hygiene, smoking, coffee, etc...) and / or intrinsic factors (chromogenic bacteria)

Treatment

- identify causative agents and discontinue usage, brushing, medications

Geographic Tongue

- "benign migratory glossitis"

⇒ Etiology

- unknown, but may be due to stress, fungus or bacteria
- strongly associated with fissured tongue, but inversely associated with cigarette
- smoking
- geographic tongue is 5 times more frequently seen in those with psoriasis

⇒ Clinical findings

- condition is characterized by the presence of multiple atrophic patches (loss of filiform papillae) bordered by elevated keratotic margins on the dorsal and lateral
- borders of the tongue
- atrophic area will start small and enlarge with a change in pattern occurring over the
- duration of the condition
- involved areas appear and regress relatively quickly over a period of a few days
- more common in females than males; more commonly noted in whites and
- African Americans as compared to Mexican Americans
- mostly asymptomatic, but may see come slight tenderness
- stomatitis areata migrans- refers to ectopic geographic tongue

Treatment

- none needed
- self-limiting

Lichen Planus

Etiology

- unknown, but possibly an autoimmune process
- microscopically, it resembles a hypersensitivity reaction
- often associated with a psychogenic component

Clinical findings

⇒ mucocutaneous disease that exhibits both skin and mucous membrane lesions

- varied appearances - 5 forms exists:

  ⇒ reticular- most common form, is associated with the presence of Wickham striae (white, symmetrical, lace-like pattern that is primarily found on the buccal mucosa as a bilateral lesion)

  – plaque-like
  – atrophic
  – erosive- this form tends to be seen in those with an increased emotional stress
  – component
  – bullous

- may have a predilection for adult women; middle aged
- severity parallels level of stress
- lesions on skin (wrist arms, legs) are reported in 20 – 60 % of those with oral lesions

Treatment

- no specific treatment, but try antiseptic mouth rinses in combination with topical steroids
- systemic steroids in severe cases

Candidiasis

⇒ common opportunistic, oral mycotic infection that develops in the presence of one of several predisposing factors

- referred to as being "the disease of the diseased"

Etiology

⇒ represents an opportunistic infection caused by an overgrowth of candida albicans (c. albicans is a commensal organism that resides in the oral cavity of a majority of healthy persons; it is of weak pathogenicity and therefore requires predisposing factors to produce a disease state)

- superficial infection may be a clinical symptom of an underlying systemic problem

⇒ Predisposing Factors for Candida Infection:
- Immunologic immaturity of infancy
- Systemic antibiotic therapy
- Endocrine disturbances:
  - Diabetes mellitus
  - Hypoparathyroidism
  - Pregnancy
  - Systemic steroid therapy / hypoadrenalism
- Advanced malignancy
- Malabsorption and malnutrition
- Cancer chemotherapy
- May see limited amount of involvement in patient using an inhaler
- Other forms of immunosuppression (e.g., HIV, AIDS)

- age is an important factor in the development of this disease- it is seen in 5% of newborns, in 5% of cancer patients and in 10% of debilitated elderly

Clinical findings

⇒ various forms exist, but it is typically thought of as presenting as the acute pseudomembraneous (thrush) form

- soft, creamy yellowish-white, curd-like lesion (soft plaque)
- lesion will rub off to leave a raw, red surface

- other forms are found intraorally
  - chronic atrophic form
  - chronic hyperplastic form
  - angular cheilitis form

Treatment

- eliminate underlying factors, antifungal (Nystatin, Miconazole, etc...)

Angular Cheilitis (Cheilosis)

- represents a form of candidiasis (chronic atrophic)

⇒ Etiology

- candidia albicans; may be in combination with the presence of staphylococci aureus (bacteria associated with nasal microflora)

  Other causes include:

  - riboflavin deficiency
  - mechanical trauma
  - decreased vertical dimension
  - may be seen in individuals who habitually lick their lips

⇒ Clinical

- presents as reddened, cracking or fissuring at the labial commissures
- lesions are moderately painful, fissured, eroded and crusted; may exhibit yellow crusting at the area
- more commonly seen in those with deep folds at the commissures as a result of
- overclosure

Treatment

- identify etiology
- managed by topical application of antimicrobial-m miconazole (has activity against candida albicans and staphylococci)

Red Lesions

Lecture Outline

1. Erythroplakia
   - definition
   - etiology
   - risk factors
   - clinical findings
   - high index of suspicion
   - treatment
2. Soft tissue changes associated with red lesions
   - diascopy
3. Sublingual varices
   - etiology
   - clinical findings
4. Hemangioma
   - etiology
   - different types
   - clinical findings
   a. Port-wine staining
      - definition
      - clinical findings
   b. Sturge - Weber syndrome
      - clinical findings
5. Pyogenic granuloma
   - etiology
   - clinical findings
   - treatment
   a. Pregnancy tumors
      - clinical findings
6. Peripheral giant cell granuloma
   - etiology
   - clinical and radiographic findings
   - histologically findings
7. Median rhomboid glossitis
   - etiology
   - associated patient types
   - clinical findings
   - treatment
8. Geographic tongue
   - can also be classified as a red lesion
9. Kaposi's sarcoma
   - etiology
   - AIDS related
   - clinical findings

    - extraoral lesions
    - intraoral lesions
10. Glossitis
    - etiology
    - clinical findings
    - treatment
11. Leukemia
    - etiology
    - acute and chronic forms
    - oral manifestations of blood disorders
    - terminology
        - petechiae
        - ecchymosis
    - clinical findings
    - gingival hyperplasia
12. Hematoma
    - etiology
    - clinical findings
    - patients prone to developing hematomas

Lecture Objectives:

1. Define the following terms: erythroplakia, petechiae, ecchymosis
2. List the 3 changes in the soft tissue that will produce a red lesion.
3. List examples for each of the soft tissue changes that will produce a red lesion.
4. List the etiology that is associated with the following lesions:

   Sublingual varices, hemangioma, pyogenic granuloma, pregnancy tumor, peripheral giant cell granuloma, median rhomboid glossitis, erythroplakia,kaposi's sarcoma, glossitis, leukemia, hematoma
5. List the clinical findings associated with the following lesions:

   Sublingual varices, hemangioma, pyogenic granuloma, pregnancy tumor, peripheral giant cell granuloma, median rhomboid glossitis, erythroplakia, kaposi's sarcoma, glossitis, leukemia, hematoma
6. Compare clinical findings that distinguish each of the erythoplakic lesions apart.
7. List the clinical management for unexplained erythroplakic lesions.
8. Identify various red lesions when clinical information and / or slides are given.

Red Lesions	**Color Atlas**
Sublingual Varices	4 - 5
Hemangioma	158 - 159
Pyogenic Granuloma	110 - 111
Pregnancy Tumor	110 - 111
Peripheral Giant Cell Granuloma	110 - 111
Median Rhomboid Glossitis	130 - 131
Glossitis	130 - 131
Erythroplakia	162 - 163
Kaposi's Sarcoma	207
Petechiae/Ecchymosis	158 - 159
Leukemia	108 - 109
Hematoma	158 - 159

RED - BLUE LESIONS

Erythroplakia

- clinical term that refers to a red patch on the mucous membrane

⇒ defined as an unexplained red patch that cannot be identified as any other specific disease

Etiology

- unknown
- causes are similar to those responsible for oral cancer (tobacco, alcohol, nutritional deficits, chronic trauma)

Clinical findings

- red patch with ill-defined margins; may exhibit speckling
- can be found on any location, but are more commonly noted in the floor of mouth, tongue, retromolar tissue and soft palate
- no gender predilection; most commonly noted in those between 50 – 70 years old
- erythroplakic lesions are seen less frequently than leukoplakic lesions, therefore they warrant a high index of suspicion

  Post-biopsy, most are found to be associated with severe tissue changes:
  - 51% - invasive carcinoma
  - 41% - severe dysplasia or carcinoma-in-situ

Treatment

- identify cause

⇒ if lesion persists for more than 14 days, it needs to be biopsied!

⇒ 3 reasons why lesions will appear red:

1. epithelial atrophy
2. increased blood supply
3. extravasation of blood

⇒ ***Diascopy*** - method used to help in the evaluation of a red lesion; it is the examination of tissue using pressure; pressure is placed on the tissue to see if it will blanch.

Sublingual Varices

Etiology

- developmental condition that relates to a benign proliferation (growth) of blood vessels

Clinical findings

- pronounced, sublingual veins that are most commonly found on the ventral surface and lateral borders of the tongue
- typically noted in older individuals

Treatment

- none needed

Hemangioma

- used as a generic term to encompass vascular lesions that are either congenital neoplasms of proliferating endothelial cells (Strawberry nevus) or lesions resulting from abnormal vessel morphology

⇒ Etiology

- relates to the developmental enlargement of localized blood vessels

⇒ Clinical findings

- different types possible and can be classified by location and / or vessel size:
  - central (bone)
  - peripheral (soft tissue)

    Location
  - capillary
  - cavernous

    Vessel size
- congenital hemangiomas present at birth or shortly afterwards, but may involute sometime afterwards
- congenital vascular malformations are persistent in nature
- for both hemangiomas and vascular malformations:
  - unilateral
  - positive diascopy
  - can exhibit a flat or raised surface
  - variations in color are possible and will depend on the vascularity and the depth of the vessel
- common locations include the lips, tongue and buccal mucosa

Port-wine staining

- refers to a capillary hemangioma that is found on the face
- found on the facial skin that is innervated by one or more branches of the trigeminal nerve

Sturge - Weber syndrome

- congenital syndrome that has Port-wine staining, increased vascularity to the head (brain, bone, etc…), ipsilateral vascular defect, contralateral paralysis and epilepsy

Treatment

- none needed; some lesions will involute in time
- surgical intervention if necessary

Pyogenic Granuloma

⇒ Etiology

- exaggerated granulation (connective) tissue response to an irritation or trauma
- poor oral hygiene is a very recognized etiologic factor, but a chronic irritant may be involved
- hormonal changes of puberty and pregnancy may modify the gingival reparative response to injury

⇒ Clinical findings

- presents as a red, soft, painless, exophytic, nodular mass that is composed mainly of hyperplastic granulation tissue (is rich in capillaries)
- located at the site of trauma as a peduculated or broad-based lesion that may be smooth or lobulated
- lesion has a tendency to bleed and is typically located on the labial gingiva
- more commonly noted in females

Treatment

- removal of irritant
- surgical excision

Pregnancy Tumors

⇒ term frequently used to refer to a pyogenic granuloma that is found on the gingiva during pregnancy
⇒ hormonal changes and exaggerated response to an irritation
- typically appear after the first trimester

Peripheral Giant Cell Granuloma

⇒ Etiology

- hyperplastic connective tissue response to an injury of the gingival tissues or PDL
- rare in occurrence

⇒ Clinical findings

- presents as a smooth, exophytic, red-blue lesion that is typically located anterior to the first molars
- lesion is firm, asymptomatic, broad-based or peduculated
- "cuffing" may be evident
- multinucleated giant cells are seen histologically

Treatment

- removal of irritant
- surgical excision

Median Rhomboid Glossitis

⇒ Etiology

- originally thought to be a congenital abnormality, it is now considered a permanent result of candida albicans infection (hyperplastic candidiasis)

⇒ Clinical findings

- presents as an well-defined, red, rhomboid (oval) lesion at the midline on the dorsal surface of the tongue (anterior to circumvallate papillae)
- involved area is devoid of filiform papillae
- lesion is typically asymptomatic lesion, but some patient may experience mild discomfort
- can presents as a flat plaque or as a raised, nodular mass
- presence of a "kissing" lesion may be noted on the hard palate
- typically seen in diabetics, immunocompromised patients or those taking antibiotics

Treatment

- none ; lesion may or may not disappear with the use of an antifungal agent

Geographic Tongue

- can also be classified as a red lesion

Kaposi's Sarcoma

⇒ Etiology

- malignant neoplasm of the blood vessels (endothelial cells) that is believed to be due to herpes virus, type 8
- most common cancer found in those infected with HIV infection (seen in 20-30% of AIDS patients)
- different types exist: Classic type, African, Immunodeficiency / Epidemic (HIV associated)

Clinical findings (of Immunodeficiency / Endemic type)

- presents as asymptomatic red macules (early)→ enlarge and become darker over time → lobulated surface with possible ulceration and pain (older lesions)
- color and surface variations exist
- single to multifocal
- lesions can be found extraorally, intraorally and in visceral organs
- about ½ of those with skin lesions will develop oral lesions as well
- extraorally, the tip of the nose is a common location for lesions
- intraorally, the palate is the most common site for lesions to be seen (also seen on the gingiva and tongue)

Treatment

- limited success- surgery, radiation, chemotherapy

Glossitis

⇒ Etiology

- mainly associated with vitamin deficiencies (B-vitamins, iron, protein)

Clinical findings

- presents as a red, bald, burning tongue due to a loss of the filiform papillae (atrophy)

Treatment

- identify etiology; replacement therapy

Leukemia

Etiology

- unknown etiology; is a malignant cancer of the blood-forming tissue characterized by defects in maturation and proliferation of WBCs and their precursors

Oral Manifestations of this and other Blood Dyscrasias (disorders) include:

- generalized gingival enlargement
- presence of petechiae and ecchymosis of the skin and mucous membranes

  Petechiae - rupture of capillaries that produces pinpoint blood hemorrhages; will not blanch

  Ecchymosis- subcutaneous bleeding that produces a non-raised lesion up to 1 cm in size; common bruise; extravasation (spillage) of blood; will not blanch
- "spontaneous" gingival hemorrhage
- prolonged bleeding following oral surgery
- delayed wound healing
- gingivitis refractory to treatment
- loose teeth
- mucosal ulcers
- candidiasis

⇒ Clinical findings

- acute and chronic forms of leukemia
- acute leukemia is more apt to display oral manifestations (80% of oral lesions
- are associated with acute form); majority of acute leukemia will be associated with children
- gingival hyperplasia represents the most common oral manifestation and may be first sign that the patient is having a problem
- any dental treatment that may produce trauma to the tissues may aggravate the underlying situation and give rise to exacerbation of acute symptoms that can
- result in death
- medical referral; no cure; may go into remission

Hematoma

⇒ "blood blister"

Hematoma- is a large area (> 1 cm) of subcutaneous bleeding that distends soft tissue

Etiology

- result of trauma to the soft tissue

Clinical findings

- presents as a flat or slightly raised, blue to purple lesion
- to help identify this lesion, obtain a thorough history; lesion also exhibits a negative diascopy
- some patients, especially those on anticoagulants, are more prone to developing hematomas

Pigmentations

Lecture Outline

1. Terminology
   - melanin
   - melanocytes
   - nevus cell
2. Influences on amount of pigmentation produced
3. Syndromes associated with oral and cutaneous macular pigmentation
   - Peutz-Jeghers syndrome
   - Addison's disease
   - Albright's syndrome
   - Neurofibromatosis
4. Physiologic pigmentation
   - etiology
   - clinical findings
5. Smoking associated melanosis
   - etiology
   - clinical findings
6. Oral melanotic macule
   - etiology
   - clinical findings
7. Nevi
   - developmental malformations
   - various types
   - clinical findings
   - treatment
8. Malignant melanoma
   - etiology
   - predisposing factors
   - clinical findings
   - early warning signs
   - treatment
9. Amalgam tattoo
   - etiology
   - clinical findings
   - radiographic finding
   - treatment
10. Clinical differentiation of oral pigmentations from early melanoma
    - Physiologic pigment
    - Melanotic macule
    - Nevi
    - Melanoma
    - Focal argyrosis

Lecture Objectives:

1. Define the following terms: melanocyte, nevus cell
2. List the etiology for physiologic pigmentation, nevi, Addison's disease, malignant melanoma, and amalgam tattoo.
3. List clinical findings associated with physiologic pigmentation, nevi, smoker's melanosis, Addison's disease, malignant melanoma, and amalgam tattoo.
4. List the predisposing factors that are associated with melanomas.
5. List clinical changes that may occur when a benign pigmentation undergoes a malignant transformation.
6. Identify various pigmented lesions when clinical information and / or slides are given.

Pigmented Lesions	**Color Atlas**
Physiologic Pigmentation	170
Nevi	172
Smoker's Melanosis	170 - 171
Malignant Melanoma	172 - 173
Amalgam Tattoo	170 - 171

PIGMENTATIONS

- **melanin** – any of a group of naturally occurring dark pigments, especially the pigment found in skin, hair, fur and feathers
- **melanocytes** – cells that produce melanin; are found throughout the oral cavity, but go unnoticed because of their relatively low level of pigment production; when localized or generally active, they may be responsible for physiologic pigmentation, neoplasia, etc….
- **nevus cell** – cell related to the melanocyte and is responsible for nevi and pigmented malignancies

The following will influence the amount of pigmentation produced:

- sunlight exposure
- hormones
- genetics

⇒ **Syndromes Associated with Oral and Cutaneous Macular Pigmentation**

Syndrome	Features
Peutz-Jeghers syndrome	Perioral ephelides, intestinal polyposis (not premalignant), autosomal dominant
Addison's disease	Diffuse cutaneous pigmentation, oral ephelides, adrenal cortical insufficiency
Albright's syndrome	Cafe'-au-lait macules, polyostotic fibrous dysplasia, precocious puberty
Neurofibromatosis	Cafe'-au-lait macules, oral and cutaneous neurofibromas (malignant potential); some inherited as autosomal dominant

Physiologic Pigmentation

- "melanoplakia"
- increased melanin within tissue
- more commonly noted in African American and Asian populations

Clinical findings:

- presents as persistent, symmetrical or asymmetrical, black / brown areas that exhibit no change in the gingival architecture
- found on any aged person
- most common to see on attached gingiva, but can also be found on the buccal mucosa, palate, tongue and lips

Smoking Associated Melanosis

- abnormal melanin pigmentation of the oral mucosa has been linked to cigarette smoking
- believed to be due to a component in the tobacco stimulates the melanocytes

Clinical findings:

- presents as increased amounts of intraoral pigmentation that typically noted in the anterior, labial gingiva
- more commonly noted in females
- intensity of the pigmentation is time and dose related

Oral Melanotic Macule

- focal pigmented lesion
  - freckle
  - macules associated with a syndrome

Clinical findings:

- presents as persistent, pigmented area that is typically noted on the vermillion border of the lips and the gingiva
- are asymptomatic and have no potential for being malignant
- if seen in excessive amounts, consider Peutz-Jeghers syndrome or Addison's disease

Nevi

⇒ "moles"

⇒ typically used to refer to a pigmented lesion composed of nevus cells or melanocytes

⇒ these are developmental malformations

- there are various types of nevi- this will relate to where the melanin is found within the tissue (depth) - epithelium, connective tissue, or junctional

Clinical findings:

⇒ presents as an asymptomatic, small (most are less than .5 cm in diameter), flat or slightly elevated pigmentation

⇒ present as tan / brown / black macules that are uniform in color and have sharply defined margins

⇒ most appear at birth or shortly afterwards; may appear during childhood

- are more commonly found on the skin

⇒ uncommon intraorally; if found, typical locations are the hard palate and buccal mucosa

Treatment:

- biopsy prn

Malignant Melanoma

Etiology:

- neoplastic transformation of nevus or melanocyte

Predisposing factors:

- sun exposure – UV light exposure has been established as a risk factor in the development of skin melanomas
- fair skin pigmentation
- skin pigmentations / precursor lesions (30% come from preexisting lesion)
- oral melanomas- there are no known predisposing factors; they may develop in clinically normal mucosa or within a pre-existing area of pigmentation

Clinical findings:

- melanomas are more commonly found on the skin
- primary lesions of the oral cavity are rare, but can come from a skin melanoma
- there are no racial predilections, but blacks and Asians appear to be proportionally more commonly affected

⇒ more commonly noted in adult (rare in children), most occurring in those over the age of 40

- males > females (2:1)

⇒ 70 -80% occur on maxilla- palate, gingiva, alveolar mucosa

- satellite lesions

⇒ Early signs:

- asymmetry
- irregular margins
- color changes
- size change

Treatment:

- surgery

Amalgam Tattoo

⇒ focal argyrosis / dyscromia

⇒ represents the most commonly found intraoral pigmentation

⇒ Etiology:

- iatrogenic lesion that is caused by the traumatic implantation of amalgam into soft tissue or a passive transfer by chronic friction of the mucosa against an amalgam restoration

⇒ Clinical findings:

- presents as an asymptomatic, flat, well defined, bluish-black macular pigmentation that
- will not exhibit any change in appearance with time
- typical locations include the gingiva, alveolar mucosa and buccal mucosa
- look for adjacent restorations
- if amalgam particles are of sufficient size, it may be verified via radiographs

Treatment:

- none

<u>Clinical Differentiation of Oral Pigmentations from Early Melanoma</u>

Physiologic pigment	Symmetric / asymmetric, not recently acquired, uniform color, no obstruction of normal landmarks
Melanotic macule	Uniform color, macular, relatively uniform contour
Nevi	Small, uniform color, congenital, uncommon intraorally
Melanoma	Recently acquired, variable color, irregular margins, satellite lesions, may be ulcerated or papular
Focal argyrosis	Uniform slate-gray color, history of dental “trauma”, macular, no change with time

Verrucal - Papillary Lesions

Lecture Outline

1. Denture stomatitis
   - etiology
   - clinical findings
   - treatment
2. Papillary hyperplasia
   - etiology
   - clinical findings
   - treatment
3. Squamous papilloma
   - etiology
   - clinical findings
   - treatment
4. Verruca vulgaris
   - etiology
   - clinical findings
   - treatment

Lecture Objectives:

1. Define the following terms: exophytic, sessile, pedunculated
2. Give the etiology for denture stomatitis, papillary hyperplasia, squamous papilloma, and verruca vulgaris.
3. List clinical findings associated with denture stomatitis, papillary hyperplasia, squamous papilloma, and verruca vulgaris.
4. List clinical findings that will distinguish verrucal papillary lesions apart.
5. Identify various verrucal - papillary lesions when clinical information and / or slides are given.

Verrucal Papillary Lesions	Color Atlas
Denture Stomatitis	168 - 169
Papillary Hyperplasia	168 - 169
Squamous Papilloma	182 - 183
Verruca Vulgaris	182 - 183

VERRUCAL - PAPILLARY LESIONS

Denture Stomatitis

- "denture sore mouth"; chronic atrophic candidiasis
- represents the most common form of chronic candidiasis

⇒ Etiology:

- chronic trauma from an ill-fitting removable prosthesis that promotes growth of candida albicans
- fungus colonization will cause inflammatory changes to the mucosa under the prosthesis

Clinical findings:

⇒ early lesion presents as red pinpoint areas of erythema

- as the irritation continues, the lesion progresses to diffuse erythema
- mucosa is erythematous with margins corresponding to the edges of the appliance
- more common to see on the maxillary arch
- most commonly noted in older females who wear their prostheses at night

Treatment:

- evaluate fit of prosthesis
- correct prn

Papillary Hyperplasia

- palatal papillomatosis
- represents a variation or progression of denture stomatitis

⇒ Etiology:

- associated with long term wearing of ill-fitting removable prosthesis that promotes the growth of candida albicans

⇒ Clinical findings:

- presents as multiple erythematous and edematous papillary projections on the hard palate (vaulted aspect)
- lesion is sessile and has a cobblestone appearance

Treatment:

- remove prosthesis (self heal), antifungal agents, surgical removal of lesion, new prosthesis

Squamous Papilloma

⇒ Etiology:

- believed to be a benign tumor (growth) of the squamous epithelium, but HPV may be involved; many have been shown to be associated with the same HPV that causes
- cutaneous warts
- mode of transmission in uncertain

Clinical findings:

⇒ lesion appears as an off-white, exophytic lesion

⇒ are asymptomatic, small, well-defined granular (pebbly) to cauliflower-like (finger-like) projection

- can be pedunculated or sessile
- typically occur as a solitary lesion, but can occur in clusters

Treatment:

- surgical excision

Verruca Vulgaris

- common wart that occurs on the skin

Etiology:

- human papilloma virus (HPV)
- intraoral v. vulgaris usually develops as a result of transmission of infection involving HPV type 2 and 4 from warts on the hands and fingers. May also be due to oral contact with venereal warts (HPV type 6, 11, 60)

⇒ Clinical findings:

- has a similar in appearance as that of squamous papilloma
- lesion is pink to whitish in color
- surface of lesion is rough and lesion is raised with finger-like projections
- lesions on skin are more commonly noted that intraoral lesions

Treatment:

- surgical removal
- laser removal

Connective Tissue Lesions

Lecture Outline

1. Gingival hyperplasia
   - etiologies
   - clinical findings
     - initial presentation
     - progressive changes
     - variations between medications
   - treatment
2. Fibroma
   - etiology
   - clinical findings
   - treatment
3. Epulis fissuratum
   - etiology
   - clinical findings
   - treatment

Lecture Objectives:

1. List the etiology associated with gingival hyperplasia, traumatic fibroma and epulis fissuratum.
2. List clinical findings associated with gingival hyperplasia, traumatic fibroma and epulis fissuratum.
3. Identify various connective tissue lesions when clinical information and / or slides are given.

Connective Tissue Lesions	Color Atlas
Gingival Hyperplasia	104 - 105
Traumatic Fibroma	180 - 181
Epulis Fissuratum	112 - 113

CONNECTIVE TISSUE LESIONS

⇒ **Gingival Hyperplasia**

- drug-induced hyperplastic enlargement of the gingiva

⇒ Etiology:

- most cases are a hyperplastic tissue response to chronic inflammation associated with local factors – plaque, calculus, bacteria
- hormonal changes (puberty and pregnancy) can significantly exaggerate the effects on local factors

⇒ unknown etiology, but reflects an enlargement of the gingiva resulting from systemic medications, most commonly anticonvulsants, calcium channel blockers and immunosuppressants (phenytoin, cyclosporine, and certain calcium channel blockers)

- onset within 3 months of taking medication

Dilantin- (used in the management of epilepsy) is the most widely recognized medication associated with fibrotic overgrowth of the gingival tissues; affects 50 % of those taking this medication. The extent or severity of the gingival enlargement that is associated with dilantin will depend on the presence of local factors. The effect of time and dose of drug taken and how it will affect the gingival tissues is not clear.

Cyclosporine- (immunosuppressant drug that is used to help against rejection of transplants) affects 10 – 70 % of those on this medication

⇒ *Calcium channel blockers*- (Verapamil, procardia) used in the management of hypertension, angina, and arrythmias) affects 1 – 10 % of those on these medications
Estrogen, (BCPs, premarin)- affects 1 – 10 % of those on these medications

Clinical findings:

⇒ in all forms of medication-related gingival hyperplasia, there will first be an enlargement of the interdental papilla gingiva

- marginal and attached gingiva will gradual become involved over time
- typically noted on labial aspect of tissues with the gingiva in the anterior sextants being most commonly affected
- initially, the gingiva is red, lumpy and bleeds easily
- as progressive growth of the tissue occurs, the results are demonstrated by fibrotic changes within the gingival tissues
  - the interdental papillae becomes enlarged, pink, firm and resilient to palpation
  - rolled margins become evident, there will be a loss of stippling and little to no tendency to bleed
- no attachment loss is evident

- variations in the clinical appearance can occur with different medications

  Dilantin
  - gingiva is pink, firm and rubbery (due to increased collagen content)

  Calcium channel blockers
  - from red to pink, firm to spongy

  Cyclosporine
  - soft, red, fragile tissue that easily bleeds

- exaggerated inflammatory response in relation to the plaque present
- plaque accumulation is not necessary for the initiation of gingival enlargement, but it will exacerbate the gingival disease

Treatment:

- improve oral hygiene; meticulous plaque control can reduce, but will not eliminate
- gingival overgrowth
- tissue re-contouring

Fibroma

⇒ "traumatic / irritation fibroma"

⇒ Etiology:

- fibrous hyperplasia (fibrous connective tissue) that is believed to be caused by acute and / or chronic trauma

⇒ Clinical findings:

- presents as a well-defined, localized, round, smooth surface, solitary mass
- sessile lesion is asymptomatic, firm to rubbery when palpated and pale in color
- located in areas that are accessible to trauma
- surface of the lesion may be ulcerated if recently traumatized

Treatment:

- surgical excision

Epulis Fissuratum

⇒ "denture or irritation hyperplasia"

⇒ Etiology:

- tissue reaction caused by chronic trauma and is associated with the flanges of an ill-fitting removable prosthesis
- ill-fitting denture acts as a source of chronic trauma → an ulcer is the initial lesion that is produced → the traumatized tissues heal by forming hyperplastic tissue with excessive collagen

⇒ Clinical findings:

- presents as an asymptomatic, firm to flabby, pink to red, hyperplastic fold of tissue
- lesion will be located at the peripheral borders (flanges) of the prosthesis
- lesion is most commonly found in the maxillary anterior region and can cause the resorption of the underlying bone
- most commonly noted in older females that do not remove their dentures

Treatment:

- surgical excision and reline/remake prosthesis

Miscellaneous Lesions

Lecture Outline

1. Ameloblastoma
   - etiology
   - clinical and radiographic findings
2. Cementoma
   - etiology
   - clinical findings
   - radiographic findings and changes as lesion ages
3. Odontoma
   - etiology
   a. Compound odontoma
      - location and radiographic appearance
   b. Complex odontoma
      - location and radiographic appearance
4. Acromegaly
   - etiology
   - clinical findings
5. Malignant neoplasms of the jaws
   - primary lesion vs. metastatic lesion
   - presenting signs and symptoms
   - radiographic appearances

Lecture Objectives:

1. Describe the clinical findings associated with an ameloblastoma and acromegaly.
2. List the etiology for a cementoma and odontoma.
3. Describe the clinical findings associated with a cementoma and odontoma.
4. Identify these lesions when clinical information and / or slides are given.

   Cementoma, odontoma
5. List the variations in the radiographic appearances for malignant neoplasms of the jaws.
6. List clinical findings that are associated with malignant neoplasms of the jaws.

Misc. Lesions	**Color Atlas**
Ameloblastoma	88 - 89, 90 - 91
Cementoma	80
Odontoma	92, 93

MISCELLANEOUS LESIONS

Ameloblastoma

⇒ is a benign tumor that will originate from one of the following:

- dental lamina
- enamel organ
- cyst epithelium
- dentigerous cyst
- primordial cyst

Clinical findings:

⇒ 80% of ameloblastomas will be found in the MD molar / ramus region

- radiographically, it appears as a well-defined RL lesion and can be either unilocular or multilocular
- locally aggressive tumor that expands involved bone

Cementoma

⇒ "periapical cemental dysplasia"

- lesion is believed to be caused by trauma or infection to the involved tooth

⇒ Clinical findings:

- presents as an asymptomatic lesion that is found during radiographic examination
- occurs on the apex of vital teeth and is most commonly found on mandibular anterior(s)
- radiographically, 3 different stages are noted:
  - radiolucent → mixed radiolucent / radiopaque → radiopaque with radiolucent ring

Odontoma

- represents a disturbance during tooth development, trauma or infection having a role
- are noted during a radiographic evaluation
- more common to see in the maxilla verses the mandible

⇒ 2 types of Odontoma:

⇒ 1. Compound Odontoma:

- found in the anterior region of the mouth and appear radiographically as several little teeth in a localized area

⇒ 2. Complex Odontoma:

- found in the posterior region of the mouth and appear radiographically as an opaque mass in a localized area

Acromegaly

- refers to a hypersecretion of growth hormone that is typically caused by a benign pituitary tumor
- in a person with acromegaly, several clinical findings are noted and these can include enlargement of jaw bones, development of class III malocclusion, large hands and a coarsening of the facial features

Malignant Neoplasms of the Jaws

- finding a malignancy in the maxilla or mandible is rare
- these type of neoplasms can be a primary (starts in the jawbone) lesion or a metastatic (originates outside the jawbones and spreads to the jawbone) lesion
- if it is a metastatic lesion, sites for the primary lesion (site of origin) include:
  - breast, kidney, lung, thyroid, prostate, colon
- presenting signs and symptoms can include pain, swelling, lip paresthesia and root resorption
- radiographic appearances of these malignant lesions can vary; typical appearances include a moth-eaten radiolucent, an irregular appearance, radiopacity, mixed radiopaque / radiolucent and a sunray effect

Salivary Gland Lesions

Lecture Outline

1. Sialolith
   - etiology
   - clinical findings
   - radiographic findings
   - treatment
2. Mucocele
   - definition
   a. Mucous extravasation phenomenon
      - etiology
   b. Mucous retention cyst
      - etiology
   - clinical findings(for either type)
   - treatment
3. Ranula
   - etiology
   - clinical findings
   - treatment
4. Salivary gland neoplasms
   a. Pleomorphic adenoma
   b. Mucoepidermoid carcinoma

Lecture Objectives:

1. Define the term mucocele.
2. Compare between a mucous extravasation phenomenon and a mucous retention cyst.
3. Give the etiology for the following lesions:

   Sialolith, mucous extravasation phenomenon, mucous retention cyst, ranula, pleomorphic adenoma
4. List clinical findings that are associated with the following diseases:

   Sialolith, mucous extravasation phenomenon, mucous retention cyst, ranula, pleomorphic adenoma
5. Identify various salivary gland lesions when clinical information and / or slides are given.
6. List the salivary glands that are most affected by neoplasms.
7. List the salivary glands that are more frequent to give rise to malignant tumors.

Salivary Gland Diseases	**Color Atlas**
Sialolith	138 - 139
Mucocele	134 - 135, 138 - 139
Ranula	138 - 139
Pleomorphic Adenoma	142 - 143
Mucoepidermoid Carcinoma	142 - 143

SALIVARY GLAND DISEASES

Sialolith

- "salivary stone"

Etiology:

⇒ a calcified mass or stone that can develop within a salivary gland or duct; they arise from calcium deposits around a core of bacteria, mucous or ductal epithelial cells

- causes ductal blockage

Clinical findings:

- presents as a rapidly appearing, painful swelling of the involved gland
  pain will increase at mealtimes
- if the blockage is at the orifice (opening) of the ductal system, the opening will show signs of inflammation
- 80% of the cases involve the submandibular gland
- radiographically, 90 % of the cases appear radiopaque; if found to involve the Parotid gland, 90 % of the time, it will be radiolucent

Treatment:

- none
- stone removal

Mucocele

- refers to the accumulation of mucous within the connective tissue or within a salivary gland duct- there are 2 types of mucoceles and both are clinically referred to as a mucocele

⇒ 2 types of mucoceles:

1. Mucous Extravasation Phenomenon
2. Mucous Retention Cyst

Mucous Extravasation Phenomenon

⇒ Etiology:

- caused by mechanical trauma to a minor salivary glands' excretory duct
- trauma causes the duct to rupture and will result in the spillage of mucous into the surrounding tissue
- body walls off the spilled mucous with granulation tissue and a "pseudocyst" results

Mucous Retention Cyst

⇒ Etiology:

- duct is obstructed or blocked by a salivary stone
- blockage will create a true cyst (because the stone is lined or walled off by epithelium)
- in 80 % of the cases, the submandibular gland is most commonly affected; the remaining are in the parotid gland, with a very small percentage being involved with the sublingual and minor glands

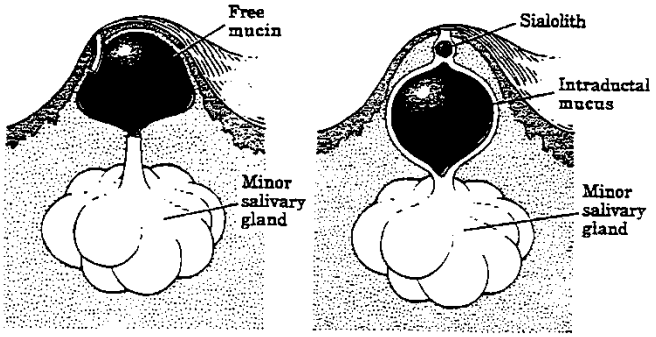


Figure 8–1. *Left*, Mucus extravasation phenomenon—note severed duct at upper left. *Right*, Mucus retention cyst.

⇒ Clinical findings (for either type):

- presents as suddenly appearing, painless, fluctuant swelling or solitary mass
- swelling is fluid-filled, spherical and has a smooth surface
- color of the lesion will vary (translucent to normal tissue color) and this will vary depending on the depth of the lesion
- most commonly found on the lower lip, between the midline and commissure
- tend to be recurrent

Treatment:

- excision / removal of associated minor salivary gland

Ranula

- "little frog"
- variation of a mucocele that occurs in the floor of the mouth

⇒ Etiology:

- trauma or ductal obstruction associated with the sublingual and submandibular salivary glands

⇒ Clinical findings:

- presents as an asymptomatic, unilateral, soft tissue mass that is located in the lateral
- aspect of the floor of mouth
- lesion is bluish-white, translucent, fluctuant and smooth
- when large, it can push the tongue upwards

Treatment:

- surgery, marsupialization

Neoplasms of the Salivary Glands

- both major and minor salivary glands can give rise to neoplasms, but the major glands are most commonly affected
- submandibular and minor salivary glands have a greater tendency of being malignant
- most salivary gland neoplasms arise from epithelial tissue

Pleomorphic Adenoma

⇒ "Benign Mixed Tumor"

- represents the most common salivary gland tumor

⇒ most will occur within the parotid gland

Clinical findings:

- typical: firm, painless swelling at the angle of the mandible

Mucoepidermoid Carcinoma

- represents a malignant tumor of salivary gland origin

Cysts

Lecture Outline

1. Cysts
   - definition
   - issues associated with cyst enlargement
2. Odontogenic cyst
   - definition
   a. Granuloma
      - etiology
      - radiographic and clinical findings
      - treatment
   b. Radicular cyst
      - etiology
      - radiographic and clinical findings
      - treatment
   c. Residual cyst
      - etiology
      - radiographic and clinical findings
      - treatment
   d. Primordial cyst
      - etiology
      - radiographic and clinical findings
      - treatment
   e. Dentigerous cyst
      - etiology
      - radiographic and clinical findings
      - treatment
   f. Lateral periodontal cyst
      - etiology
      - radiographic and clinical findings
      - treatment
3. Non-odontogenic cysts
   a. Globulomaxillary cyst
      - etiology
      - radiographic and clinical findings
      - treatment
   b. Nasopalatine canal cyst
      - etiology
      - radiographic and clinical findings
      - treatment

Lecture Objectives:

1. Define the following terms: cyst, odontogenic, non-odontogenic
2. List the etiology for the following cysts:

   Granuloma, Radicular cyst, Residual cyst, Primordial cyst, Dentigerous cyst, Lateral periodontal cyst, Globulomaxillary cyst, Nasopalatine canal cyst
3. List clinical findings that are associated with the following cysts:

   Granuloma, Radicular cyst, Residual cyst, Primordial cyst, Dentigerous cyst, Lateral periodontal cyst, Globulomaxillary cyst, Nasopalatine canal cyst
4. List radiographic findings that are associated with the following cysts:

   Granuloma, Radicular cyst, Residual cyst, Primordial cyst, Dentigerous cyst, Lateral periodontal cyst, Globulomaxillary cyst, Nasopalatine canal cyst
5. Identify various cystic lesions when clinical information and / or slides are given.
6. Describe the method of treatment for the following cysts:

   Granuloma, Radicular cyst, Primordial cyst, Dentigerous cyst
7. List complications that may arise from the following cysts:

   Primordial cyst, Dentigerous cyst

Cysts	**Color Atlas**
Granuloma	90 - 91
Radicular Cyst	85
Residual Cyst	85
Primordial Cyst	83
Dentigerous Cyst	82 - 83
Lateral Periodontal Cyst	87
Globulomaxillary Cyst	87
Nasopalatine Cyst	141

CYSTS

Cyst

⇒ defined as an epithelial lined pathologic cavity that may contain fluid or cellular debris

⇒ Cyst Enlargement- when a cyst enlarges, this can create additional consequences

- root resorption of an involved or adjacent tooth / teeth
- displacement (movement) of a tooth
- bone resorption
- perforation of the outer surface of the bone where the cyst is located

Terminology:

⇒ Odontogenic cyst

- cyst that has a dental origin (involved with a tooth)

⇒ Non-odontogenic cyst

- cyst of non-dental origin; arise from an area in the soft tissue

Odontogenic Cysts

Granuloma

- represents the most commonly found periapical radiolucency

⇒ Etiology:

- due to chronic pulpal irritation or necrosis
- as the toxins from the dead tooth are emptied into the periapical tissues, the body walls off the toxins

Clinical findings:

⇒ radiographically, the lesion will appear as a well-defined radiolucent lesion at the periapical region of the involved tooth

- clinically, the involved tooth will have a deep restoration, caries, a fracture or a history of trauma

⇒ involved tooth is non-vital

- involved tooth will be asymptomatic (no hot/cold sensitivities, no pain with percussion)

Treatment:

- root canal or extraction

Radicular Cyst

Etiology:

- lesion originates in a pre-existing periapical granuloma
- a cell change occurs within a granuloma that constitutes it being a radicular cyst

⇒ Clinical findings:

- findings are the same to those found with a granuloma
- histologic evaluation will distinguish between the two lesions

Treatment:

- root canal or extraction

Residual Cyst

⇒ Etiology:

- due to a periapical cyst not being removed or curetted after the involved tooth was removed

Clinical findings:

- radiographically, the cyst appears as a well-defined, rounded radiolucent lesion in the apex region, but no tooth is present

Treatment:

- surgical removal

Primordial Cyst

⇒ Etiology:

- cyst originates from the remnants of a tooth bud
- cyst develops in place of a tooth

Clinical findings:

- radiographically, it appears as a well-defined radiolucent lesion; it can be unilocular or multilocular
- patient will give a history that the "involved" tooth failed to erupt
- most commonly seen to "involve" the 3rd molars

Treatment:

- surgical removal

Dentigerous Cyst

⇒ "follicular cyst"

⇒ Etiology:

- although it has an unknown etiology, it originates from the epithelium that is associated with tooth development

Clinical findings:

⇒ radiographically, the lesion appears as a well-defined, unilocular radiolucency that is associated with the crown of an unerupted tooth

⇒ this cyst is associated with the crown of unerupted or developing tooth

- the lesion is asymptomatic and may slowly expand the bone in the immediate area

⇒ most commonly involves maxillary and mandibular 3rd molars and maxillary canines and late eruption is questioned

Treatment:

- tooth removal, marsupialization

Lateral Periodontal Cyst

Etiology:

- unclear etiology, but is believed to be a proliferation of the remnants associated with the dental lamina or epithelial rests of Malassez

Clinical findings:

⇒ radiographically, the lesion appears as a well-defined radiolucent lesion on the lateral surface of a root

⇒ most commonly seen in the mandibular canine and premolar region

⇒ cyst is asymptomatic, occurs in the middle third of the root on the involved tooth

- adjacent teeth are vital

Treatment:

- excision

Non-Odontogenic Cysts

Globulomaxillary Cyst

⇒ Etiology:

- believed to originate from epithelial cells that were trapped during palatal fusion

Clinical findings:

⇒ cyst occurs between the maxillary lateral and canine

- radiographically, the lesion appears as a well-defined radiolucency that has an inverted pear-shape; the cyst may cause root divergence and / or expand the cortical plate
- adjacent teeth are vital

Treatment:

- cyst removal
- curettage

Nasopalatine Canal Cyst

⇒ "incisive canal cyst"

⇒ Etiology:

- rises from epithelial cells that were trapped during palatal development

⇒ Clinical findings:

- radiographically, the lesion appears as a well-defined radiolucent lesion that has a "heart" shape; the cyst can produce root divergence or root resorption
- on occasions, this cyst may appear over the root apices- in these cases, the need to distinguish it from a granuloma or radicular cyst becomes apparent
- clinically, a symmetrical swelling at the anterior, palatal midline may be seen the adjacent teeth are vital

Treatment:

- cyst removal