

**FINAL
MOA FALL LECTURE**

The background of the entire page is a photograph of a sunset over a body of water. On the left, a tall, dark lighthouse stands prominently. In the foreground, the silhouettes of several people are visible, some standing and some walking along a path or pier. The sky is a vibrant mix of orange, red, and yellow, with some clouds catching the light. The water is dark, and a few sailboats are visible in the distance.

Cellulitis, What you Think you Know

Friday October 25, 2024

9:30-10:30 am

MOA Annual Autumn Conference

October 25-27, 2024

L.V. Eberhard Center
301 West Fulton Street
Grand Rapids, MI 49504

ANTHONY OGNJAN DO FACD

Metro Infectious Disease Consultants

43134 Dequinder Rd

Sterling Heights Mi 48314

Phone: 586 446-8688

Fax: 586 446-9994

MIDC Metro
Infectious Disease
Consultants, L.L.C.

Anthony Ognjan, D.O., FACP

Serbo-Croatian - French Canadian



Diploma. Algonac High School (Long time ago!)

B.S. Microbiology & Public Health MSU 1975

B.S. Pharmacy, Ferris State College 1978

MSU College of Osteopathic Medicine 1983

Board Certified IM: 1987 [ABIM]

Board Certified Infectious Diseases: 1991 [ABIM]

Past President, Macomb County Osteopathic Medical Association

Chairman, Michigan Osteopathic Association [MOA] Political Action Committee

Member, Counsel of Governmental Affairs (MOA)

Vice-Chair MOA House of Delegates (MOA)

Associate Professor of Medicine MSU-COM

Hospital Orderly: (Orthopedics), Ingham Medical Center

Pharmacist: Children's Hospital of Michigan

Internship: Detroit Osteopathic Hospital / BCCH

Resident: Internal Medicine, Henry Ford Hospital, Detroit

Infectious Disease Fellowship: Henry Ford Hospital, Detroit

Work experience

- ❖ Staff Emergency room
Physician
Henry Ford hospital System
- ❖ Staff physician HIV/ID Clinic
Macomb County Jail
- ❖ Staff physician
Wound Care Center
McLaren Macomb hospital

1989 McLaren Macomb Hospital / Ascension Macomb-Oakland



A few personal observations ...

Who is
is this
guilty
way?

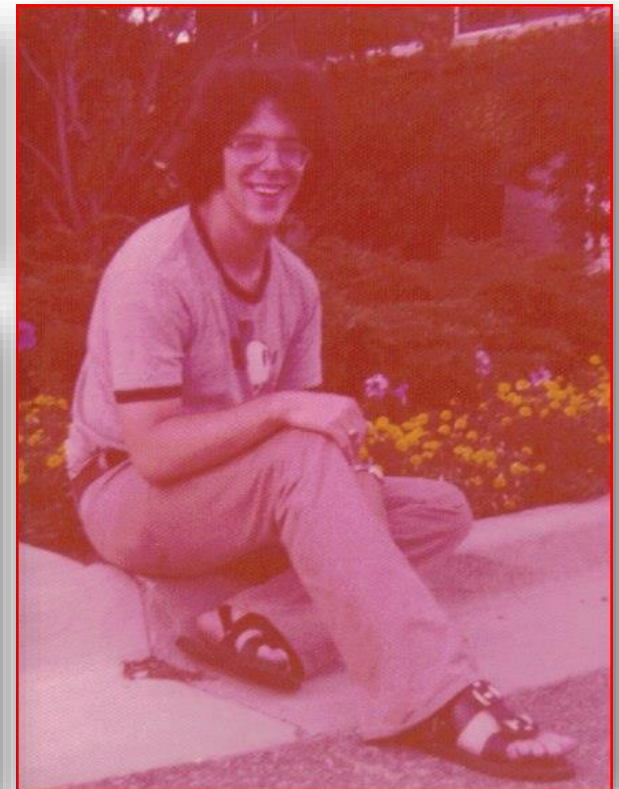
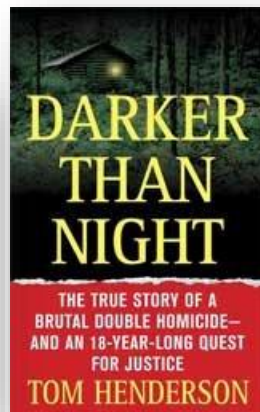


What is the QAAP tax in Michigan?

A QAAP is a process authorized by the Legislature that levies an assessment on Physician service to, leverage additional federal dollars into the Medicaid program, theoretically allowing higher reimbursement rates to be paid when providing services to Medicaid recipients



Michigan state capital steps
"Norma Rae" [1979 Sally Fields]





DISCLOSURES

Anthony Ognjan, DO FACP

- ❖ ***No Financial or corporate Relationships***
- ❖ ***1993: MOA Education Committee (“Hotel California”)***
- ❖ ***Greater than 25 years “Wound Care Clinic” Staff physician***
- ❖ ***Irony: Began 1983 HIV Pandemic - Ending with COVID-19 Pandemic***

I am a “Vaxer”: I encourage ALL Vaccines

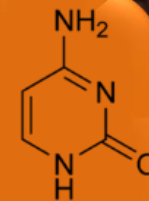
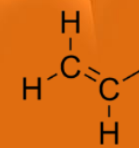
I don't believe in Mandatory COVID-19 Vaccination.

Major Objectives

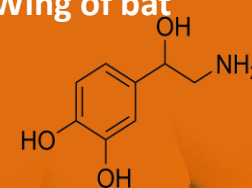
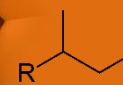
I intend Not to be boring ..

Offering a flowing presentation with some useful clinical pearls

Cellulitis,
What you Think you Know



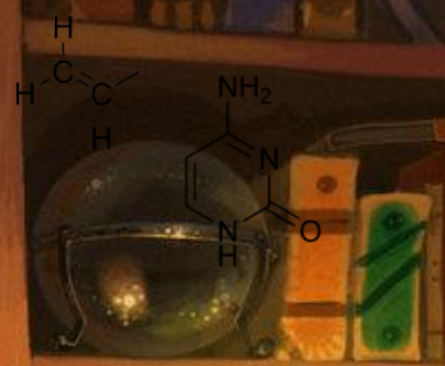
Eye of Newt...
Wing of bat



Major Objectives

1

Cellulitis, What you Think you Know



1. Increase knowledge and understanding of selected Soft tissue infections and diseases
 2. Improving the understanding of applicable skin and soft tissue anatomy, defining and exploring various soft tissue infections and pathology
 3. Explore Clinical pathology utilizing slide presentation featuring varies types of soft tissue Syndromes and infection infections
 4. Using illustrative case studies and summaries as applicable
5. **Brief overview of clinical medical issues associated with cellulitis and soft tissue infections**
- Microbiology
 - Antibiotics
 - Circulation
 - Miscellaneous : trauma, inflammatory issues



CELLULITIS

What you think you know

PEARLS



Lecture Design

- The lecture can be best appreciated by watching and listening to the overall narrative...
- There will be points along the way, where clinical **“Pearls”** may emphasize specific points of interest

Let the GAMES
begin....



I know....

I talk fast...

I have too many slides...

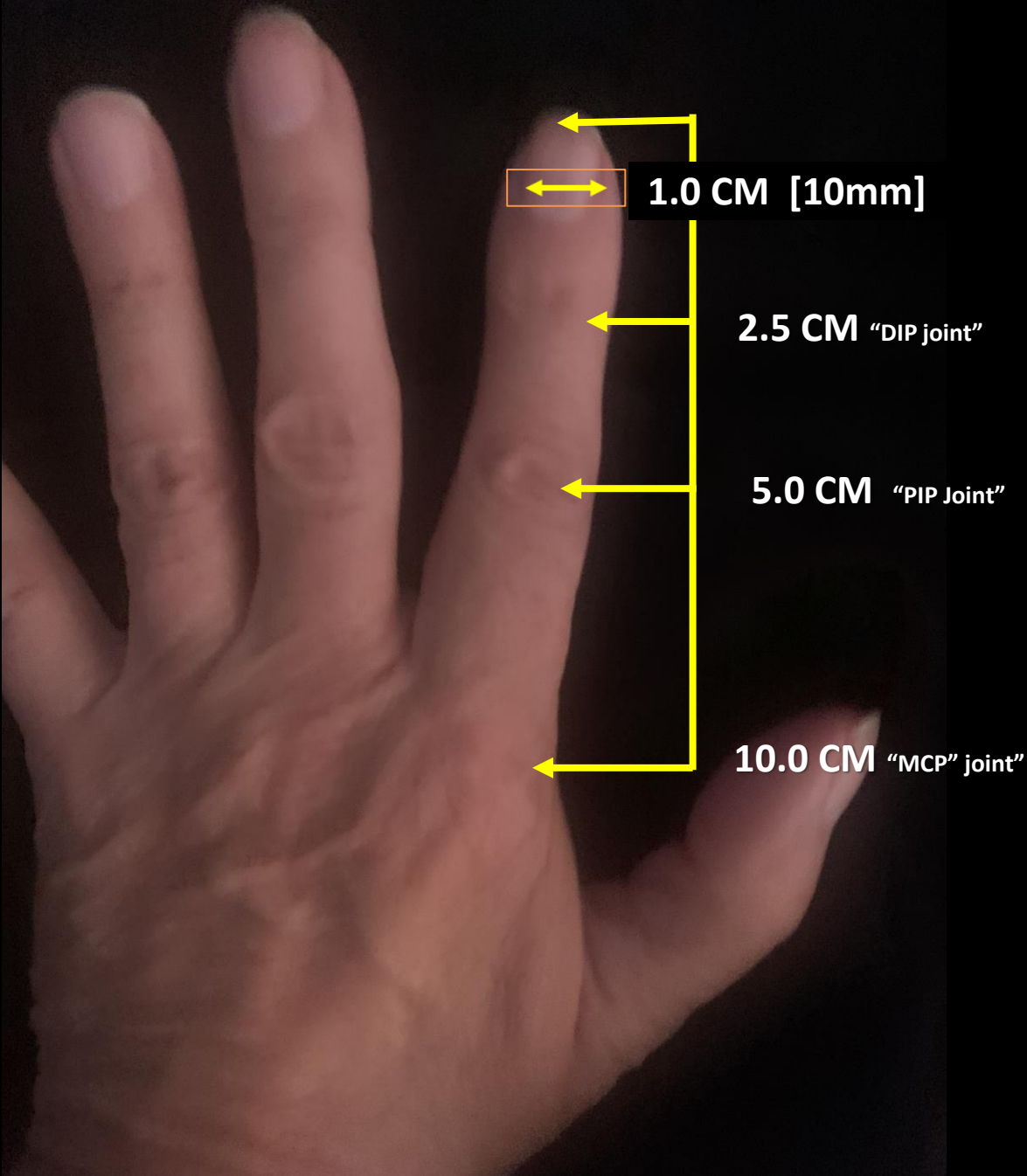
Respectfully of your attention....

Buckle Up.....Here we go....



Cellulitis

"The Ognjan ruler"





A. Rubin
Roulette

Dystrophic Epidermolysis Bullosa



Epidermolysis Bullosa

- ❖ Inherited connective tissue disease
- ❖ Blisters of the skin and mucosal membranes
- ❖ Incidence of 1 / 50,000.

Result of a defect in “anchoring” between the epidermis and dermis

resulting in friction and skin

Severity ranges from mild to lethal.





Right Arm

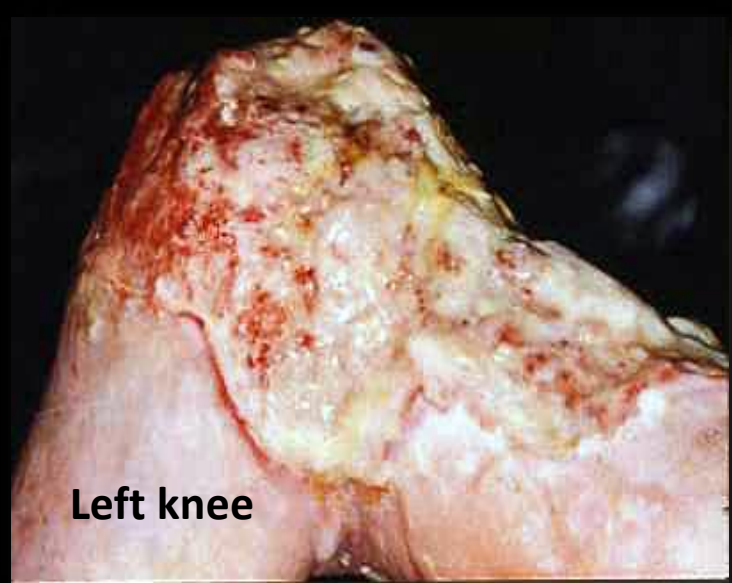


Left Arm

Mr. Paquette was a talented, prolific, and very successful watercolor artist, known through out the Great Lakes region for great lake landscapes



Left Leg



Left knee



Jonathan “Jonny” Kennedy

Born Alnwick, United Kingdom, spent most of his life in neighbouring Northumberland.

He had a severe form of dystrophic epidermolysis bullosa and was born with no skin on his left leg. Ultimately died age 36 from complications of skin cancer.

Jonny Kennedy

(10/04/1966 – 09/26/2003)

“The Boy Whose Skin Fell Off”,

Documented the final months of his life
Dystrophic Epidermolysis Bullosa .

<https://www.youtube.com/watch?v=OUWqyQYkZrg>

Re-Accessed 10/15/2024

CELLULITIS

What you think you know

RULES / Observations



Cellulitis??...Antibiotics??

RULES?



DO YOU THINK SO?

DON'T NEED NO
STINKING RULES".....

Alfonso Bedoya
"The Treasure of the Sierra Madre"

Ognjan

#1 Rule

“I never intend, to Die
from a Contagious
Infectious Disease”

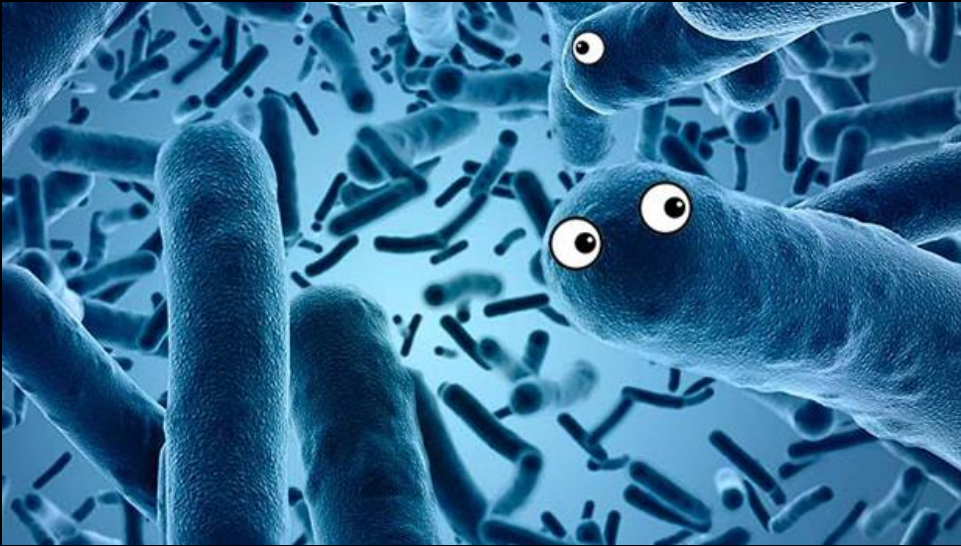
*Anthony Ognjan, DO FACP
Infectious Disease*

AO-9



#2 Rule

Bacteria Are Not BUGS!!!



Any Questions?

Cellulitis

Human / Bacterial Ecology

Bacteria with EMERGING β -LACTAM RESISTANCE



GRAM POSITIVE

(Skin, Mucus Membranes, Soft tissues)

MICROCOCCUS

- ❖ **S. aureus** (MSSA, MRSA, VRSA, CA-MRSA)
- ❖ S. epidermidis
- ❖ Micrococcus

STREPTOCOCCI

- ❖ **S. pyogenes** (β Streptococci)
- ❖ **S. agalactae** (β Streptococci)

- ❖ Pneumococcus
- ❖ Enterococcus (GDE, VRE)

- α Streptococci
- ❖ Viridian streptococcus gr.

Anaerobic Bacteria:

- ❖ Coccus, Bacillus, Spirochetes

GRAM NEGATIVE

(Enterobacteriaceae, GI tract)

FIVE TRIBES



- I. E. Coli
- II. Salmonella, Shigella
- III. Proteus, Morganella
- III. Klebsiella Enterobacter**
- Serratia (KES)*** →
- V. Yersinia

H. influenza

- ❖ H. Influenza β ("HIB")

ANAEROBIC

- ❖ Bacteroides fragilis
(β -lactamase)

GRAM NEGATIVE

Environmental

- ❖ P. aeruginosa
- ❖ Aeromonas spp.
- ❖ Acinetobacter spp.



MISCELLANEOUS

[Resistance]

"S.P.I.C.E." BACTERIA*

- ❖ Serratia
- ❖ Pseudomonas
- ❖ Indole-positive Proteus
- ❖ Citrobacter
- ❖ Enterobacter

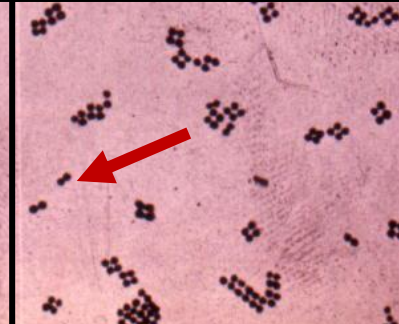
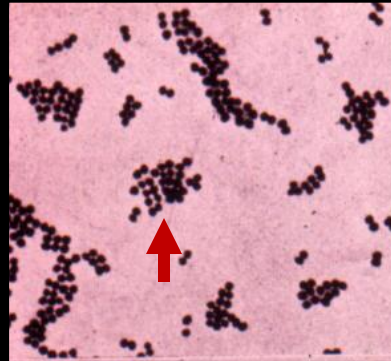
Cellulitis

Bacteriology

GRAM POSITIVE ORGANISMS

Staphylococcus
(Micrococcus)
"Clusters"

Staphylococcus
(Micrococcus)
"Tetrads"



Streptococcus
"Chains"

Streptococcus
"Diplococcus"

Micrococcus

Staphylococcus

Streptococcus

α β γ



GRAM POSITIVE CLUSTERS



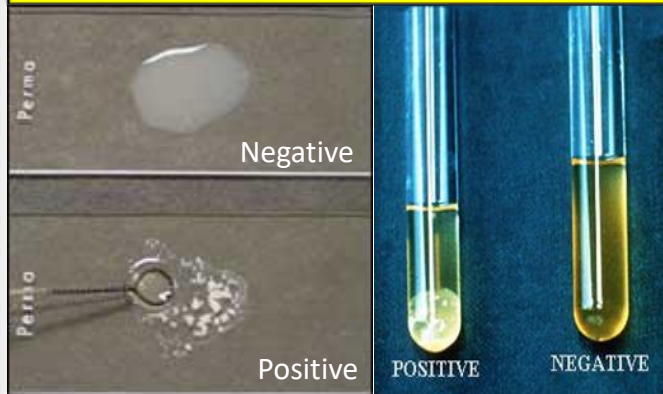
MICROCOCCUS

Micrococcus species

Staphylococcus aureus
"Gold"

Coagulase Negative
Staphylococcus
(Albus) "White"

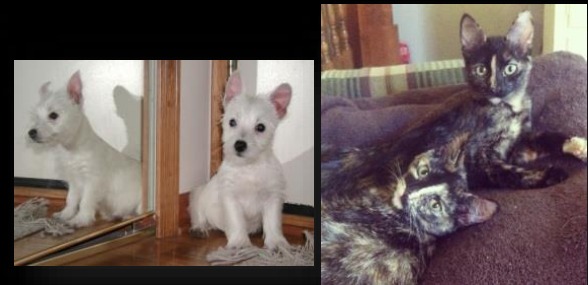
COAGULASE + / -
Slide Test Tube



**COAGULASE
"POSITIVE"**



**COAGULASE
"NEGATIVE"**





Streptococcus Taxonomy

Lancefield's group classification, introduced in 1933,

Based upon the carbohydrate composition of bacterial antigen*

Rebecca Craighill
Lancefield
1895-1981



| Beta Hemolysis β | Alpha hemolysis “Viridian Group” α | Gamma Hemolysis (Enterococcus) γ |
|--|---|---|
| S. pyogenes (Group A)* “GAS” S agalactiae (Group B) “GBS” | S. millerii S. mutans S. mitor S. sanguis (H) S. mitus S. salavarius (K) S. mutor S. anginosis (F,G) | S. bovis (D) S. equis S. durans S. cannis S. susis <hr/> E. faecalis E. faecium (VRE) |




*Complex sugar molecule “Polysaccharide”: C substance, major cell wall component in all streptococci

- ❖ “Groups” are designated by the letters **A through O**
- ❖ The most common species causing human disease: **Streptococcus pyogenes**: “Group A”.
- ❖ Among the group A streptococci, Lancefield found another antigen:
“M protein” - matt appearance in colony formations.

Cellulitis

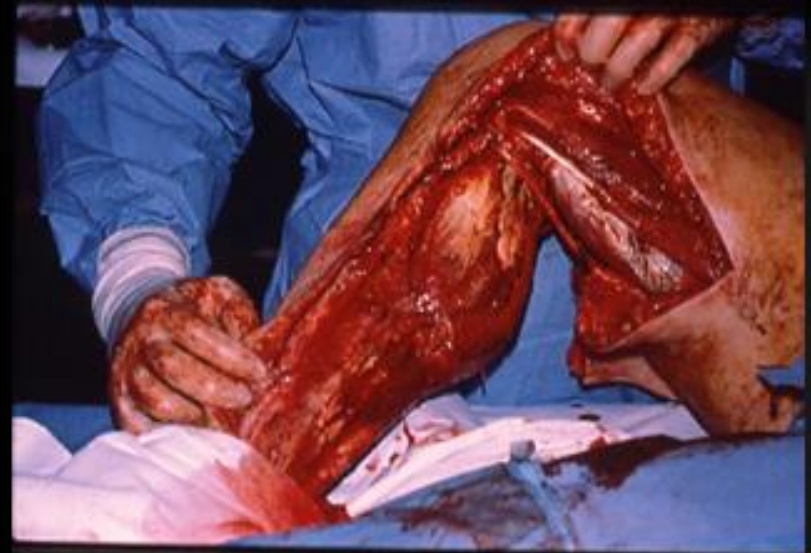
Synergistic Gangrene



| GRAM POSITIVE (Skin, Mucus Membranes, Soft tissues) | GRAM NEGATIVE (Enterobacteriaceae, GI tract) | GRAM NEGATIVE Environmental |
|--|---|--|
| MICROCOCCUS ❖ S. aureus (MSSA, MRSA, VRSA, CA-MRSA) ❖ S. epidermidis ❖ Micrococcus | FIVE TRIBES  I. E. Coli II. Salmonella, Shigella III. Proteus, Morganella III. Klebsiella Enterobacter Serratia (KES)* → V. Yersinia | ❖ P. aeruginosa ❖ Aeromonas spp. ❖ Acinetobacter spp.   |
| STREPTOCOCCI ❖ S. pyogenes (β Streptococci) ❖ S. agalactiae (β Streptococci) ❖ Pneumococcus ❖ Enterococcus (GDE, VRE) α Streptococci ❖ Viridian streptococcus gr. | H. influenza ❖ H. Influenza β ("HIB") ANAEROBIC ❖ Bacteroides fragilis (β-lactamase) | MISCELLANEOUS [Resistance] "S.P.I.C.E." BACTERIA* ❖ Serratia ❖ Pseudomonas ❖ Indole-positive Proteus ❖ Citrobacter ❖ Enterobacter |
| Anaerobic Bacteria: ❖ Coccus, Bacillus, Spirochetes | | |

Synergic gangrene

A Progressive bacterial synergistic mixed bacterial infection that usually occurs at the site of abdominal or thoracic surgery. It's typically caused by microaerophilic streptococci, Staphylococcus aureus, and Enterobacteriaceae species



Cellulitis

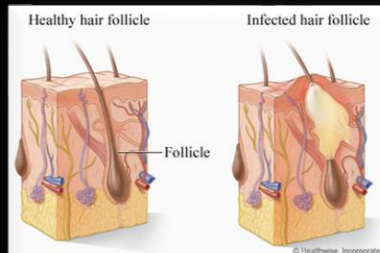
Folliculitis and Furunculosis [*Pseudomonas aeruginosa*]



Hot tub Folliculitis

Pseudomonas Folliculitis

“Hot tub buns”




37 year old home builder. With peri-rectal abscess. Hospitalized and underwent I&D.

Recommended sitz bath to the area post discharge...

Instead had one of his building crews build a \$12,000 Hot tub at his home.

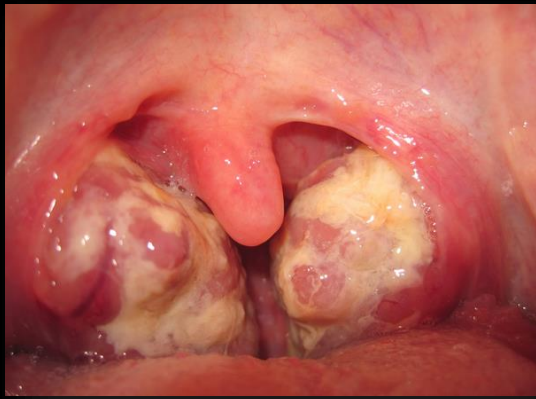
Shared tub with other family members; Not so good at maintenance of the tub....



Ognjan

#3 RULE

"If it weren't for Pus....
I wouldn't HAVE a job..."



Paronychia



“Pus”:

- A protein-rich fluid called “Liquor puris”: Whitish-Yellow, Yellow, or Yellow Brown in color.
- Pus consists of a buildup of Dead leukocytes (White Blood Cells) from the body's immune system in response to infection.
- It accumulates at the site of inflammation.



Ognjan

#4 RULE

"MRSA...Put my Kids
through college..."

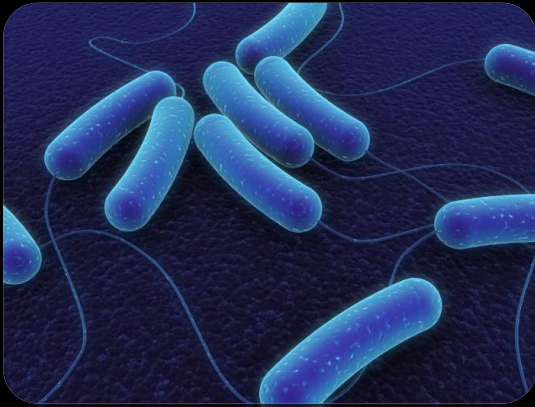
Ognjan

#5 Rule

ANTIBIOTICS ARE NOT POWERFUL



ANTIBIOTICS ARE APPROPRIATE



Bacteria



Fungus

ANTIBIOTICS **“Against Life”***



***1889 by Louis Pasteur's pupil Paul Vuillemin** 1861–1932

- ❖ Single “Scoop” of soil, contain Bacteria and Fungi number in the Millions.
- ❖ **With Thousands of varieties and survive by “fighting” each other.**
 - ❖ **Past century, several newly discovered Antibiotics have been found by isolating them from the Bacteria and Fungi that produce them to defend their own lives.**

Bacterial Antibiotic targets

Metabolic *

Bacterial DNA
Circular Double Strand

Transfer RNA

Ribosome 50s

Ribosome 30S

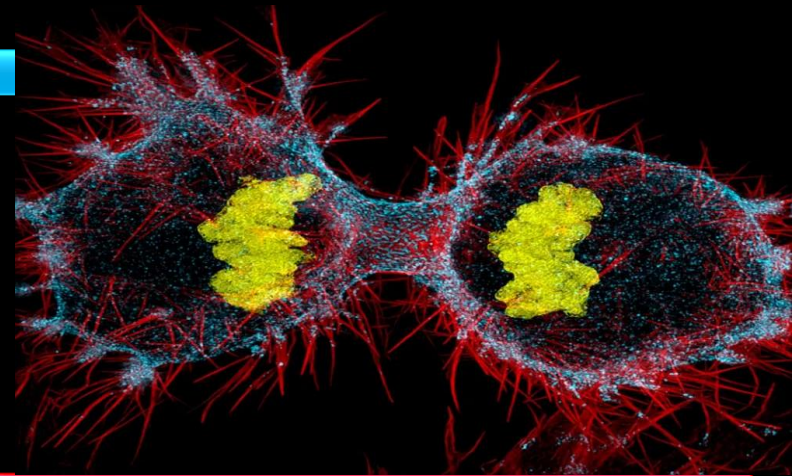
- ❖ Adenine
- ❖ Thymine
- ❖ Cytosine
- ❖ Guanine

❖ Metabolic *

DNA
Plasmid

- ❖ Cell Wall:
- ❖ Plasma / Lipid Membrane

1940 - 2017



Daughter cells

Cell division occurs every 20 minutes
Survival depends upon creation of
CELL COMPONENTS for 2 cells

- ❖ Metabolic Enzymes
- ❖ Structural proteins
- ❖ Cell wall
- ❖ DNA RNA repair / Synthesis

Antibiotic targets bacteria

DNA target:

- ❖ QUINOLONE
- ❖ METRONIDAZOLE
- ❖ RIFAMPIN

Ribosome 50s:

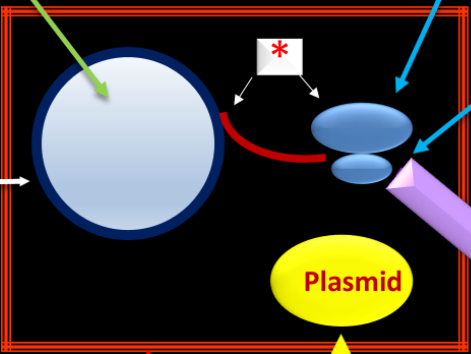
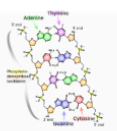
- ❖ CLINDAMYCIN
- ❖ TETRACYCLINES
- ❖ MACROLIDES
- ❖ BIAXIN / ZITHROMAX
- ❖ LINEOZOLID (23s)

Ribosome 30s

- ❖ AMINOGLYCOSIDES

❖ Adenine
❖ Thymine
❖ Cytosine
❖ Guanine

Metabolic SULFONAMIDES*



Membrane Detergents

- ❖ Daptomycin

Cell Wall:

- ❖ BETA LACTAMASES
- ❖ GLYCOPEPTIDES

1940 - 2017



Disruption:

- ❖ Structural and organelle proteins
- ❖ Cell wall synthesis and repair
- ❖ Damage / depletion Metabolic enzymes
- ❖ Disruption DNA/RNA Repair / Synthesis

Daughter Cell creation



Bacterial Death

CELLULITIS

Anatomical consideration



Anatomical consideration

Normal Skin Flora

- ❖ Staphylococcus sp [>14]
- ❖ Corynebacteria
- ❖ Proteobacteria
- ❖ Flavobacteriales
- ❖ Proteobacteria
- ❖ Cutibacterium

Introduction

The skin is the largest organ in the body, covering its entire external surface.

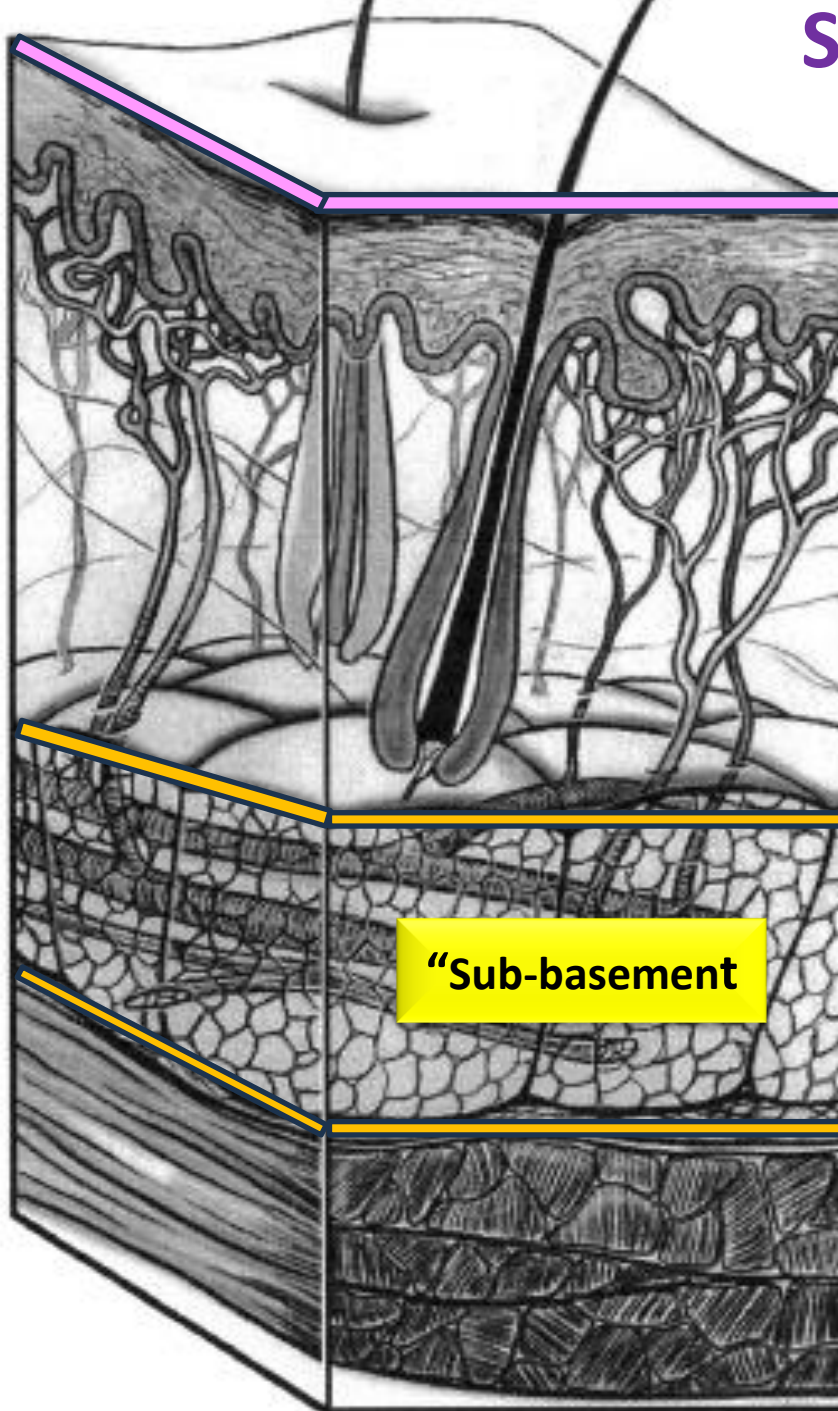
The skin has 3 layers:

Epidermis

Dermis

Hypodermis

SKIN SOFT TISSUE ANATOMY



ANATOMY

SYNDROME

Epidermis

- ❖ Hair
- ❖ Stratum Corneum
- ❖ Squamous Layer
- ❖ Basal Cell
- ❖ Merkle cells

- ❖ Erysipelas
- ❖ Impetigo
- ❖ Ecthyma

Dermis
"Skin"

Dermis

- ❖ Sweat Glands
- ❖ Apocrine glands
- ❖ Hair follicles
- ❖ Connective tissue

- ❖ Folliculitis
- ❖ Furunculosis
- ❖ Carbuncles
- ❖ Hydranitis Suppurative

Superficial Fascia

Subcutaneous Tissues

- ❖ Subcutaneous adipose
- ❖ Nerves, Arteries
- ❖ Veins, Lymphatic

- ❖ Panniculitis
- ❖ Lymphangitis
- ❖ Arteritis

Hypodermis

- ❖ "Necrotizing Fasciitis"
- ❖ Gangrene

Deep Fascia

❖ **Muscle**

- ❖ Myonecrosis
- ❖ "Gangrene"
- ❖ Osteomyelitis

CELLULITIS

What you think you know

DEFINITIONS



Cellulitis

what you think you know

cel·lu·li·tis

Noun Medicine

A localized or diffuse inflammation of connective tissue with severe inflammation of dermal and subcutaneous layers of the skin

Cellulitis classically presents with erythema, swelling, warmth, and tenderness over the affected area.

AND

Cellulitis:

An infection involving the deep dermis and subcutaneous tissue

ABSCCESS or CELLULITIS



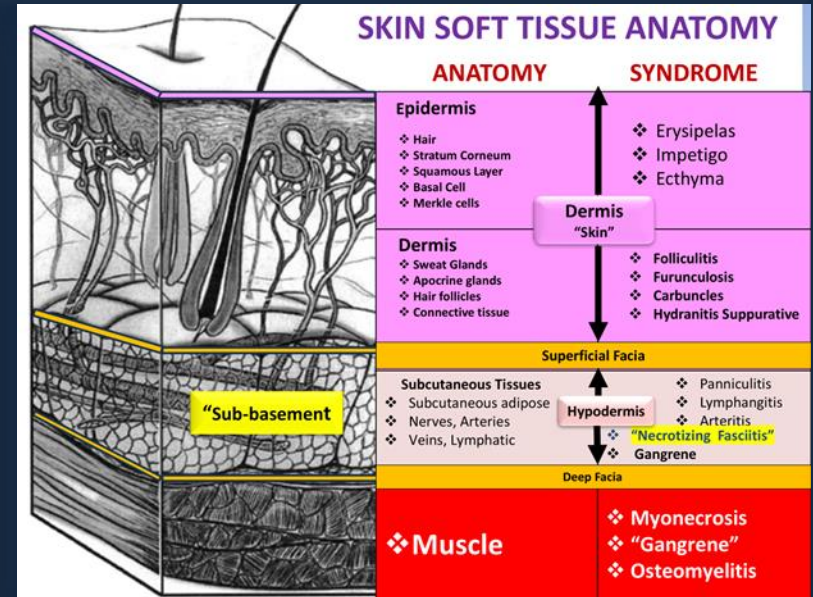
CELLULITIS : Erythema, Indurated, Spreading lymphangitis, Bullous Weeping.....
“Streptococcus”

ABSCCESS : Localized, Sinus tract, Indurated flocculent, Painful
“Staphylococcus aureus”

Cellulitis

Pyoderma Gangrenosum multiple sites

Cutaneous ulcerations with mucopurulent or hemorrhagic exudate. Painful ulcers present with undermined bluish borders with surrounding erythema.



CELLULITIS

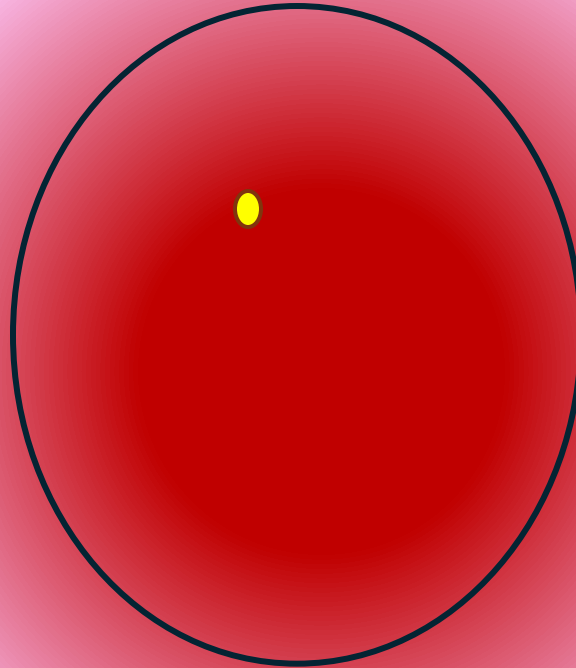
What you think you know

“Can get redder before it gets better”



“Cellulitis can get redder, before it gets better”*

ANTHONY OGNJAN DO FACP



Pathogen concerns

- ❖ Streptococcus: *S pyogenes* [GAS]; *S agalactia* [“GBS”]
- ❖ Staphylococcus aureus
- ❖ “Synergistic infection” [Mixed: GPC, Anerobic, GNB]

Cellulitis

Chronic Ulcers



NORMAL ulcer “SLIME FLORA”

- ❖ Staphylococcus epidermidis Sp
- ❖ Anerobic bacteria
- ❖ Enterobacteriaceae sp
- ❖ Enterococcus Species
- ❖ Pseudomonas sp [?]

Ulcer “Slime”

Potential Pathogen concerns

- ❖ Streptococcus: S pyogenes [GAS]; S agalactia [“GBS”]
- ❖ Staphylococcus aureus
- ❖ “Synergistic infection” [GPC, Anerobic, GNB]

CELLULITIS

A word about Cultures



- ❖ Sterile Bone Culture NO FORMILIN)
- ❖ Sterile operative Culture (Tissue, Exudate)
- ❖ “Prepped” tissue aspirate
- ❖ Post Debridement Ulcer surface
- ❖ “Cleaned” Ulcer surface
- ❖ Ulcer wound “slime” Layer
- ❖ Osteomyelitis “Sinus” tract drainage



Reliability of culture results:
Pathogen recovery
Limited Contamination



Cellulitis

Probe to Bone test : Underlying Osteomyelitis



| Signs / Predictive | Sensitivity | Specify | |
|--------------------|-------------|---------|-------------------------|
| Probe to bone | 66% | 86% | Negative Predictive 56% |



Cellulitis

Contributing Clinical factors

Clinical, Anatomic, vascular, issues

Hydrostatic pressure - Lymphedema

Vascular Ischemia

Topical Irritants

Trauma

Infectious (“typical Vs. atypical”)

Cellulitis

Comorbid Circulation Pathology

Vascular Circulation

Impacting Tissue Perfusion and Circulation

- Major vessel compromise
- Small and Medium vessel disease
- Extensive vessel scarring
- Inflammatory Arteritis
- Post Radiation fibrosis trauma
- Chronic Lymph Edema
- Tobacco abuse (>2 packs per day)
- Neuropathy (Central, Peripheral, “Mobility”)
- Venous stasis



Increased cellulitis risk

- ❖ Trauma history
- ❖ Fracture repair
- ❖ Arterial damage
- ❖ Vein damage
- ❖ Lymphatic trauma
- ❖ Soft tissue scarring

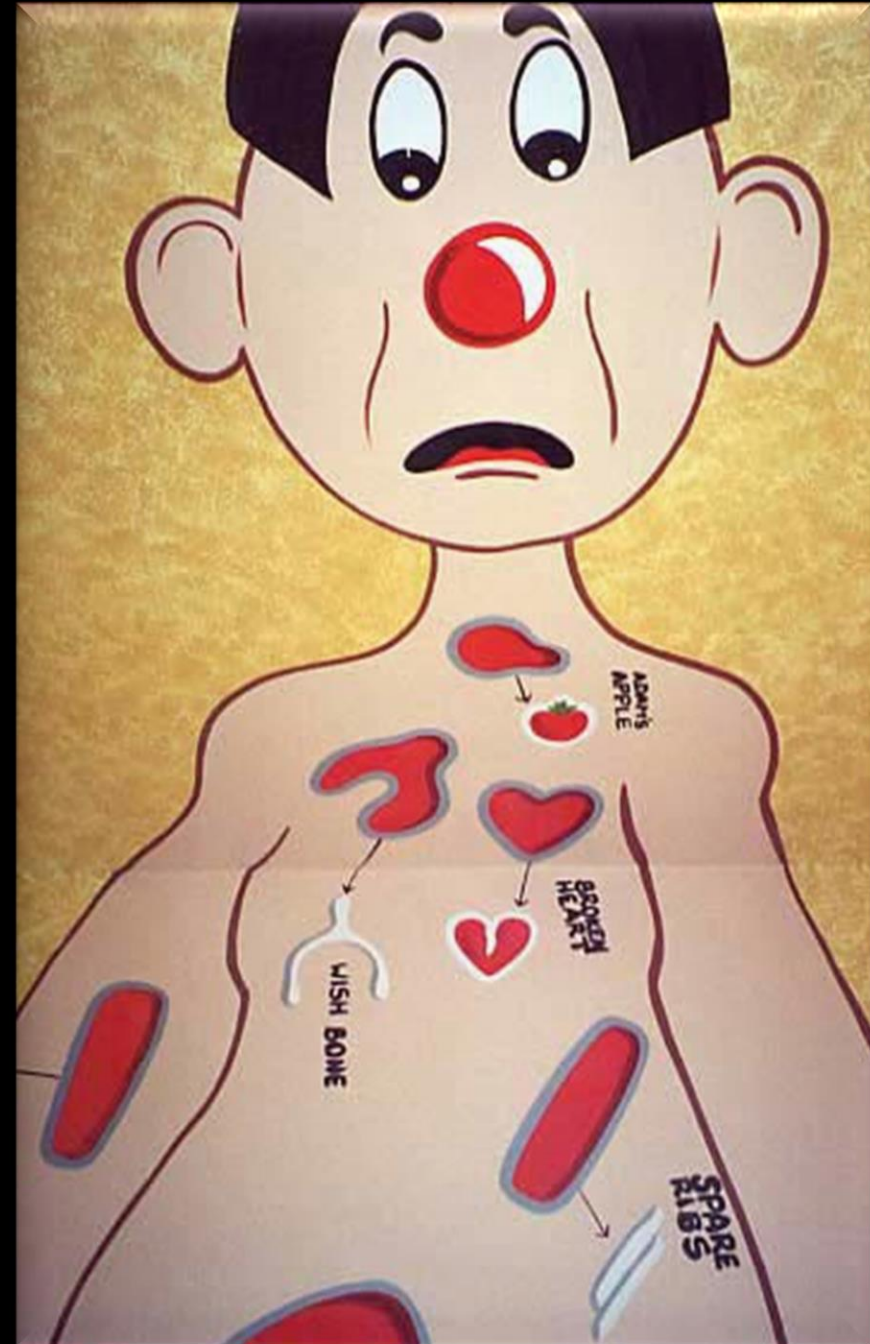
- ❖ Recurrent cellulitis

Cellulitis

Pathophysiology

“The Human Condition”:

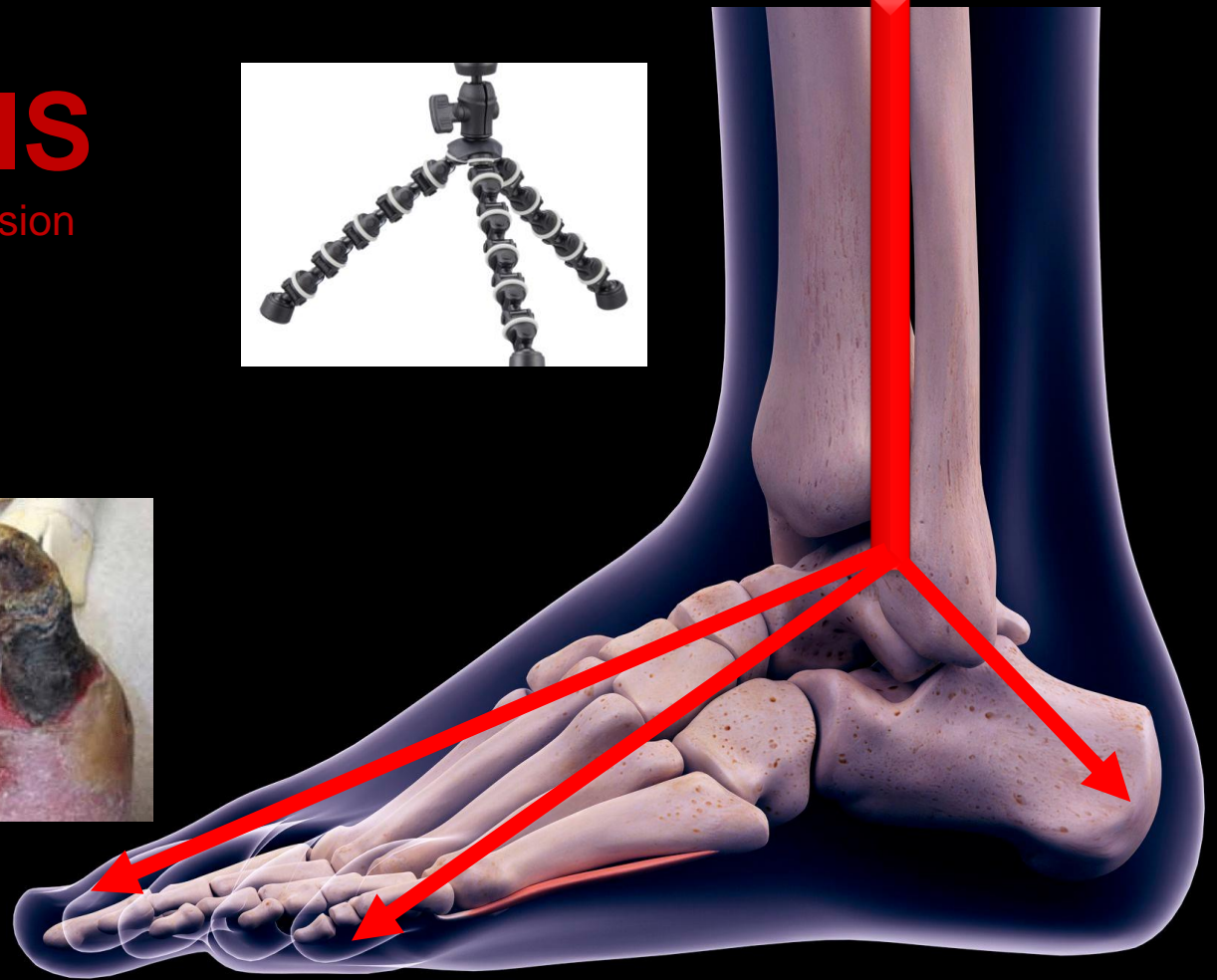
- Diabetes Mellitus
- Renal, Hepatic failure
- Malnutrition
- Chronic Hypoxia / Smoking
- Immunosuppression
- Immunodeficiency
- Malignancies
- Autoimmune disease
- Extremes of age

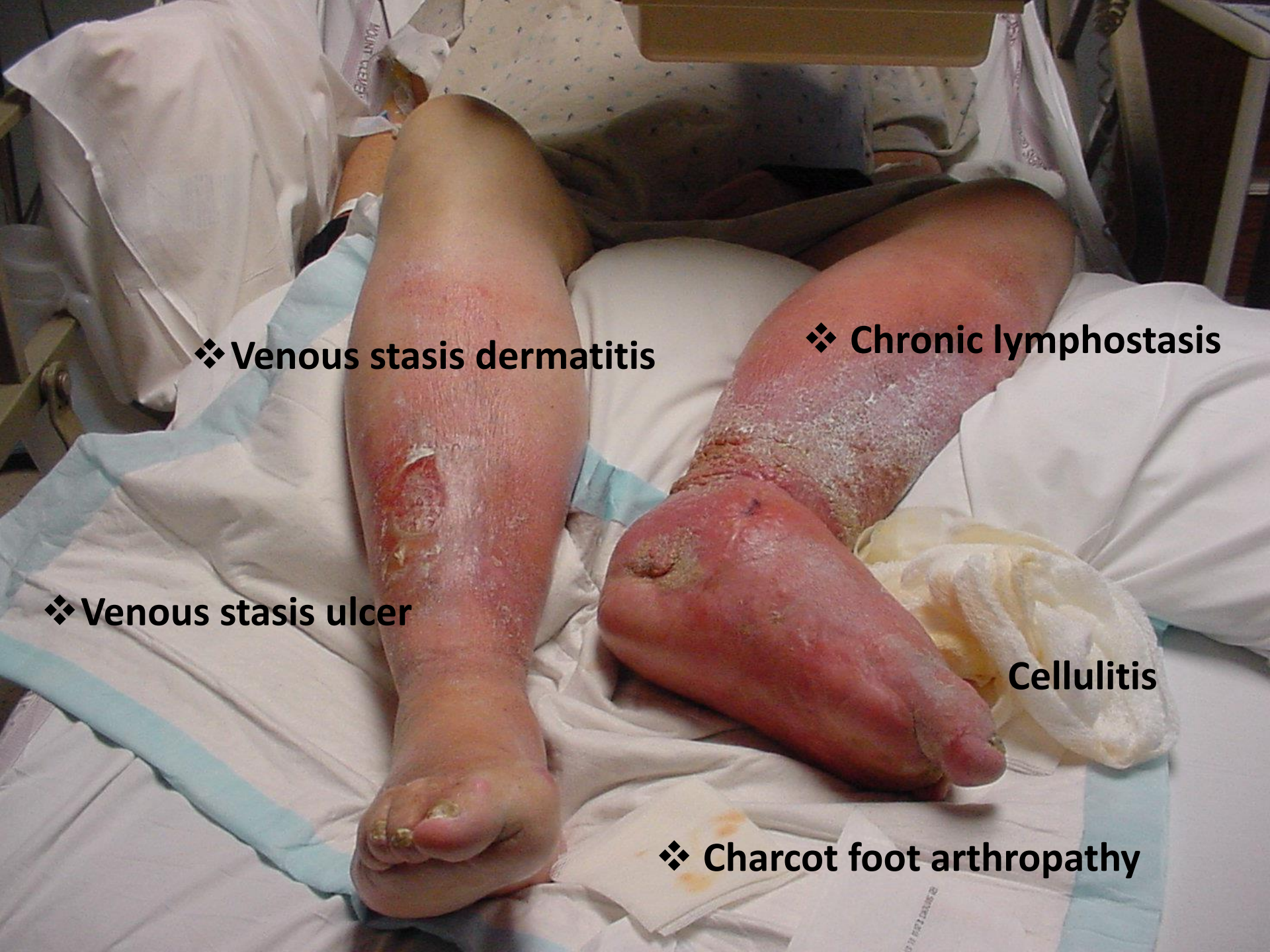


CELLULITIS

General Foot Arterial Perfusion

“TRIPOD”





❖ Venous stasis dermatitis

❖ Chronic lymphostasis

❖ Venous stasis ulcer

Cellulitis

❖ Charcot foot arthropathy

Cellulitis

Elephantiasis nostras verrucosa

Congestive Heart Failure,
Diabetic Nephropathy, and Obesity



Anatomical Vascular consideration
Lymphedema

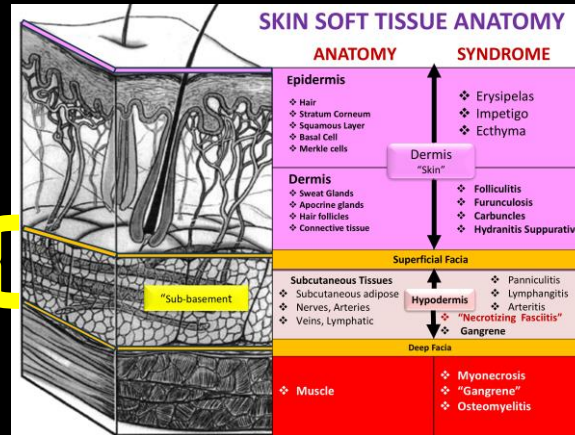


Elephantiasis



Cellulitis

Diseases Masquerade as infectious Cellulitis



Panniculitis

Septal

- Erythema nodosum
- Lipodermatosclerosis
- Morphea
- Eosinophilic fasciitis
- Eosinophilia myalgia syndrome

Lobular

- Physical panniculitis
- Cold-induced
- Traumatic
- Chemical
- Factitious
- Post irradiation

Panniculitis associated with systemic disease

- Pancreatic panniculitis
- Lupus panniculitis
- 1-Antitrypsin deficiency
- Weber-Christian disease
- Cytophagic histiocytic panniculitis
- Post-steroid panniculitis
- Nodular vasculitis

Malignant disorders

- Lymphoma
- Leukemia
- Paget disease of the breast
- Extramammary Paget disease
- Glucagonoma

Other

- Calciphylaxis
- Compartment syndrome

Panniculitis :

Group of diseases

- ❖ Hypodermis Pathology
- ❖ Neurovascular adipose



Cellulitis

Sulfa Allergy

Toxic epidermal necrolysis

“Stevens Johnson syndrome:

keratinocyte necrosis with separation of the epidermis from the underlying dermis.



CELLULITIS

What you think you know

“It’s a mystery to me”





Gee, I wonder
why this
“Uncultured”
Infection
won't heal...



I've tried every
antibiotic I know...
for the past six
weeks....
Not any better...

A futuristic laboratory setting with a large, glowing, spherical object suspended in the center. The room is filled with complex machinery, including large vertical columns and circular structures. A person in a white lab coat is standing at a control console in the background. The overall atmosphere is high-tech and industrial.

**...I will just have
to try my
“New”
Antibiotic...**



NAPALMICIIN



OR...

Biopsy...

Culture...

- ❖ Typical Bacteria
- ❖ Atypical Bacteria
- ❖ Fungi



Sporothrix

- ❖ Itraconazole
- ❖ terbinafine
- ❖ IV amphotericin B
- ❖ Supersaturated potassium iodide



“Cat Scratch” Bartonella
Suppurative Epitrochlear
Lymphangitis



Sézary disease
Cutaneous T-Cell
Lymphoma



Pyoderma
Gangrenosum



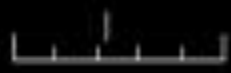
Herpes Zoster
“Shingles”



INST: McLaren Macomb
MG_D: 2012.06.20
MG_T: 18:18:33
MG#: 55
SL: 108.0 mm
ST: 0.9 mm

L

KVP: 120
XRAYTUBE: 224
GANTRYTILT: 0.0



20 year history
Neglected Basal cell cancer
(Secondary MSSA)



An “anxious” third year medical student assigned to an History and physical, notices a “peculiar” physical finding

Curiously, not mentioned in the DEM admit note







What the heck?

- What do you see?
- Differential?
- Treatment options?



Condyloma acuminata

- Twenty year growth of “venereal warts”
- Both are contagious
- Condyloma lata “Lotta syphilis organisms”



Condyloma lata

Each weighed 1.5 lbs

**Giant condylomata of Buschke-Loenstein
(HPV-6)**

CELLULITIS

What you think you know

Foreign Bodies // Trauma



CELLULITIS

What you think you know

Foreign Bodies /Trauma

Wooden splinter



Remove Foreign body
if possible



Infected arterial graft



Infected knee
arthroplasty



Infected Venous IV catheter

Trauma

Penetrating/Lacerating

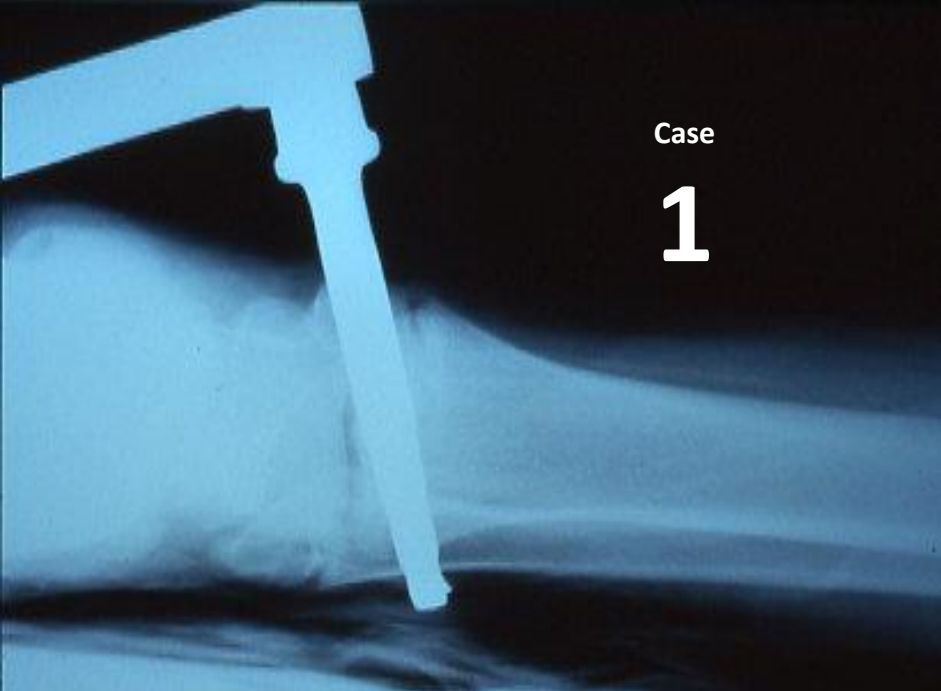


AR 15

Both AR-15; AR-16
Use
Remington .223 or
Military 5.56 mm rounds

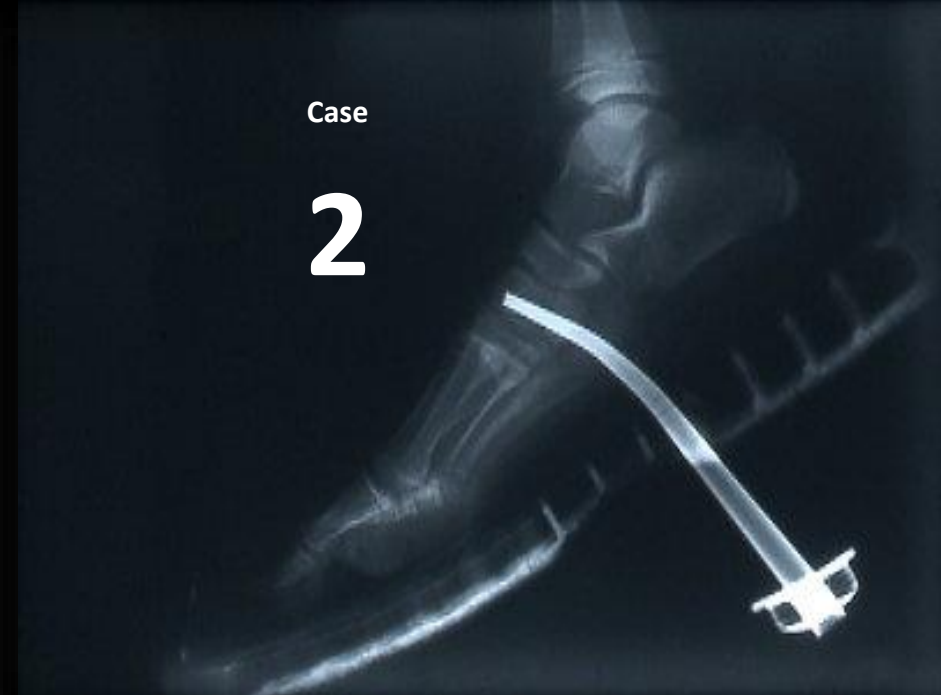


Lawn mower



Case

1



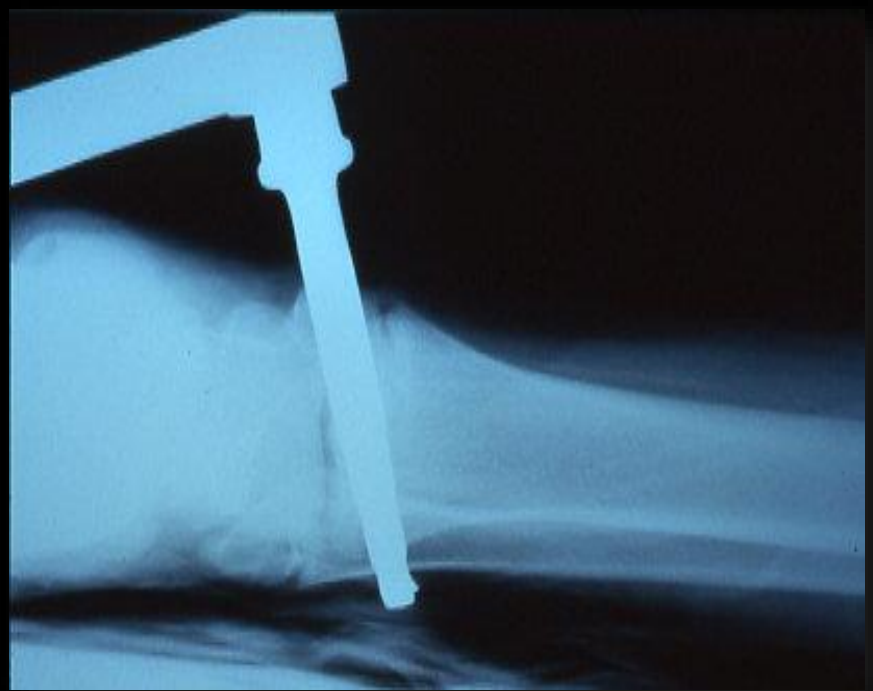
Case

2

What's this X-ray??....

New orthopedic fixation
"devices"?.....





No, Motorcycle pedal.....

24 year old lost control of his motorcycle hitting the back of a parked car.



AND

Oil can funnel through tennis shoe.

13 year old, "hyperactive" male, "playing" at a construction site, jumped down from the construction on to a oil can.....



- ❖ Piperacillin / Tazobactam
- ❖ Cefepime / Metronidazole
- ❖ ?Quinolone / Clindamycin

Question:

True

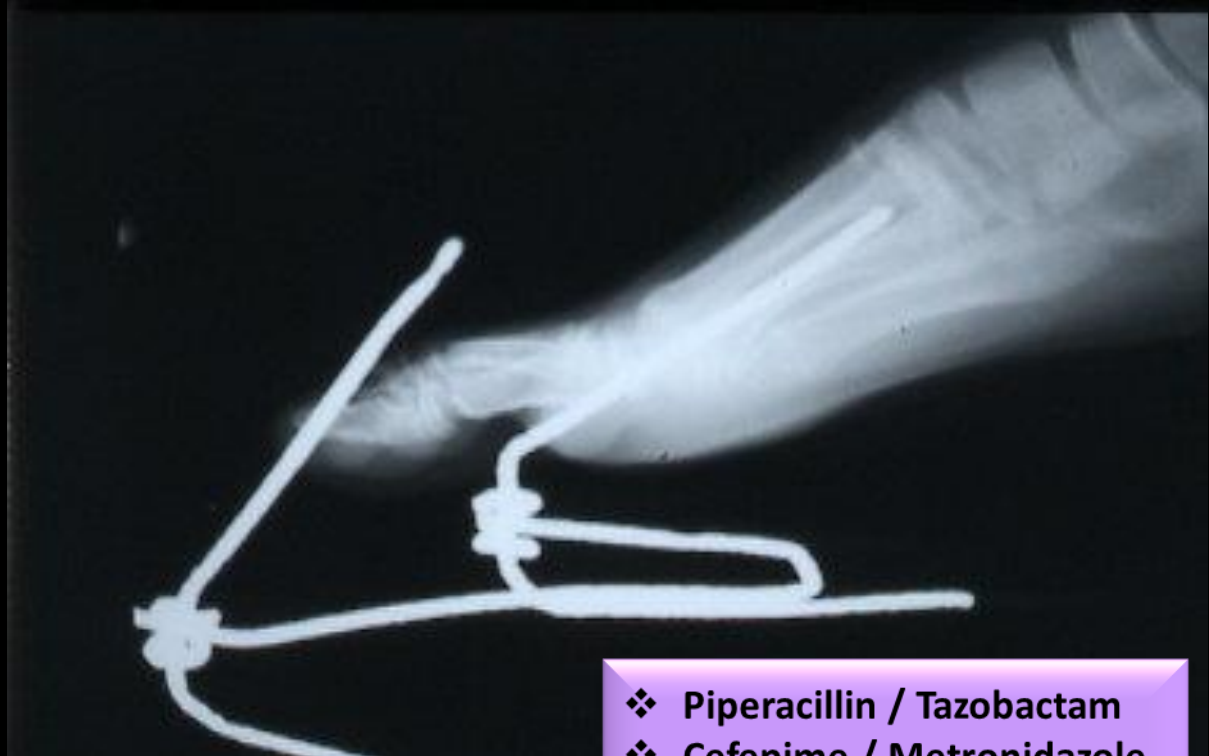
False

White milk comes from White cows.....
Chocolate milk comes from Brown cows.....





- 13 year old running barefoot through a farm pasture with several friends, sustaining a foreign body (FB) to his bare foot.
- Brought into the DEM by his family, not particularly painful, bleeding controlled.



- ❖ Piperacillin / Tazobactam
- ❖ Cefepime / Metronidazole
- ❖ ?Quinolone / Clindamycin

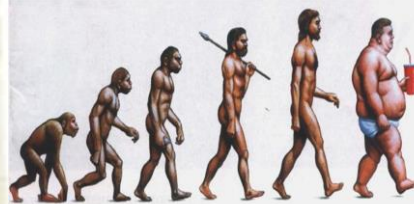
- **Note the proximity of the barbed wire to the bone (traumatic osteomyelitis)**
- **Antibiotics are appropriate, as well as tetanus.**
- **Be sure removal of a foreign body does not cause further damage.**
- **Appropriately, vaccinated people need tetanus vaccinations:
during “mid decade” 15 yr, 25 yr, 35 yr, 45 yr, etc.**

CELLULITIS

What you think you know

Community acquired MRSA

As humans evolve...



... So do Bacteria

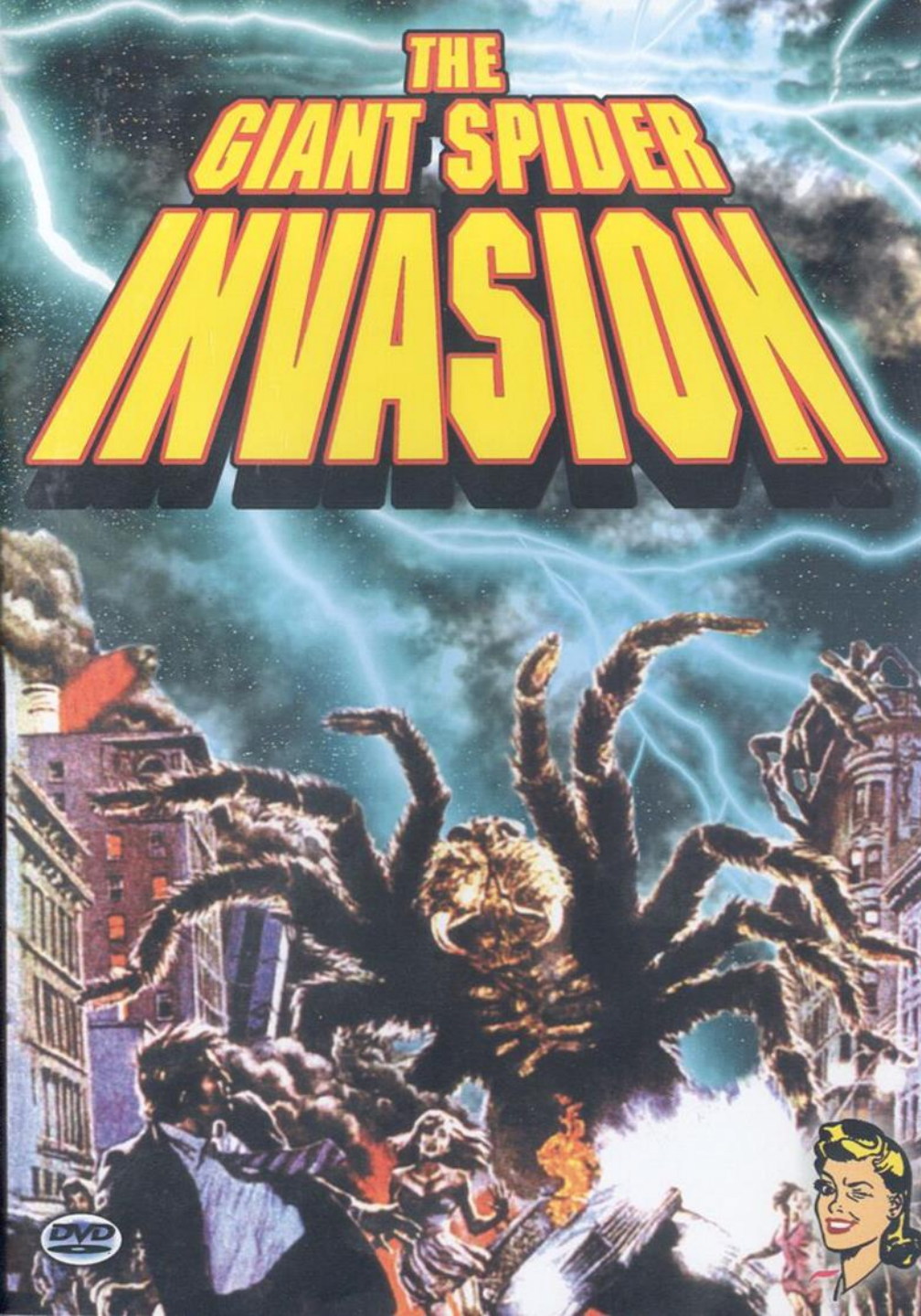
CELLULITIS

What you think you know

“Has to be a spider bite Doc...
What else could it Be?”



2002



2002

*It must be a "Spider bite"....
.....What else could it be?*

Loxosceles Reclusa



CELLULITIS

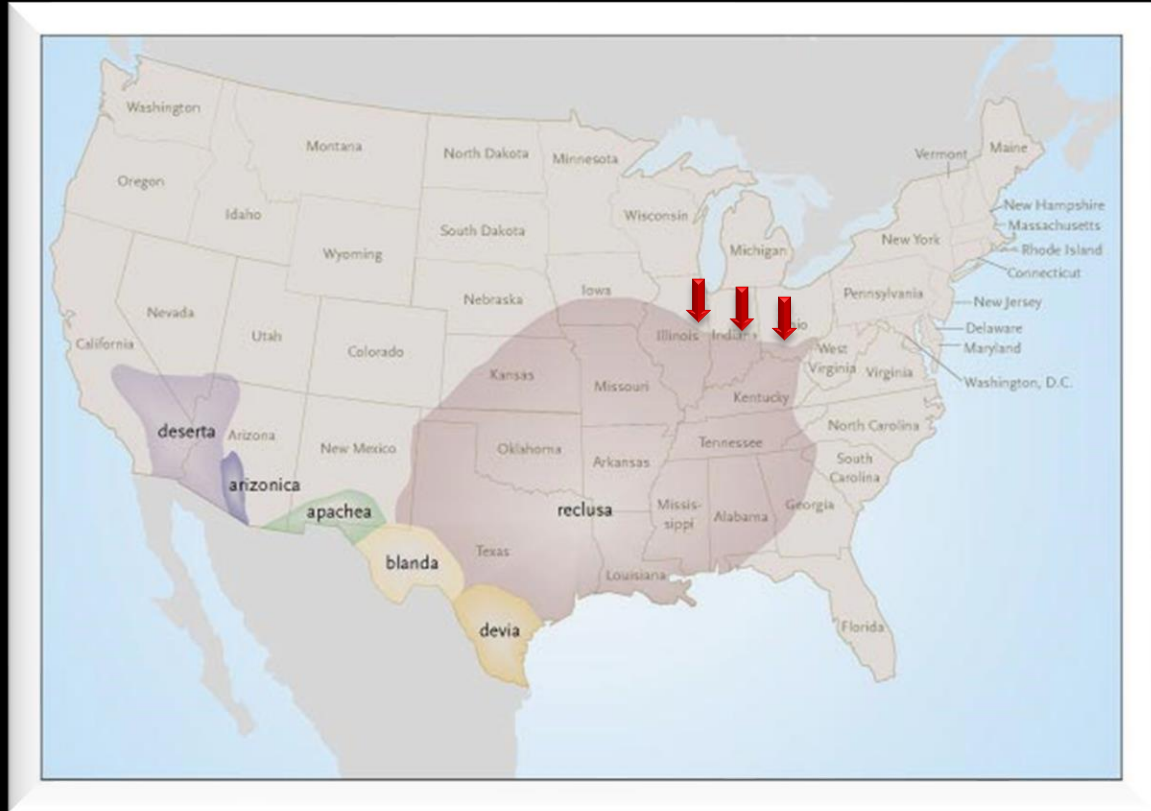
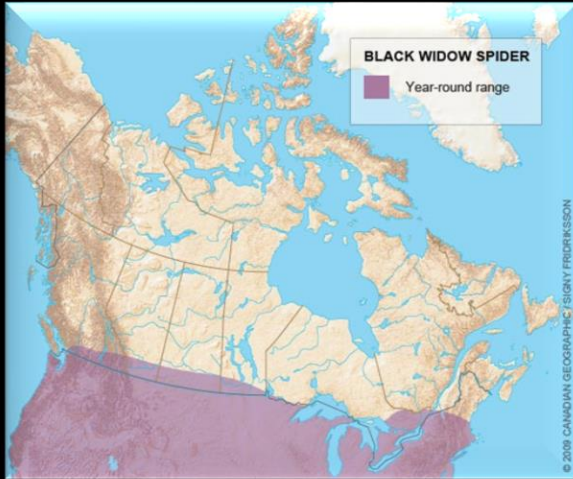
What you think you know

Only one thing in Michigan...



2002

U.S. Geographic Distribution of Verified Widespread Populations of Six Native Loxosceles Species. (“Brown Recluse Spider”)



Michigan's *biting* spider
Northern Black Widow Spider
(*Latrodectus variolus*)

Underside view, displaying red hourglass marking

Brown recluse spiders are VERY rare.

Preventative Treatments for Michigan's Spiders
Creature Control Insect Pests. Spiders
<https://www.creaturecontrol.net/insect-pest-control/spiders/>

Loxosceles spiders “purportedly” are transported beyond the areas where they are endemic in household goods and warehouse cargo...
...But uncorroborated...actual epidemiology essentially **does not** occur beyond the spiders' usual habitat.

- ❖ **The brown recluse is not indigenous to Michigan and cannot live in temperatures colder than 40°F, so they are extremely rare in the state*.**

CELLULITIS

What you think you know

Staphylococcus aureus

Gee Doc....

I have?....

“Staff-ala-roar-e-us”

“Staff-full-of-oreos”

“Staff-full-of-coccus”



MAYBE



MRSA Genome



Survival



**One drop MSSA Pus...
Contains 250,000 organisms...
1 of these bacteria have a
chromosomal mutation**

mecA 2a

**This Mutation confers Antibiotic Resistance
to all BETA LACTAM ANTIBIOTICS...**

**The mutation can be transmitted
“Daughter” Bacteria Cells**



MRSA

1964 Likely original MRSA infections developed in Immunocompromised; hospitalized patients with initial **Methicillin sensitive** (MSSA) infections. Antibiotics eliminated the Penicillin / MSSA “sensitive” Bacteria....



HOWEVER

The patients in their “weaken” immune state could not clear the residual **Methicillin resistant** (“MRSA”) bacteria leading to “super” infections with MRSA

1970’s the MRSA organisms “escaped” the hospital ... and began infecting and colonization the General Population.....



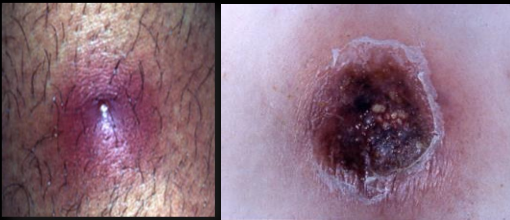
2002

Where Did all this Community Acquired
CA-MRSA Come from?



MRSA Hospital
1960-1970s

“Community MSSA”



“Spider Bites”
Furuncle / Carbuncle
“Boils”



Community MRSA
(USA 300)
2000

Increased Virulence

- ❖ Panton-Valentine leucocidin genes
- ❖ Expression of core genome-encoded toxins



Bullous impetigo



Ecthyma



Ecthyma



Folliculitis

S. aureus
Skin lesions



Ecthyma"



Folliculitis



"Boil" Furuncle



Carbuncle

Representative Antimicrobial Susceptibilities (%) Of CA-MRSA and Health Care-Associated MRSA

(Kowalski et.al Mayo Clinic Proceedings)

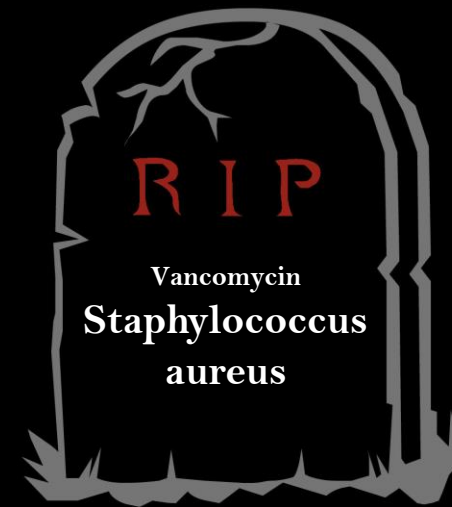
| Antimicrobial | CA-MRSA | Hospital MRSA |
|----------------------------|-------------|---------------|
| Agent | % Sensitive | |
| Oxacillin (Cephalosporins) | 0 | 0 |
| ❖ Ciprofloxacin | 79 | 16 |
| ❖ Clindamycin * | 83 | 21 |
| Erythromycin | 44 | 9 |
| Gentamycin | 94 | 80 |
| Rifampin | 96 | 94 |
| ❖ Tetracycline | 92 | 92 |
| ❖ Sulfa-Trimethoprim | 95 | 90 |
| ❖ Vancomycin | 100 | 100 |

 Greater than 50% sensitive

 Less than 50% sensitive



**Vancomycin
Ace in the hole**



HOWEVER:

S. aureus (MSSA or MRSA)

WILL ALWAYS

Remains Sensitive to Vancomycin:

**ANY De-novo MUTATIONAL attempt
for S. aureus to become resistant to Vancomycin**

is a

**Lethal Chromosomal mutational event
for the Bacteria....**

For the present:

**IV VANCOMYCIN : Remains “Ace in the Hole” for MRSA and MSSA
infections**

Cellulitis

Vancomycin resistant staphylococcus aureus “VRSA” 2002

Vancomycin-resistant Staphylococcus aureus (VRSA)

A rare but difficult-to-treat bacterial infection caused by a strain of Staphylococcus aureus that has become resistant to the antibiotic vancomycin

Transmission of the VanA gene,
one off the vancomycin resistance genes,
found among
“VRE” Vancomycin Resistant Enterococcus

Summer 2002 Metro Detroit Michigan

40 year old Michigan woman became the first person worldwide known to have been infected with a strain of Staphylococcus aureus that was resistant to the antibacterial vancomycin

Since the initial report
total 52 worldwide cases have been reported



CELLULITIS

S aureus

Epidemiology: Human Carriers

Transmission:

Close personal contact

Infected secretions

❖ (“Pus”) / Objects

Skin Contact:

❖ Athletic competition

❖ Draining lesions

❖ Shared Personal items

❖ Towels, Razors, Medical Equipment etc.



30 - 40% Healthy human carriers At any given time

1% Humans are MRSA carriers

***335 Million Americans:
100 Million MSSA carriers
1 million MRSA Carrier***



Coagulase Negative
Staphylococcus

Provides

“Protective Suit of Armor”

- ❖ **ALL Skin, Mucus Membranes**
 - Not the GI tract
- ❖ **Humans can't survive without colonization**
- ❖ **Very LOW Pathogenic Potential:**
 - Increased with “Prosthetic” materials
- ❖ **Often Methicillin Resistant**
 - Humans take too many antibiotics

So... if it NOT....S. Aureus..... What is it?.....

“Coagulase Negative Staphylococcus”

S lugdunensis
S schlieferi

S intermedius
S pseudintermedius

S. epidermidis species group

- S epidermidis
- S haemolyticus
- S hominis
- S saccharolyticus
- S warneri
- S capitis
- S auricularis

S. saprophyticus group

- S saprophyticus
- S cohnii
- S xylosum
- S arelettae
- S equorum
- S gallinarum
- S kloosii

S. sciuri species group

- S Sciuri
- S lentus

S. simulans Species group

- S simulans
- S carnosus

Unspecified Group

- S caseolyticus
- S hyicus

“Cluster” Gram Positive Cocci
Are Either S. aureus
or are not !!!!!

Any questions?

Staph?
**“I don’t give a
sh—t-a-cus”**



CELLULITIS

What you think you know

Streptococcus Bacteria



S. pyogenes
Clinical disease

Bacterial Toxic Shock Syndrome "GAS"

| Factoid | Streptococcus | Staphylococcus |
|---------------------------------------|--------------------|----------------|
| Identifiable Infectious source | +++++ | + |
| Prodrome 2-3 days | Yes | Yes |
| Fevers, chills | Yes | Yes |
| Nausea, Vomiting | Yes | Yes |
| Watery Diarrhea /abdominal Pain | Yes | Yes |
| Myalgias Arthalgias | Yes | Yes |
| Pharyngitis / Headaches | Yes | Yes |
| Confusion | ? | More Common |
| Bacteremia | More common | Rare |
| Pain at site of infection | More common | ? |

Non-invasive Disease

- ❖ Strep throat ("Scarlet fever")
- ❖ Cellulitis
- ❖ Impetigo
- ❖ Erysipelas

Invasive Disease

- ❖ Necrotizing fasciitis
- ❖ Myositis, Endocarditis
- ❖ Streptococcal toxic shock
- ❖ Pneumonia, Osteomyelitis

IMMUNE MEDIATED DISEASE

- ❖ Rheumatic fever
- ❖ Glomerulonephritis



Jean Harlow
March 3, 1911 - June 7, 1937
(Age 26 years)



James Maury Henson
September 24, 1936 – May 16, 1990
(Age 53)



Ignaz Phillip Semmelweis
1818- 1865
"Wash your Hands" -1846
Maternity Vienna General Hospital



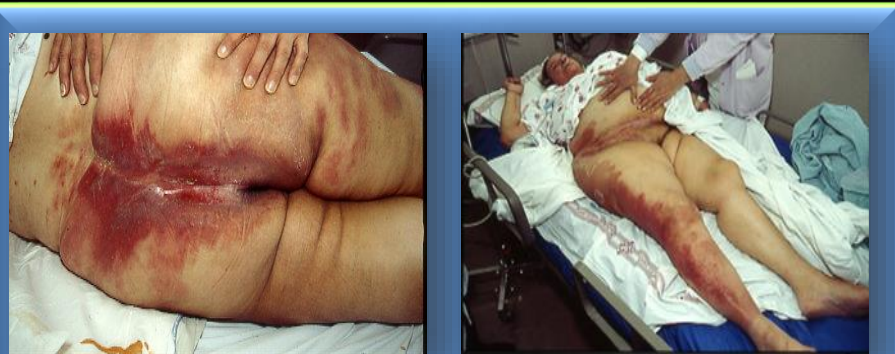
Streptococcal Necrotizing cellulitis
"Flesh Eating Bacteria"

NORMAL 48 HOURS AGO...
 "FLESH EATING" bacteria
 Evil "M" TYPE: rare (type 1,3)
 ❖ **Ampicillin / Sulbactam** to kill
 ❖ Clindamycin : "Stop" ribosome protein/toxin production
 ❖ **AMPUTATION**



❖ Necrotizing fasciitis caused by *Streptococcus pyogenes* infection can be rapidly fatal
 ❖ This is probably the result of a toxic shock syndrome ⁽¹⁾

1 P M Donaldson, B Naylor, J W Lowe. *Rapidly fatal necrotising fasciitis caused by Streptococcus pyogenes*. J Clin Pathol. Jul 1993; 46(7): 617-620.



"Millions" Non-invasive (Pharyngitis) / 10-15,000 invasive/year

CELLULITIS

What you think you know

Necrotizing fasciitis





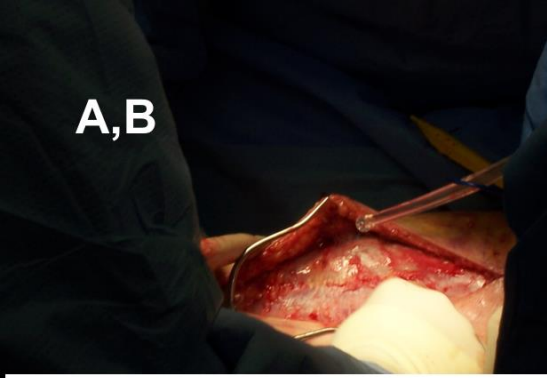
Cellulitis

Streptococcus pyogenes

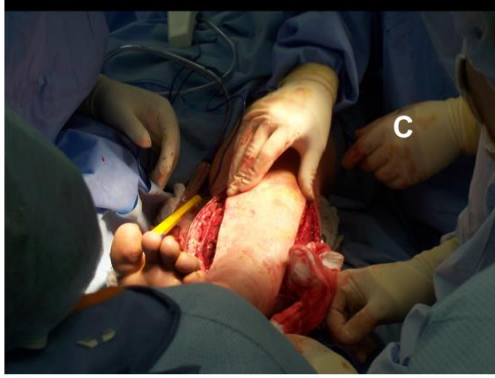
Streptococcal pyogenes
Necrotizing fasciitis and
Toxic shock



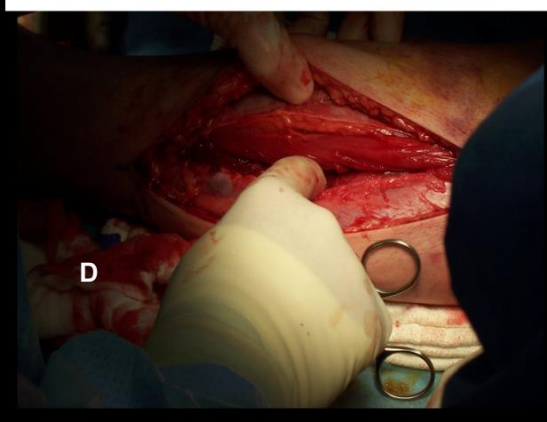
A,B



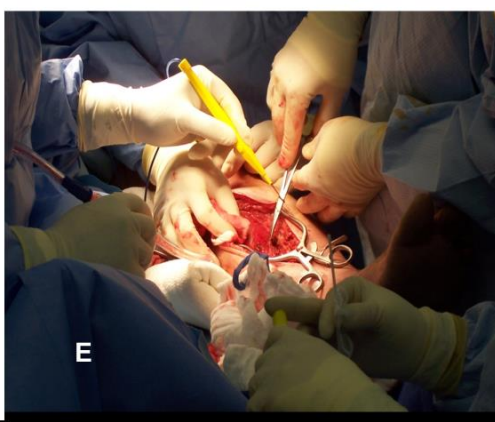
C



D



E



Right Axilla "Satellite" lesion

Necrotizing Fasciitis surgical findings

Surgical exploration and debridement:

- A- Gray Necrotic Tissue
- B- "Dishwater Pus"
- C- Lack of Bleeding
- D- Lack of resistance to finger dissection
- E- Non-contractile muscle



Back to the O.R. 24 Hours later for an Above the knee Amputation

DAY 5

Last revision



- Wound was monitored closely. Ceftriaxone / clindamycin maintained for 3 weeks past the last Debridement.
- ICU 5 days Post Op, extubated. (Remarkably no Nosocomial complications) .
- Patient was discharged to rehabilitation after three weeks of hospital confinement.....



CELLULITIS

What you think you know

CASE STUDIES



Osteomyelitis

History Neuropathic Foot

In 1703, William Musgrave
first described a
“Neuropathic joint”
as an “anthelia” * caused by
venereal disease**.

Syphilis**

**Anthelia : faint halo sometimes seen in polar or high altitude regions around the shadow of an object cast onto a thick cloud bank or fog.*



William Musgrave
(1665-1721)

British physician and antiquary.

Jean-Martin Charcot

Neuropathic Joints

“Charcot Joint”



Born in Paris France, a professor at the University of Paris for 33 years. In 1862 he began a life-long association with Salpetriere hospital.



Jean-Martin Charcot
1825-1893

- An excellent teacher, he attracted students from all over Europe.
- Among his students were **Alfred Binet** and **Sigmund Freud**.
- In 1882 his focus turned to neurology establishing a clinic at his hospital
- He has been called the “father” of modern neurology

n

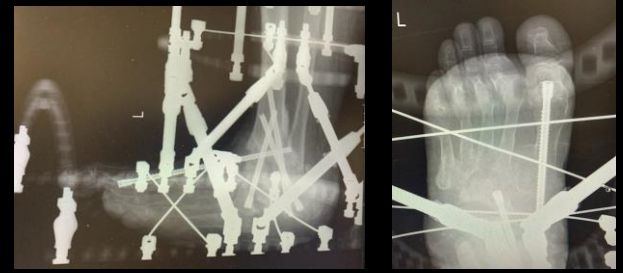
1883, Charcot described the ‘**piéd tabétique**’ or ‘**tabetic foot,**’ as tabes dorsalis; As Neurosyphilis was the most common cause of neuroarthropathy at the time

Tabes dorsalis is a slow degeneration of the nerve cells and nerve fibers that carry sensory information to the brain. The degenerating nerves are in the dorsal columns of the spinal cord (the portion closest to the back of the body) and carry information that help maintain a person's sense of position

Jean-Martin Charcot

Neuropathic Joint

Charcot Joint 'Rocker Bottom foot'



Diabetes Mellitus:

In US has long surpassed syphilis as the leading cause of Charcot Neuropathy

- ❖ Prevalence among DM patients : 0.08%–7.5%
- ❖ Although rare, Charcot Neuropathy one of the most destructive complications of diabetes
- ❖ Subluxation, Dislocation, Deformity, Ulceration of the foot and ankle joints



Rocker bottom foot deformity



Broken foot?

What broken foot...



Charcot foot

Neuropathic joint disease

Wound Care Center



36-year-old obese (350 lb) 15-year DM-II male
Now with several years IDDM

Undergoing treatment for a ***“Neuropathic Left”*** foot,
associated osteomyelitis and digital gangrene

Patient Has been ambulating with Orthotic shoes and
crutches...

Occasional oral antibiotics ... Local wound therapy.....
At a Wound Care Center



Missing two weeks of clinic appointments:

Now reports

He had tripped and fallen while climbing stairs, destroying his crutches and damaging his left foot orthotic device.....

He rescheduled his clinic visit when he noticed his right foot was swollen, he could no longer wear his right shoe...



His socks were soaked with a pungent watery fluid, and it was becoming difficult to “hobble around” without his crutches.....



- Full-thickness plantar skin and soft tissue injury.
- Necrotic tissues and bone fragments at the wound base
- The ankle and foot clearly swollen and discolored...



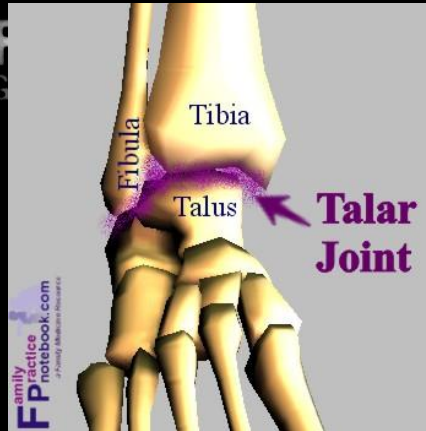
With Diabetic Foot neuroarthropathy...

“Charcot foot”

Not unexpected the patient feels “No pain”



Normal



ANKLE MORTISE *



Patient

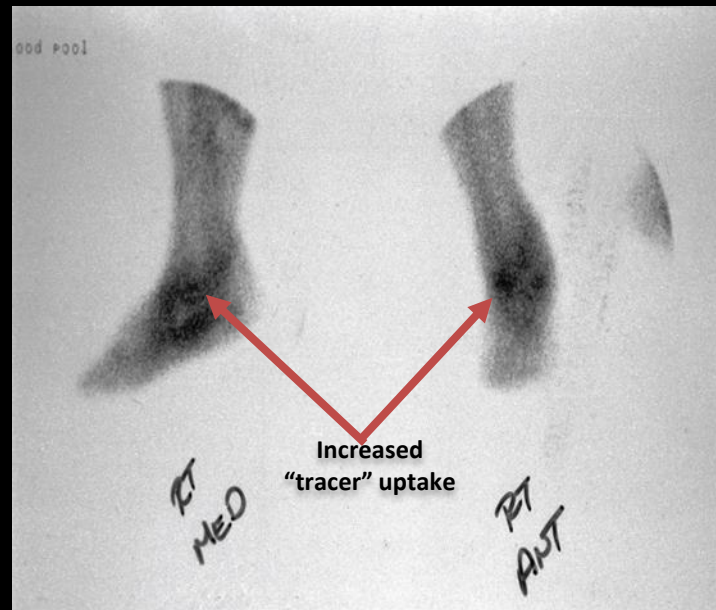
X-RAY: Ankle mortise* is disrupted; bone fragments can be seen a "piece of the talus has broken off" and migrated toward the plantar foot surface

*** The ankle mortise is the "hinge" that connects the ends of the tibia and fibula to the talus**

**Radiographic the bone fragment can clearly be seen
As a “moot” point the bone scan is “Positive”.**



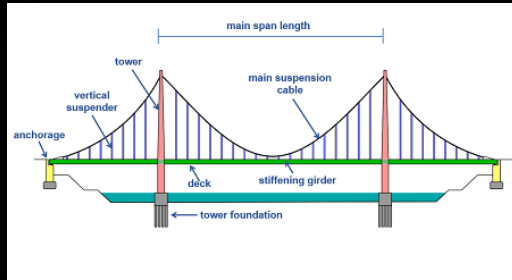
Talus bone fragment



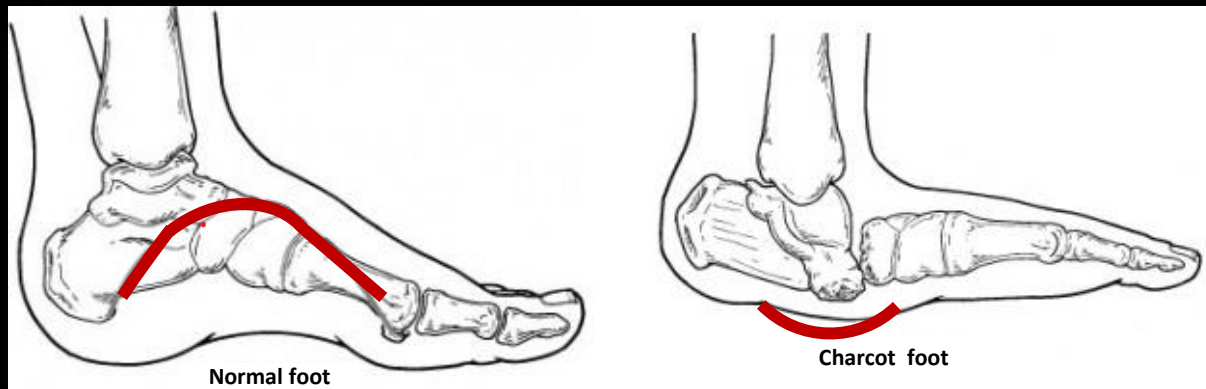
in 2024
National cost \$1,255

The patient refused amputation... but later consented.

Power of “Peripheral neuropathy”



Our 350 lb patient... feeling No Pain
For 3 weeks: ~900 pounds pressure* each step...
On a Trimalleolar ankle fracture...
No LESS!



- ❖ Supporting foot tendons and ligaments support the foot arch
- ❖ Healthy foot arching provides reflexive spring and balance.
- ❖ Weight, trauma-injury weakens the tendons - ligaments making up foot / ankle architecture
- ❖ **Over time, the arches will collapse, causing flat feet and/or abnormal plantar bone pressure points**
NORMALLY significant pain when standing and walking patients persons will off load
Feeling No pain....No offloading....Continuing his foot injury

*Estimated that every pound of body weight causes three pounds of force that your feet have to absorb when you're walking, and seven pounds when running. That means a 200 pound person's feet would be subject to 600 pounds of force with every step, and 1,400 pounds of force when running.

Tri-malleolar Ankle Fracture

Medial malleolus [Tibia]
Lateral malleolus [Fibula]
Posterior malleolus [Tibia]



Trimalleolar ankle fracture



Patient

**Under anesthesia
the foot could be “rotated”
almost 360° around the Tibiotalar joint
of the ankle mortise**

Question:

Which are true regarding Animal Bites? (Except)

- A) *Pasturella multocida* is common with cat bites.
- B) *S. aureus* and *Streptococcus* are common.
- C) Keflex[®] (Cephalexin) is appropriate first line antibiotic.
- D) Augmentin[®] (Amoxil/Clavulanate) is an acceptable first line antibiotic

Question:

ALL are true regarding Animal Bites? [EXCEPT]

A) *Pasturella multocida* is common with cat bites.

B) *S. aureus* and *Streptococcus* are common.

C) Keflex[®] (Cephalexin) is appropriate first line antibiotic.

D) Augmentin[®] (Amoxil/Clavulanate) is an acceptable first line antibiotic

Alternate

- Tetracycline or Doxycycline plus Metronidazole
- IV Ceftriaxone / Clindamycin
- Fluoroquinolone / Clindamycin

Cellulitis

Miscellaneous



- ❖ Staphylococcus species
- ❖ Streptococcus species
- ❖ Eikenella species
- ❖ Pasteurella species
- ❖ Proteus species
- ❖ Klebsiella species
- ❖ Haemophilus species
- ❖ Enterobacter species

DOG BITES

Amoxil Clavulanate
Quinolone / Clindamycin



❖ Pasteurella multocida

- ❖ Streptococcus
- ❖ Staphylococcus
- ❖ Fusobacterium
- ❖ Porphyromonas
- ❖ Bacteroides



Cat scratch disease
Bartonella

CAT BITES

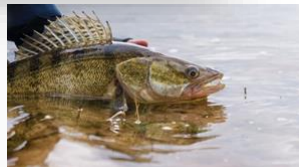
Amoxil Clavulanate
Quinolone / Clindamycin



- ❖ Eikenella corrodens
- ❖ Staphylococcus
- ❖ Streptococcus

HUMAN BITES

Amoxil Clavulanate
Quinolone / Clindamycin



Aeromonas hydrophila

Lacerations or puncture wounds sustained in freshwater environments are susceptible to contamination by Aeromonas hydrophila.

tetracyclines, sulfonamides, trimethoprim + SMX/MP or ciprofloxacin



Here kitty, kitty.....



36 year old male

- Puncture wounds to right hand
- Injury occurred 12 hours ago....

Sister is at the bed side

TALL TALES AND OTHER BIG LIES



STORIES OF RAY WYLIE HUBBARD

According to his sister
Patient Has a history of alcohol abuse
and telling “tall tales”.



Physical examination

- Significant erythema, edema and induration
- Seemingly : “Puncture” wounds over the first MCP and thenar region
- Significant mucopurulent discharge freely expressed
- Pain seems to be limiting range of motion of the digit



“Panther bite”

Patient story:



The patient lives in the Metro Detroit area...

(11 mile and Schoenherr –Warren Michigan)

➤ Has a “pet panther” that lives in his “basement” ...

(His sister denies he has a “pet panther”)

“The cat needed to pee....So I let him out in the back yard

He wouldn't come back inside, so, when I picked him up to bring him into the house, he bit me on the hand”



Provoked Vs. non-provoked bite?



- Note the space between the puncture wounds (4.0 cm)
- Identical “spaced” marks on palmar surface
- Purulent material was a “Gram negative bacteria”
- Organism identified **Pasturella Multocida.....**

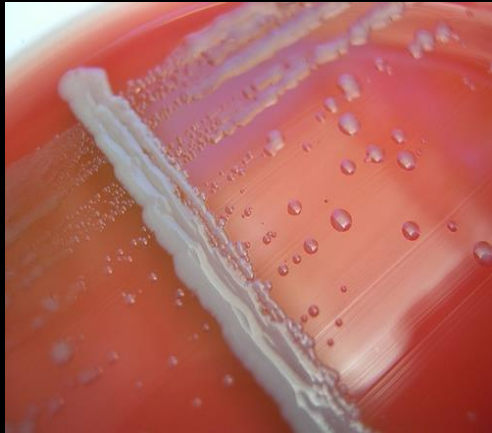
Can you Name 6 pathogens that cause infections after a cat bite?

- *Pasteurella species*
- Anaerobic bacteria: e.g., *Fusobacteria*
- *Bartonella henselae*
- Rabies virus
- *S. aureus*
- *Streptococcal species*

- ❖ Ampicillin / Sulbactam
- ❖ Amoxil / Clavulanate
- ❖ Quinolone / Clindamycin



P. multocida

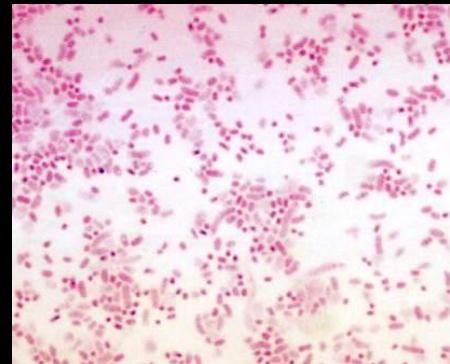


- *P. multocida* is the most common cause of infection from animal injuries.
- Clinically a relatively high WBC
- **Generally, a diffuse localized Cellulitis and purulence develops rapidly**
- Bacteremia-Sepsis can result
- Cellulitis Osteomyelitis, Endocarditis

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gamma Proteobacteria
Order : Pasteurellales
Family: Pasteurellaceae
Genus: *Pasteurella*

Species

- *Pasteurella multocida*



Pasteurella Multocida



Cat and Dog saliva :

- CATS: > 90% of Cats (Over 80% of wounds get infected)
- DOGS: ~50% of Dogs (Different species), *Pasteurella canis*
(Only 2-10% get infected)
- Small aerobic Gram-Negative bacillus
- Hard to remember antibiotic susceptibility profile,
but amoxicillin sensitive





- Treated with surgical decompression of the Index finger tendon sheath
- I.V. Unasyn[®] (Ampicillin/Silbactam) then P.O Augmentin[®] (Amoxil/Clavulnate)

SO.....???????

**WHAT
THE
HECK?**



Is going on here??



Local police (Warren)

Came to the hospital

**Looking for the man
with a pet “Panther”**





100% 



75% 



50% 



25% 



0% 

THANK YOU!!!!

Thank you!

Baby

