



Wound Infections

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Shotgun wound in torso

Wound Infections

- Disease production in infected wounds depends on
 - How virulent infecting organisms are
 - How many organisms infect the wound
 - Is the host immunocompetent
 - Nature of the wound
 - Does it contain crushed material or foreign material
 - Such wounds do not heal until foreign material is removed

Wound Infections

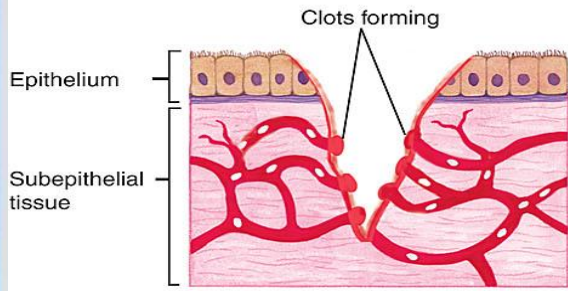
Wounds can be classified as

- Incised (incision)
 - Produced by a knife or other sharp object
- Puncture
 - From penetration of a small sharp object
- Lacerated (laceration)
 - When tissue is torn
- Contused (contusion)
 - Injury caused by a blow
 - Crushed tissue
- Burn
 - A.k.a thermal burn

Wound Infections

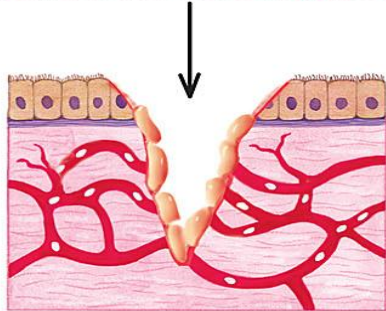
- **Wound Abscesses**

- A localized collection of pus surrounded by body tissue
- Abscess formation helps to localize infection
 - Microorganisms in abscesses are potential source of infection if they escape from localized area
- To effect cure, abscess must rupture to a body surface or be surgically drained

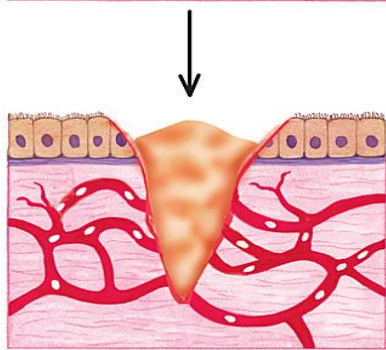


Circulating fibrinogen is converted to fibrin, resulting in clots in the severed capillaries.

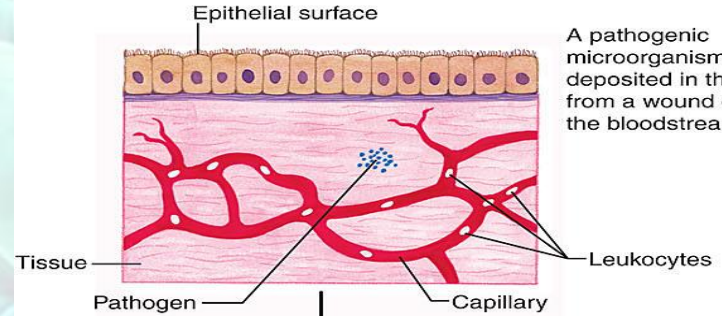
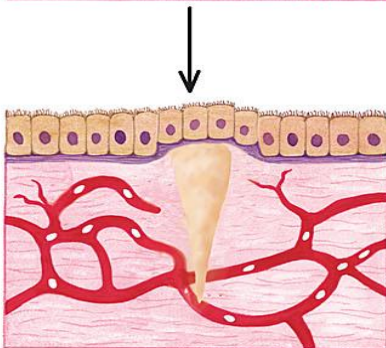
Cells from the capillaries and subepithelial tissue cells called fibroblasts multiply, forming buds of tissue protruding into the wound.



The wound is filled with bright red granulation tissue, which differentiates into abundant new capillaries and bleeds easily.

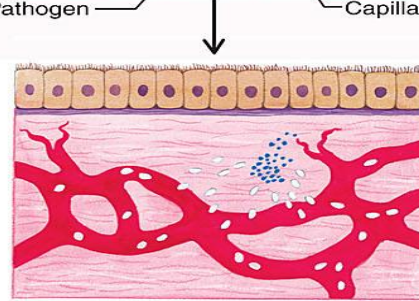


The fibroblasts multiply, producing a dense, strong substance called collagen, the main component of scar tissue. The collagen contracts and may distort the tissue in the process; epithelium covers the repaired wound.

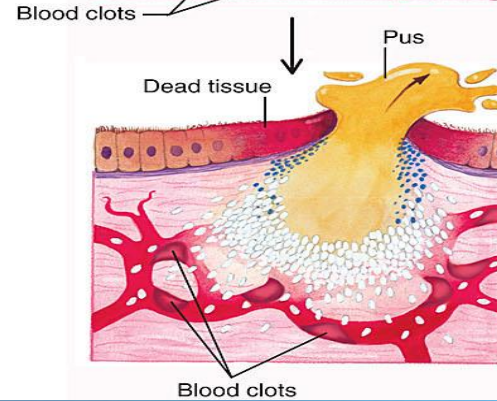
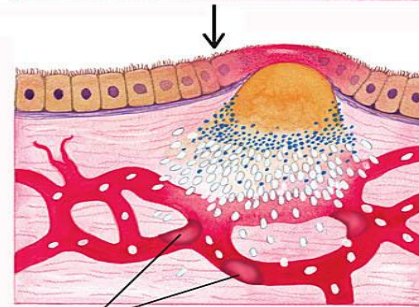


A pathogenic microorganism is deposited in the tissue from a wound or from the bloodstream.

Blood vessels dilate, and leukocytes migrate to the area of the developing infection.



Pus forms, composed of the products of tissue cell breakdown, leukocytes, and bacteria; clotting occurs in the adjacent blood vessels.



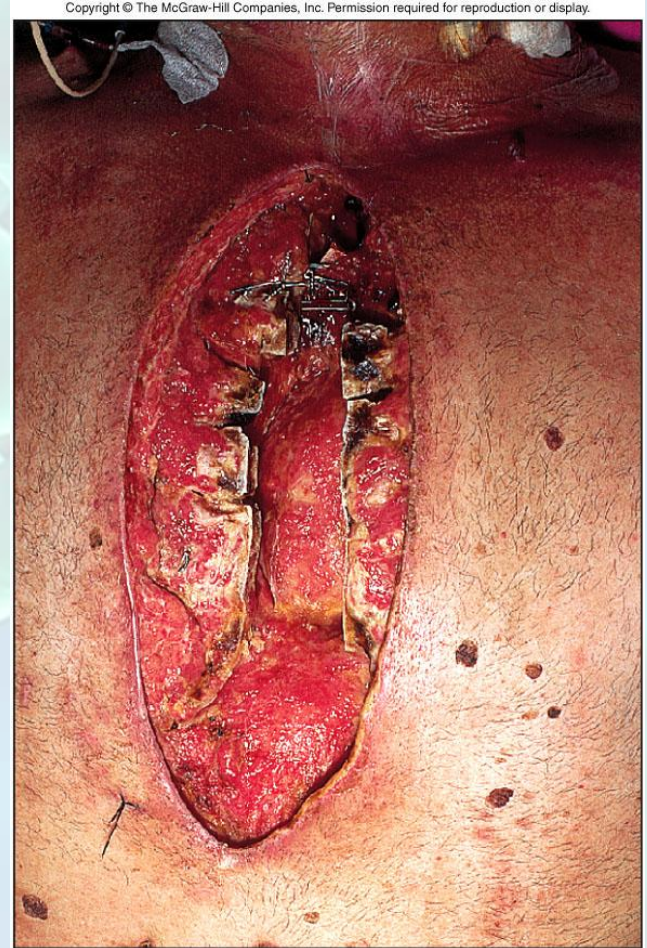
Buildup of pressure causes the abscess to expand in the direction of least resistance; if it reaches a body surface, it may rupture and discharge its contents.

Common Bacterial Wound Infections

- Common bacterial wound infections include
 - Staphylococcal wound infections
 - Group A Streptococcal wound infections
 - *Pseudomonas aeruginosa* infections
- Consequences of wound infection include
 - Delay in healing
 - Formation of abscess
 - Extension of bacteria or their products to adjacent tissues or bloodstream

Staphylococcal Wound Infections

- Staphylococci are leading cause of wound infections
- Bacteria commonly present in nose and on skin
- More than 30 recognized strains
 - Two account for most human infections
 - *S. aureus*
 - *S. epidermidis*



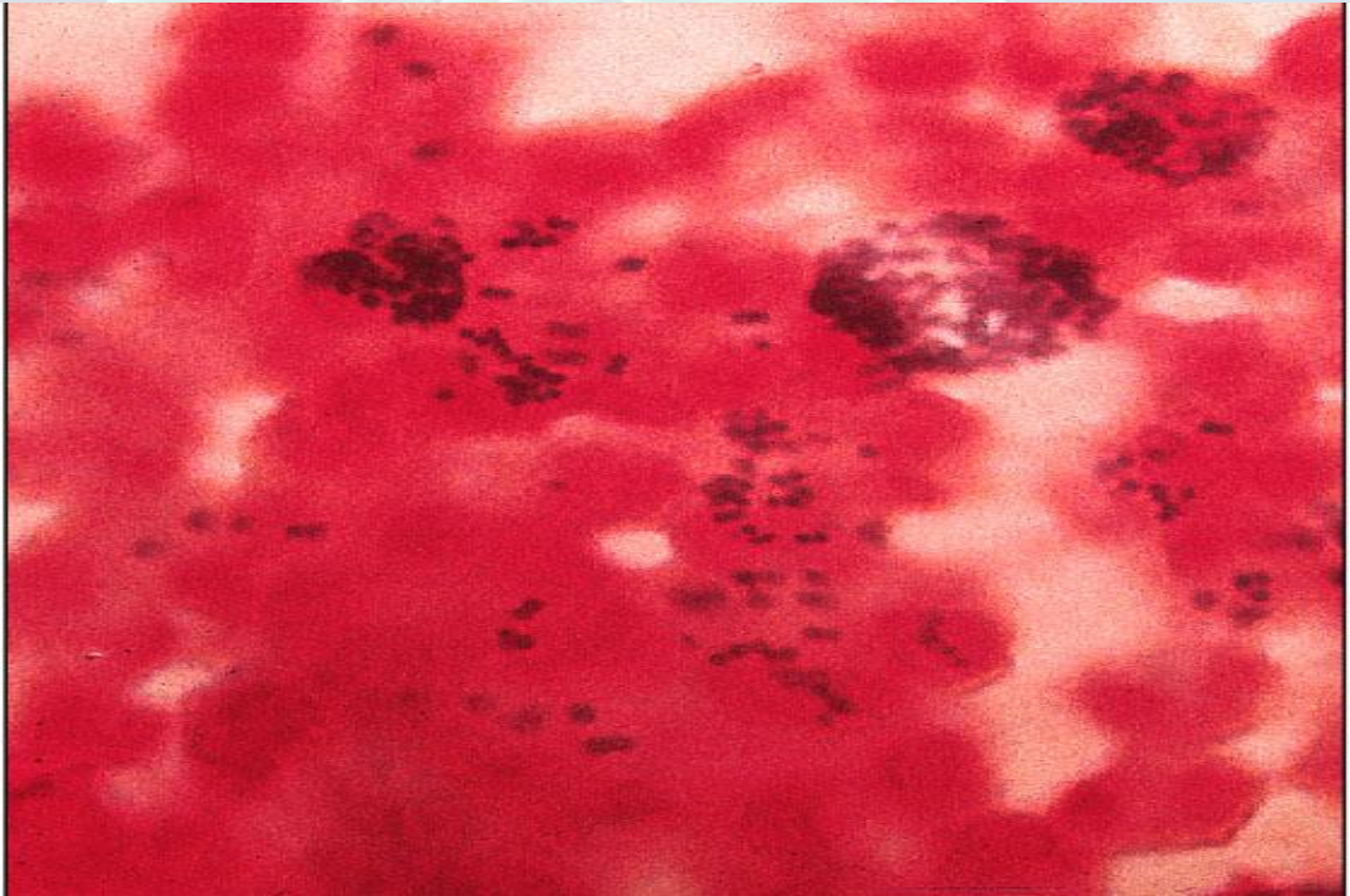
Staphylococcal Wound Infections

- Symptoms
 - Bacteria are pyogenic
 - Produce pus
 - Infection causes
 - Inflammation
 - Fever in cases where infection has spread
 - Some strains produce toxic shock syndrome

Staphylococcal Wound Infections

- Causative Agent - Staphylococci
 - Gram-positive cocci in clusters
 - Grow aerobically or anaerobically (facultative)
 - Salt tolerant
 - Allows survival in numerous foods
 - Most important species are *S. aureus* and *S. epidermidis*
 - Both survive well
 - Easy to transfer from person to person

Staphylococcus aureus in pus – dark areas are *staph.* And the red area is leukocytes



Staphylococcal Wound Infections

- Causative Agent - *S. aureus*
 - Virulence due to the production of extracellular products
 - Coagulase - Causes blood clotting to evade phagocytosis
 - Clumping factor - Aids in bacterial wound colonization
 - Protein A - Hide bacteria from phagocytic cells
 - α toxin - Produces hole in host cell membrane

Staphylococcal Wound Infections

- Pathogenesis - *S. aureus*
 - Multiple virulence factors produce signs of infection
 - Clumping factors coagulase and protein A
 - Attach organism to clots and tissue
 - Coat organism with host protein
 - Hide from phagocytosis
 - Systemic spread can lead to abscesses in other tissue
 - Commonly heart and joints
 - Certain species produce toxins
 - Toxin in blood acts as superantigen
 - Can lead to toxic shock syndrome

Staphylococcal Wound Infections

- Causative Agent - *S. epidermidis*
 - Bacteria have little or no invasive ability
 - Maintained on skin surface
 - Introduced into body from wound
 - Example: surgical incision
 - Internalized strains bind and allow colonization of indwelling devices
 - Colonization produces biofilm which protects organism from phagocytosis

Staphylococcal Wound Infections

- Pathogenesis - *S. epidermidis*
 - Infections usually cleared by healthy immune system
 - Organisms can migrate to heart and other tissues
 - Organisms from biofilms carried in bloodstream
 - Can cause subacute bacterial endocarditis or multiple tissue abscesses
 - Generally in immunocompromised

Staphylococcal Wound Infections

- Epidemiology
 - Nasal carriers 2 to 7 times greater risk of surgical wound infection
 - 30% to 100% due to patient's own flora
 - Factors associated with infection include
 - Advanced age
 - Immunosuppression or poor general health
 - Prolonged postoperative hospital stay

Staphylococcal Wound Infections

- Prevention
 - Prevention of infection is directed at
 - Cleansing wound
 - Removing dirt and crushed tissue
 - Prompt closure
 - Pre-surgical antistaphylococcal medication
 - Surgical wound infections reduced by half

Staphylococcal Wound Infections

- Treatment
 - Treatment can be problematic
 - Many strains develop resistance to antibiotics
 - Most strains are resistant to penicillin
 - Many strains treated with anti β lactamase penicillins and vancomycin
 - Vancomycin resistant strain identified in 1997

Group A Streptococcal Infections

- Also known as “flesh eaters”
- Primary pathogen is *S. pyogenes*
 - Can cause rapidly deteriorating disease and death
 - Not a lot of antimicrobial resistance

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Group A Streptococcal Infections

- More severe infections called invasive
- Include
 - Pneumonia
 - Meningitis
 - Puerperal (childbirth fever)
 - Necrotizing fasciitis (flesh eating disease)
 - Streptococcal toxic shock

Group A Streptococcal Infections

- Symptoms
 - Acute pain at the site of the wound
 - Swelling
 - Fever and confusion
 - Overlying skin tightens and becomes discolored
 - Shock and death
 - In the absence of treatment

Group A Streptococcal Infections

- Causative Agent - *S. pyogenes*
 - Some strains cause invasive infection
 - These are more virulent than strains that do not
- Pathogenesis
 - Subcutaneous fascia is destroyed in necrotizing fasciitis
 - Muscle tissue is also destroyed when bacteria penetrate muscle tissue
 - Organisms multiply and produce toxic products
 - Organisms and toxic products enter bloodstream
 - Can cause shock

Group A Streptococcal Infections

- Epidemiology
 - “Flesh eating” infections have been described since the 5th century B.C.
 - 2,000 cases reported during Civil War
 - Cases generally sporadic
 - Small epidemics have occurred
 - Outbreak in San Francisco in 1996
 - Traced to use of contaminated “black tar” heroine
 - Approximately 9,000 cases of invasive *S. pyogenes* in 2002
 - Resulted in 1080 deaths
 - 135 from necrotizing fasciitis

Group A Streptococcal Infections

- Prevention and Treatment
 - No proven prevention measures
 - Urgent surgery required due to rapidity of toxin spread
 - Amputation is sometimes required
 - Penicillin is still an effective treatment
 - Must be given early
 - Has little or no effect on bacteria in necrotic tissues
 - No effect on toxin
 - Surgery may still be necessary



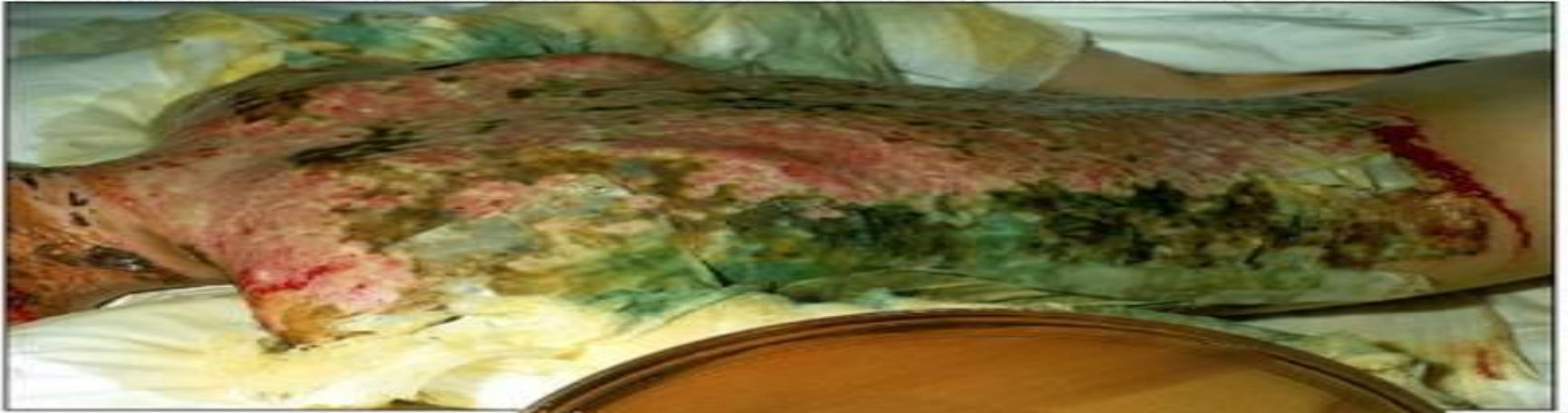
- Boy who contracted flesh-eating bacteria getting a new face
- Flesh-eating bacteria forces boy to have 28 surgeries

Pseudomonas aeruginosa Infections

- *P. aeruginosa* is an opportunistic pathogen
- Major cause of nosocomial infections
 - Occasional cause of community acquired infections
- Nosocomial infections include
 - Lung infections
 - Burn infections

Pseudomonas aeruginosa Infections

- Community acquired infections include
 - Rash and external ear infections
 - Obtained from contaminated swimming pools and hot tubs
 - Infection of foot bones
 - Eye infections
 - Heart valve infections
 - Lung biofilms



(a)



(b)



(c)

10 μm

Pseudomonas aeruginosa

Symptoms

- Change in tissue color
 - *P. aeruginosa* releases pigments that often color tissues green
- Chills, fever, skin lesions and shock
 - Caused by bacterial infection in bloodstream

Pseudomonas aeruginosa

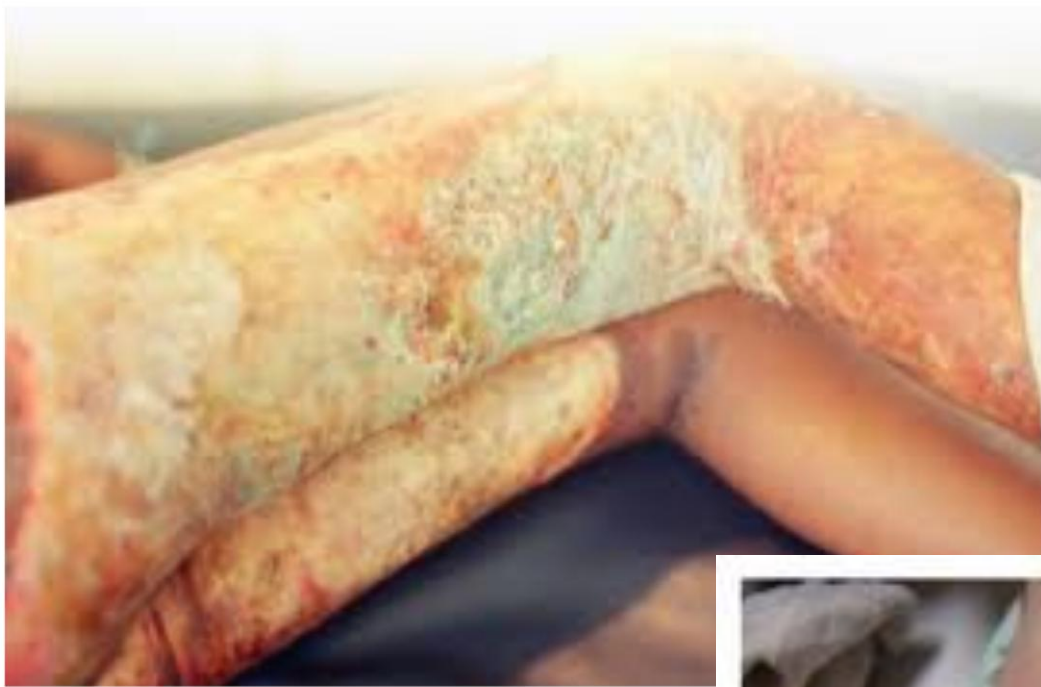
- Causative Agent - *Pseudomonas aeruginosa*
 - Generally aerobic
 - Produces numerous pigments that produce color change in tissues
- Pathogenesis
 - Overall effect is tissue damage, prevention of healing and increased risk of septic shock
 - Some strains produce enzymes and toxins to enhance virulence

Pseudomonas aeruginosa

- Epidemiology
 - *P. aeruginosa* is widespread in nature
 - Found extensively in soil, water and on plants
 - Introduced in hospitals, on the soles of shoes, on ornamental plants and flowers and on produce
 - Bacteria will persist in dampness or standing water
 - Contaminates soaps, ointments, eye drops, swimming pools and hospital equipment

Pseudomonas aeruginosa

- Treatment
 - Prompt wound care
 - Removal of dead tissue from burns
 - Followed by application of antibacterial cream
 - Silver sulfadiazine
 - Established infections are extremely difficult to treat
 - *P. aeruginosa* is multi-drug resistant
 - Medications must be administered intravenously at high doses



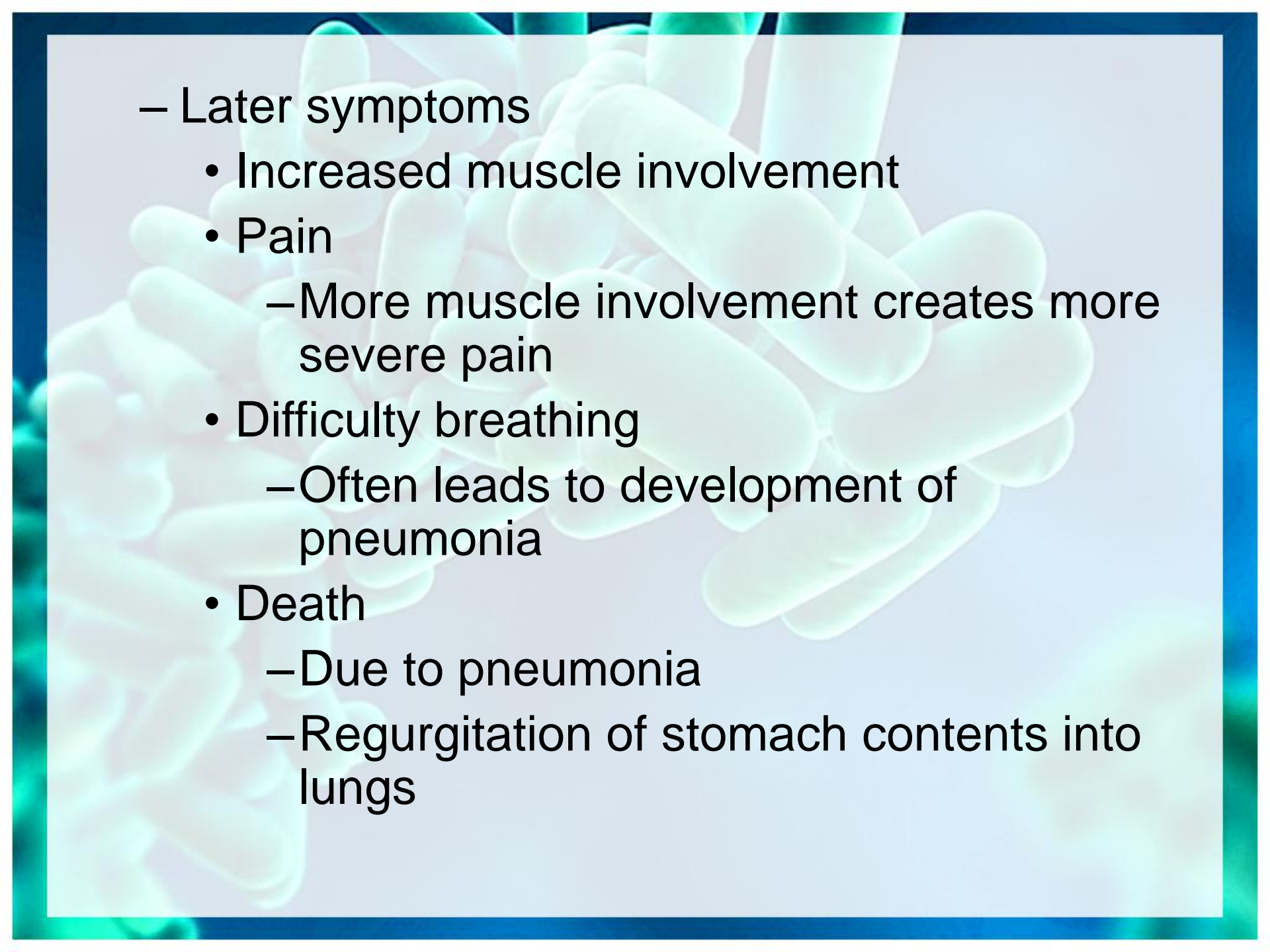
Tetanus

- Also known as “Lockjaw”
- Frequently fatal; however, rare in the developed world
- Bacterial spores prevalent in dust and soil
 - Difficult to avoid exposure



Tetanus Symptoms

- Symptoms
 - Divided into early and late symptoms
 - Early symptoms
 - Restlessness
 - Irritability
 - Difficulty swallowing
 - Contraction of jaw muscles
 - Convulsions
 - Particularly in children

- 
- Later symptoms
 - Increased muscle involvement
 - Pain
 - More muscle involvement creates more severe pain
 - Difficulty breathing
 - Often leads to development of pneumonia
 - Death
 - Due to pneumonia
 - Regurgitation of stomach contents into lungs

Tetanus

- Causative Organism - *Clostridium tetini*
- Pathogenesis
 - Colonization is generally contained to wound
 - Bacteria produce toxin
 - Toxin = tetanospasmin
 - Toxin is responsible for pathological effects
 - Tetanospasmin blocks inhibition of motor neurons, causing paralysis
 - Muscle contraction is uncontrolled
 - Muscles do not relax
 - Paralysis usually begins in the jaw

Tetanus - Epidemiology

- *C. tetani* found in dirt and dust and gastro intestinal tract of humans and other animals
- Nearly half of infections result from puncture wounds including
 - Body piercing, tattooing, animal bites, injected drug abuse
- 30 to 60 cases in United States annually with 25% mortality rate
- Immunization has decreased incidences in economically advanced countries -----
Infection more common in developing countries

- Prevention
 - Immunization best preventative
 - Vaccine is inactivated tetanospasmin
 - Given in combination with diphtheria and pertusis vaccine – DPT

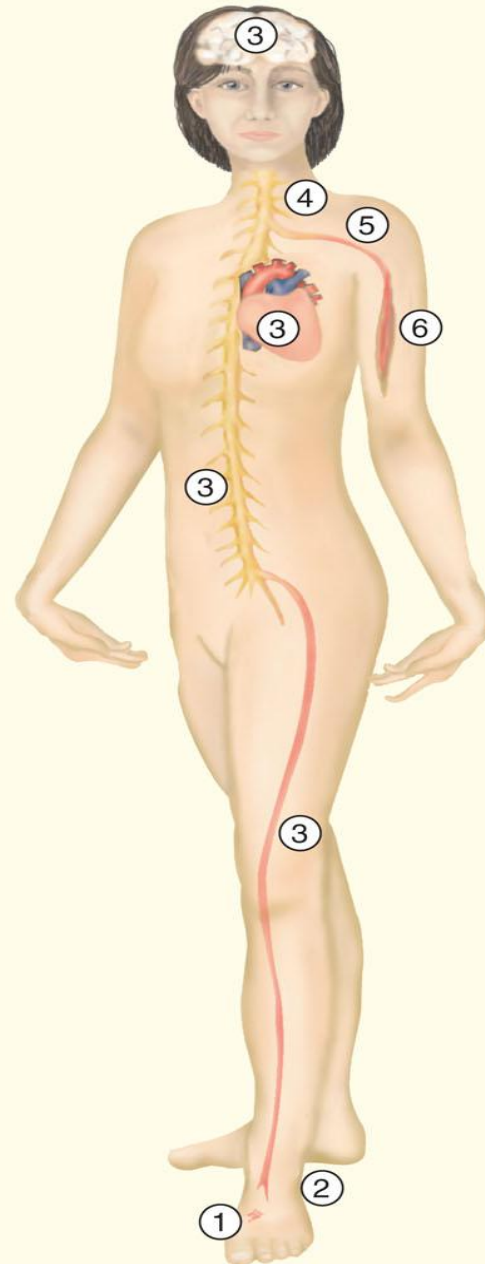


Tetanus - Treatment

- Thoroughly clean wound
 - Remove all dead tissue
- Antimicrobial treatment given to kill multiplying bacteria
 - Metronidazole
 - Antimicrobials do not kill endospores
 - Antitoxin - antibody against tetanospasmin
 - Neutralizes toxin not attached to nerve cells

TABLE 24.3 “Lockjaw” (Tetanus)

- ① *Clostridium tetani* spores from dust or dirt enter a wound.
- ② In wounds sufficiently anaerobic, the spores germinate, vegetative bacteria release an exotoxin called tetanospasmin.
- ③ Tetanospasmin is carried to the central nervous system by motor nerve axons or by the bloodstream.
- ④ The toxin prevents any inhibitory neurons it reaches from functioning.
- ⑤ The corresponding neurons, which cause muscles to contract, act unopposed by inhibitory neurons.
- ⑥ The result is a sustained, painful cramplike muscle spasm.



Symptoms	Restlessness, irritability, difficulty swallowing; muscle pain and spasm in jaw, abdomen, back, or entire body
Incubation period	3 days to 3 weeks; average 8 days
Causative agent	<i>Clostridium tetani</i> , an anaerobic, spore-forming, Gram-positive rod
Pathogenesis	Tetanus results from tetanospasmin, an exotoxin produced by the bacterium. The toxin is carried to brain and spinal cord by motor nerve axons or circulating blood; toxin acts against nerve cells that normally inhibit muscle contraction. Other nerves that normally cause muscle contraction then act unopposed, causing muscle spasms.
Epidemiology	Organisms common in soil; spores contaminate wounds, germinate in those having anaerobic conditions, particularly dirty or puncture wounds.
Prevention and treatment	Immunization of children at ages 2 months, 4 months, 6 months, 18 months; booster dose at time of entering school and at 10-year intervals after that; tetanus immune globulin (TIG), cleaning wound. Treatment: metronidazole, tetanus antitoxin.

Clostridial Myonecrosis – Gas Gangrene

- Endospores of causative bacillus are innumerable
 - Spores found in nearly all soil or dusty surface
- Primarily disease of wartime
 - Due to neglected wounds containing debris



Gas Gangrene Symptoms

- Begin abruptly
- Rapidly increasing pain
 - Pain localized to area of wound
- Increased swelling
- Thin, bloody fluid leaks from wound
 - Fluid is often brownish and may appear frothy
 - Frothy appearance is due to gas production by infection bacteria
- Skin becomes stretched and mottled with black spots
- Patient appears very ill but alert
 - Delirium and coma occur late in illness followed by death

Gas Gangrene

- Causative Agent
 - Several species of *Clostridium*
 - Most common offender, *C. perfringens*
 - Two factors foster development of clostridial myonecrosis
 - Presence of dirt and dead tissue in wound
 - Long delays in treatment
 - Immediate medical attention in severe wound is especially important

Gas Gangrene

- Pathogenesis
 - Bacteria is a toxin producer
 - Toxin attacks host cell membrane
 - Toxin diffuses and kills tissue cells
 - *C. perfringens* unable to grow in healthy tissue
 - Survives well in dead or poorly oxygenated tissue
 - Releases toxin in tissue
 - Bacteria produces gas through fermentation
 - Gas accumulates in tissue, contributing to spread

Gas Gangrene

- Epidemiology
 - *C. perfringens* found in feces
 - Of humans and animals
 - Present in vaginal tract
 - Established in the vaginal tract of 1% to 9% of healthy women
 - Gas gangrene of uterus
 - Fairly common after self-induced abortion
 - Rarely seen after miscarriage and childbirth

Gas Gangrene

- Prevention
 - Vaccine unavailable
 - Prompt cleaning and debridement of wound
 - Surgical removal of dead and damaged tissue
 - Highly effective at preventing disease



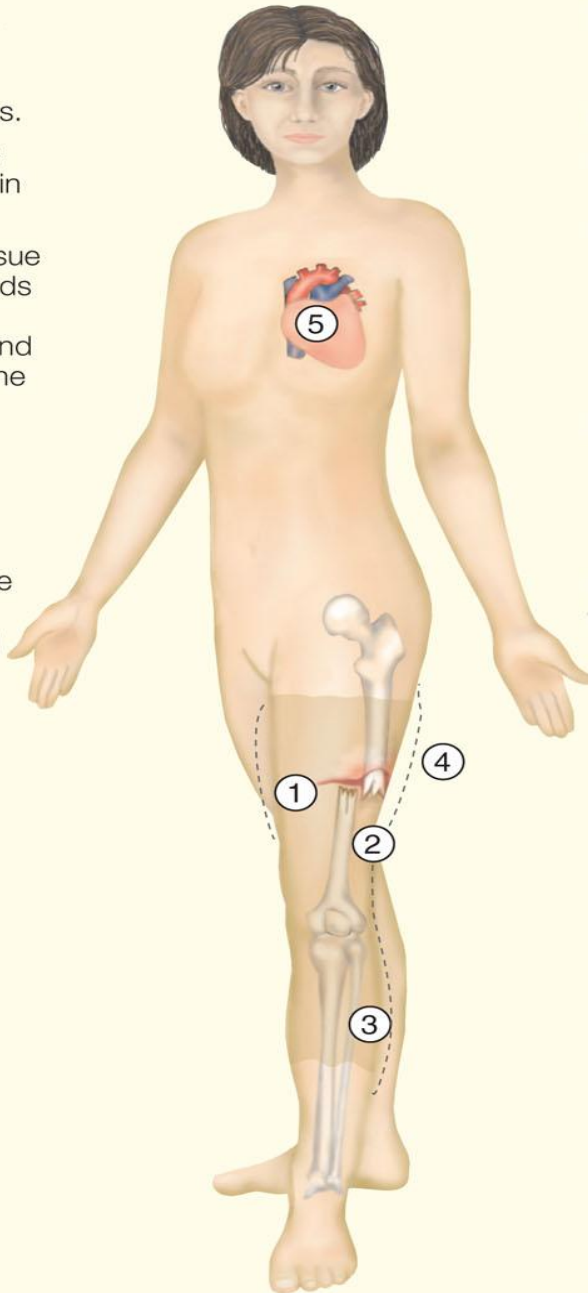
Gas Gangrene

- Treatment
 - Treatment depends primarily on prompt removal of affected tissue
 - Amputation may be required
 - Hyperbaric oxygen treatment
 - Inhibits growth of clostridia
 - Stops release of toxin
 - Penicillin is given to halt bacterial growth
 - No growth = no toxin production



TABLE 24.4 Gas Gangrene (Clostridial Myonecrosis)

- ① *Clostridium perfringens* spores enter a wound having two essential characteristics: dead tissue and anaerobic conditions.
- ② The spores germinate, and the vegetative bacteria multiplying in dead tissue produce α -toxin.
- ③ α -toxin diffuses into normal tissue and kills it. The infection expands into the newly killed tissue, the bacteria utilizing amino acids and growth factors released from the tissue by bacterial enzymes.
- ④ Swelling and gas produced by fermenting amino acids and muscle glycogen aid rapid progress of the infection.
- ⑤ Massive amounts of α -toxin are produced and diffuse into the bloodstream, destroying blood and other cells throughout the body.



Symptoms	Severe pain, gas and fluid seep from wound, blackening of overlying skin; shock and death commonly follow
Incubation period	Usually 1 to 5 days
Causative agent	Usually <i>Clostridium perfringens</i> ; other clostridia less frequently
Pathogenesis	Organism grows in dead and poorly oxygenated tissue and releases α -toxin; toxin kills leukocytes and normal tissue cells by degrading the lecithin component of their cell membranes; involvement of muscle causes shock by unknown mechanism.
Epidemiology	Wounds of war; dirt contamination of wounds, tissue death, impaired circulation to tissue as in persons with poor circulation from diabetes and arteriosclerosis; self-induced abortions.
Prevention and treatment	Prompt cleaning and debridement of wounds is preventive; no vaccine available. Treatment: surgical removal of dirt and dead tissues of primary importance; hyperbaric oxygen of possible value; antibiotics to kill vegetative <i>C. perfringens</i> of marginal value.

Actinomycosis – “Lumpy jaw”

- Symptoms
 - Progresses slowly
 - Sometimes includes painful swelling under the skin
 - Swollen regions open and drain pus
 - Chronic condition
 - Openings usually heal
 - Lesions reappear at the same or nearby region within days or weeks
 - Most cases involve the jaw or neck
 - Recurrent lesions may develop on chest and abdominal wall or genital tract of women
 - Scars and swelling give rise to name “lumpy jaw”



Actinomyces

- Pathogenesis
 - *A. israelii* cannot penetrate healthy mucosa
 - Disease progresses to skin and can penetrate bone or central nervous system
 - In tissue, culture grows as dense yellow colonies
 - Good identifier for diagnosis
 - Nearly 50% of cases originate in mouth

Actinomyces

- Epidemiology
 - Can be normal flora
 - Found in mouth mucosa, upper respiratory tract, intestine and vagina
 - Commonly found in gingival crevices
 - Disease is sporadic and non-communicable

Actinomyces

- Prevention and Treatment
 - No proven prevention
 - Responds to numerous antibacterials
 - Penicillin and tetracycline
 - Treatment must be given orally for several weeks or months
 - This is due to the slow growing nature of the organism

TABLE 24.5

“Lumpy Jaw” Actinomycosis

Symptoms	Chronic disease; recurrent, sometimes painful swellings open and drain pus, heal with scarring; usually involves face and neck; chest, abdomen, and pelvis other common sites
Causative agent	<i>Actinomyces israelii</i> , a filamentous, branching, Gram-positive, slow-growing, anaerobic bacterium
Incubation period	Months (usually indeterminate)
Pathogenesis	Usually begins in a mouth wound, extends without regard to tissue boundaries to the face, neck, or upper chest; sometimes begins in the lung, intestine, or female pelvis. <i>A. israelii</i> always accompanied by normal microbiota. In tissue, grows as dense yellowish colonies called sulfur granules.
Epidemiology	No person-to-person spread. <i>A. israelii</i> commonly part of normal mouth, upper respiratory, intestine, and vagina microbiota. Dental procedures, intestinal surgery, insertion of IUDs can initiate infections.
Prevention and treatment	No proven preventive measures. Because of its slow growth. <i>A. israelii</i> infections require prolonged treatment; a number of antibacterial medications are effective.

Pasteurella multocida

- Responsible for bite infections from numerous animals including
 - Dogs
 - Cats
 - Monkeys
 - Humans
- More common than rabies

Pasteurella multocida

- Symptoms
 - No reliable symptoms that distinguish one bite from another
 - Generalized symptoms include
 - Spreading redness
 - Tenderness
 - Swelling of adjacent tissues
 - Pus discharge

Pasteurella multocida

- Epidemiology
 - Best known as cause of devastating chicken disease
 - Fowl cholera
 - Also causes disease in a number of other animals
 - Epidemics of fatal pneumonia in rabbits, cattle, sheep and mice
 - Healthy animals carry organism as part of oral and respiratory normal flora
 - Diseased and healthy animals act as reservoir for human infection

Pasteurella multocida

- Prevention and Treatment
 - No vaccine available for human use
 - Immediate cleansing and prompt medical attention
 - Usually prevents development of serious infection
 - Organism is susceptible to penicillin
 - Usually Augmentin is administered before diagnosis
 - Other antibacterials are effective if given early

TABLE 24.6***Pasteurella multocida* Bite Wound Infections**

Symptoms	Spreading redness, tenderness, swelling, discharge of pus
Incubation period	24 hours or less
Causative agent	<i>Pasteurella multocida</i> , a Gram-negative, facultatively anaerobic, encapsulated coccobacillus
Pathogenesis	Introduced by bite, <i>P. multocida</i> attaches to tissue, resists phagocytes because of its capsule; probable cell-destroying toxin. Extensive swelling, abscess formation. Opsonins develop, allow phagocytic killing, limit spread.
Epidemiology	Carried by many animals in their mouth or upper respiratory tract.
Prevention and treatment	No vaccines to use in humans. Prompt wound care is preventive. Treatment with penicillin, other antibacterials, effective if given promptly.

Cat Scratch Disease

- Symptoms
 - Disease begins within a week of scratch or bite
 - Development of pus-filled pimple
 - Painful enlargement of lymph nodes
 - Nodes at region of wound enlarge in 1 to 7 weeks
 - In about 50% of patients, nodes become pus filled
 - Fever
 - Fever develops in about one-third of patients



Cat Scratch Disease

- Symptoms
 - Disease is self-limiting
 - Disease disappears in about 2 to 4 months
 - 10% of cases develop eye irritation
 - With local lymph node enlargement
 - Encephalitis can be a complication
 - Seizures and coma can result
 - Acute or chronic fever are associated with blood stream or heart valve infection

Cat Scratch Disease

- Pathogenesis
 - Virulence factors and disease process are not understood
 - Organism enters body through scratch or bite
 - Carried to the lymph nodes
 - Disease is arrested by immune system in most cases with systemic spread in some individuals
 - Complicating conditions can occur mostly in immunocompromised

Cat Scratch Disease

- Epidemiology
 - Mainly occurs in people under the age of 18
 - Zoonotic disease
 - Particularly from cats to humans
 - Mainly by kittens
 - Cats infected by bite of flea
 - Person-to-person spread does not occur
 - Bites and scratches usual mode of transportation
 - Asymptomatic bacteremia common in cats

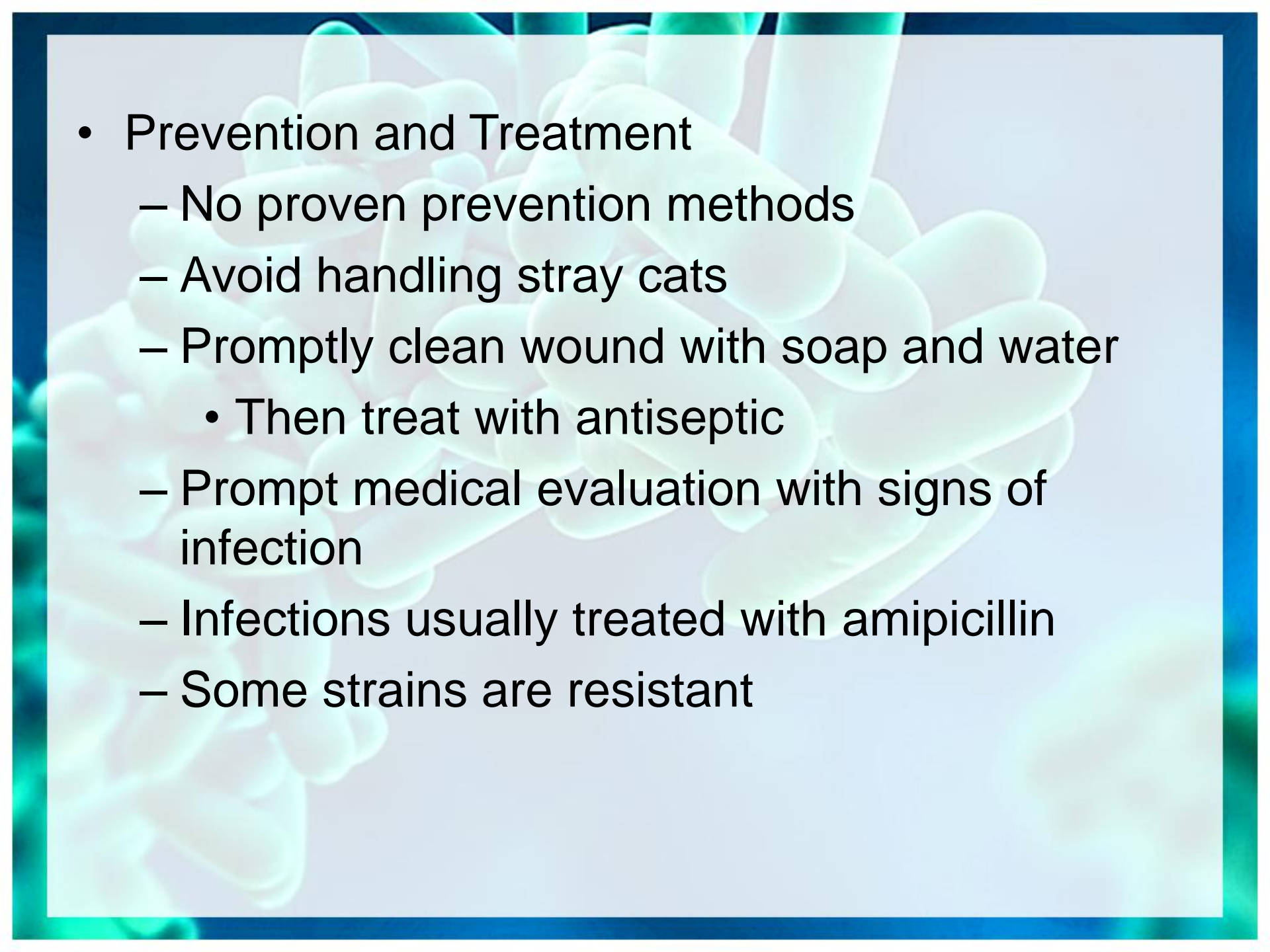
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- Prevention and Treatment
 - No proven prevention methods
 - Avoid handling stray cats
 - Promptly clean wound with soap and water
 - Then treat with antiseptic
 - Prompt medical evaluation with signs of infection
 - Infections usually treated with ampicillin
 - Some strains are resistant

TABLE 24.7**Cat Scratch Disease**

Symptoms	Pimple appears at the bite or scratch site, followed by local lymph node enlargement, fever; nodes may soften and drain pus; prolonged fever, convulsions, indicate spread to other body parts
Incubation period	Usually less than 1 week
Causative agent	<i>Bartonella henselae</i> , a tiny Gram-negative rod
Pathogenesis	The bacteria enter with cat bite or scratch and reach lymph nodes, where the disease is usually arrested. May spread by bloodstream, cause infections of the heart, brain, or other organs. Peliosis hepatis, bacillary angiomatosis mostly in those with AIDS.
Epidemiology	A zoonosis of cats, spread from one to another by cat fleas, which are biological vectors and have <i>B. henselae</i> in their feces. Infected cats usually asymptomatic, but often bacteremic. Humans are accidental hosts. No person-to-person spread. Mainly a disease of those under 18 years old.
Prevention and treatment	Avoiding rough play. Promptly wash skin breaks, apply antiseptic. Flea control in cats. Most <i>B. henselae</i> susceptible to antibacterial treatment.

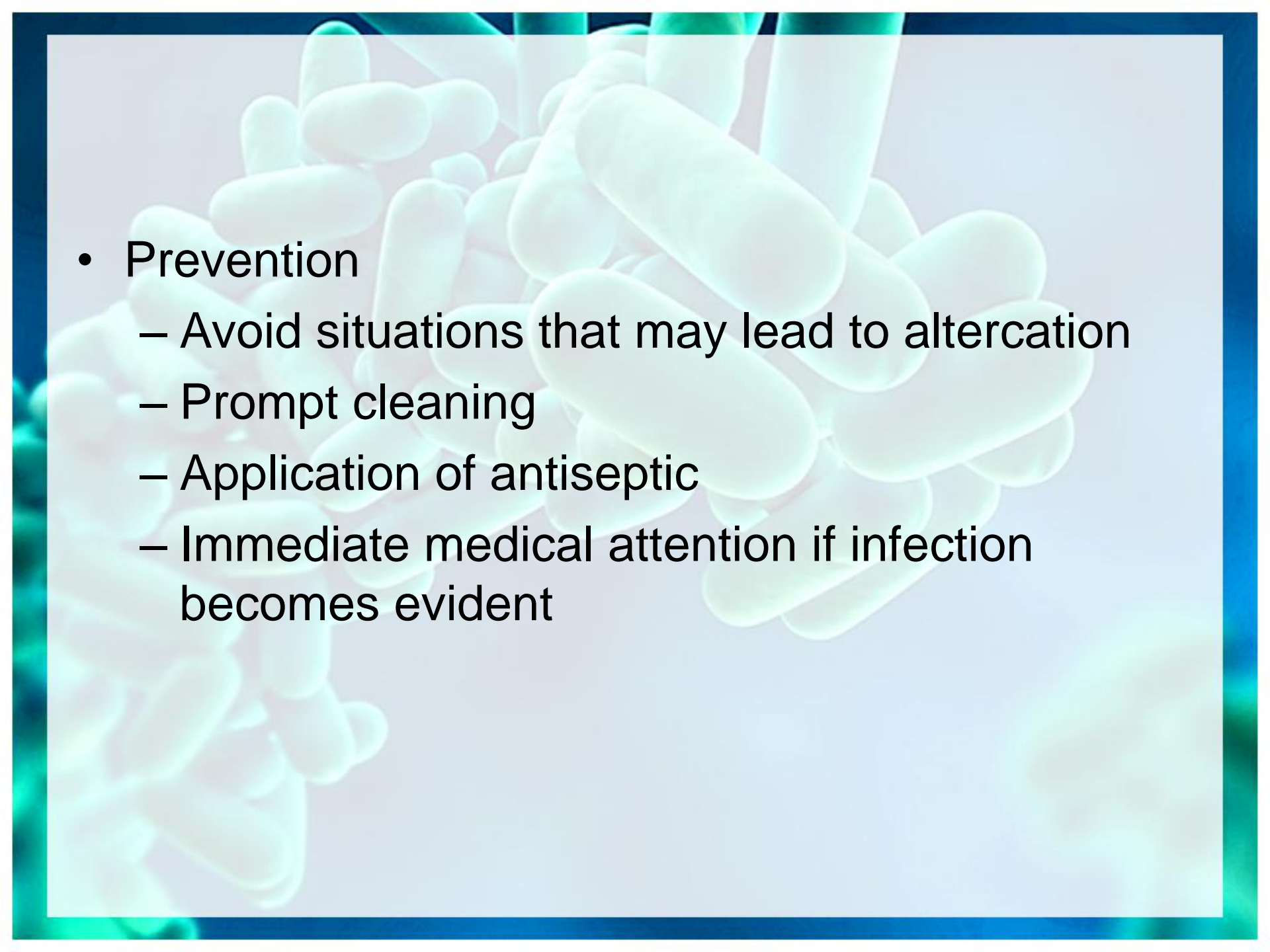
Human Bites

- Symptoms
 - Wound may appear insignificant
 - Painful with massive swelling
 - Pus discharge
 - Pus often foul smelling
 - Most wounds are on exterior of hand
 - Swelling may involve palm
 - Movement may be hampered

- Pathogenesis
 - Mouth flora generally harmless
 - Produce numerous toxins and enzymes
 - » Toxins and enzymes destroy tissues and immune complexes
- Epidemiology
 - Most serious human bite results from violent altercations
 - Risk of infection increases when biting individual has poor oral hygiene
 - Bites by small children are usually inconsequential

- Epidemiology
 - Most serious human bite results from violent altercations
 - Risk of infection increases when biting individual has poor oral hygiene
 - Bites by small children are usually inconsequential



- 
- A microscopic view of numerous rod-shaped bacteria, likely bacilli, in shades of blue and green. The bacteria are scattered across the frame, with some appearing in sharp focus and others blurred in the background. The overall image has a soft, ethereal quality with a light blue and green color palette.
- Prevention
 - Avoid situations that may lead to altercation
 - Prompt cleaning
 - Application of antiseptic
 - Immediate medical attention if infection becomes evident

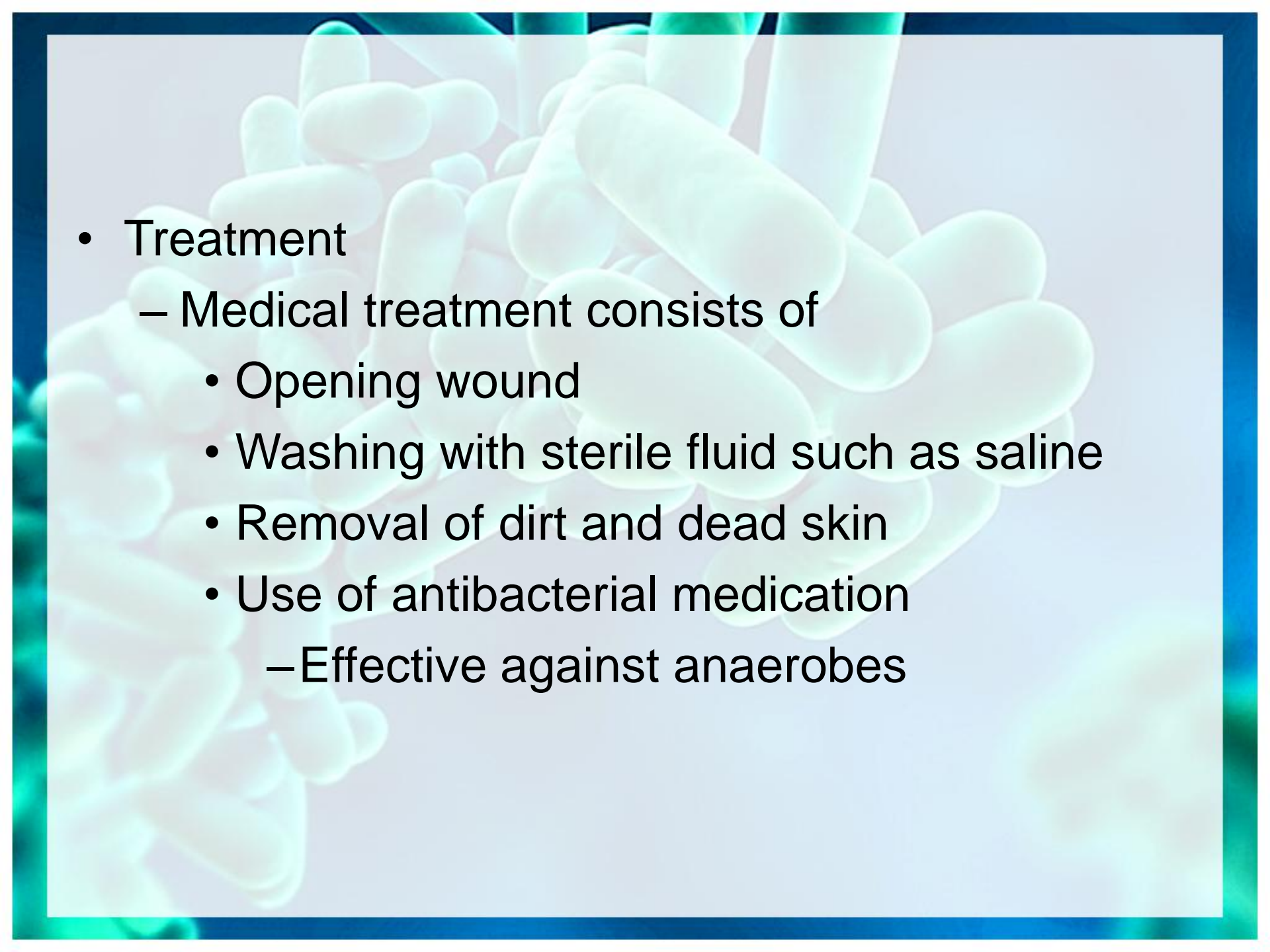
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- A microscopic view of various bacteria, including rod-shaped and spherical forms, some with flagella, set against a light blue background. The bacteria are rendered in a semi-transparent, glowing style.
- Treatment
 - Medical treatment consists of
 - Opening wound
 - Washing with sterile fluid such as saline
 - Removal of dirt and dead skin
 - Use of antibacterial medication
 - Effective against anaerobes

TABLE 24.9

Human Bite Wound Infections

Symptoms	Rapid onset, pain, massive swelling, drainage of foul-smelling pus
Incubation period	Usually 6 to 24 hours
Causative agent	Mixed mouth flora: anaerobic streptococci, fusiforms, spirochetes, anaerobic Gram-negative rods; sometimes <i>Staphylococcus aureus</i>
Pathogenesis	Various mouth bacteria act synergistically to destroy tissue.
Epidemiology	Alcohol-related violence; forcible restraint; poor mouth care and extensive dental disease.
Prevention and treatment	No proven preventive measures except to avoid altercations. Prompt cleansing of wound and application of antiseptic is advised. Treatment is usually surgical.

Sporotrichosis

- Also known as “rose gardener’s disease”
- Distributed worldwide
- Associated with puncture wound from vegetation
- Sporadic
 - Occurs in specific occupations
 - Epidemics have occurred in United States



Sporotrichosis - Symptoms

- Hand or arm primary site of involvement
 - Trunk, legs and face can also be infected
- Chronic ulceration occurs at site of wound
- Development of ulcerating nodules
 - Develop sequentially towards center of body
- Lymph node enlargement
- Healthy individuals rarely become ill
 - Can be life threatening to immunocompromised

Sporotrichosis

- Pathogenesis
 - Spores introduced via injury caused by plant material
 - Incubation period ranges 1 to 3 weeks
 - Small nodule forms
 - Due to multiplying fungi
 - Lesion enlarges
 - Ulcerates and produces red, bleeding skin defect

Sporotrichosis

- Epidemiology
 - Fungi distributed worldwide
 - Mostly in warmer and temperate regions
 - Mostly in Mississippi and Missouri River Valley in United States
 - Occupational disease of
 - Farmers
 - Carpenters
 - Gardeners and
 - Greenhouse workers
 - Not reported – incidence unknown
 - Risk factors of disease include
 - Diabetes, immunosuppression and alcoholism

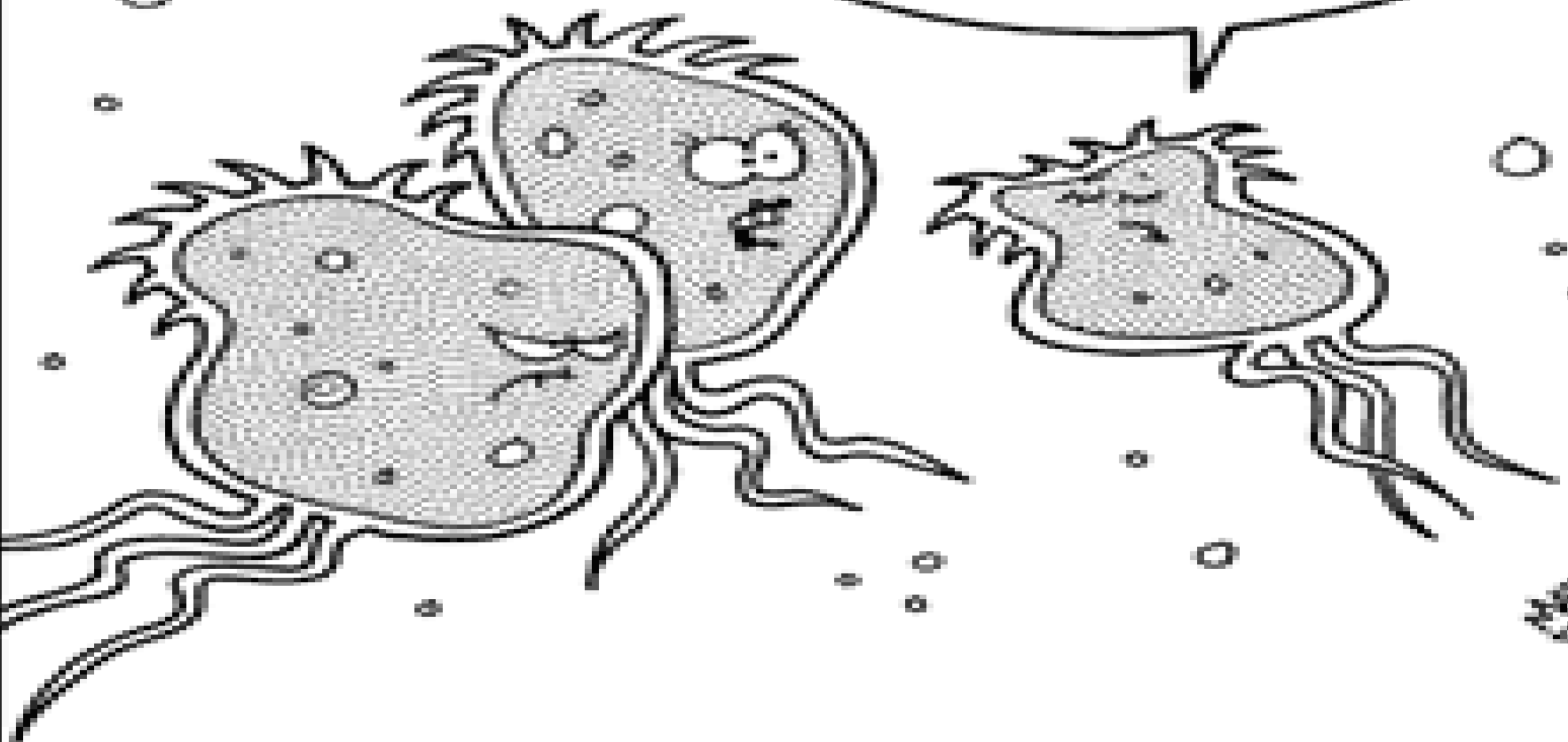
Sporotrichosis - Pathogenesis

- Ulceration process repeats itself
- Disease progression usually follows flow of lymphatic vessel
- In healthy individuals process does not proceed beyond lymph node
- Without treatment disease becomes chronic

Sporotrichosis

- Prevention and Treatment
 - Protective clothing
 - Gloves and long-sleeved shirt
 - Disease is often misdiagnosed
 - Leads to delayed and inappropriate treatment
 - Usually cured with oral potassium iodide (KI)
 - Enhances body's ability to reject fungus
 - Itraconazole and amphotericin B used in rare cases
 - Generally when disease is systemic

MOM, DAD...
I'M GOING
VEGAN...



WHEN FLESH-EATING
BACTERIA REBEL

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