CHS 2413
Pathology and Physiopathology

Assoc.Prof.Dr. Thavatchai Kamoltham

MSc.MD.FICS.FRCST.Dr.PH

Environmental pathology

- Environmental pollution
- Injury by chemical agents
- Injury by physical agents
- Nutritional diseases

Environmental pollution

Environmental pollution

- Air pollution
- smog (smoke+fog)
- 2 types - reducing smog - coal combustion - sulfur oxides+particulates

  - photochemical oxidant smog - incompletely burned hydrocarbons - CO, CO_2,

acute or chronic inflammation, emphysema, asthma, pneumoconiosis, tumors

Contents of Toxic Waste Dumps

Acetone	**DDT, DDE, DDD**
Aldrin/Dieldrin	**1,1 and 1,2-Dichloroethane**
Arsenic	**Lead**
Barium	**Mercury**
Benzene	**Methylene chloride**
2-Butanone	**Nickel**
Cadmium	**Pentachlorophenol**
Carbon tetrachloride	**Polychlorinated biphenyls**
Chlordane	**Tri- and Tetrachloroethylene**
Chloroform	**Toluene**
Chromium	**Vinyl Chloride**
Cyanide	**Zinc**

Indoor Air Pollution

- Carbon Monoxide CO
- Nitrogen Dioxide NO_2 (from acid rain)
- Wood Smoke
- Formaldehyde
- Manufactured Mineral Fibers
- Bioaerosols

Lead

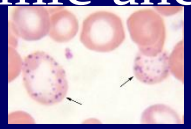
- Lead is classified as a heavy metal (others include mercury, arsenic, and cadmium)
- Source of exposure
  - lead paint
  - lead solder in plumbing (older houses)
  - lead-glazed ceramics
  - industrial exposure
- Route of exposure
  - inhalation with industrial exposure
  - ingestion with household exposure

Lead Distribution and Excretion

- Lead is a divalent cation that is taken up by bone and developing teeth in children (80% to 85%)
  - Half-life of lead in bone is 30 years
- Blood accumulates 5% to 10% of lead, but lead is rapidly cleared from the blood
  - lead in blood indicates recent exposure
  - blood level does not allow the determination of total body burden
- Remainder is distributed in the soft tissues
- Excretion is via the kidneys

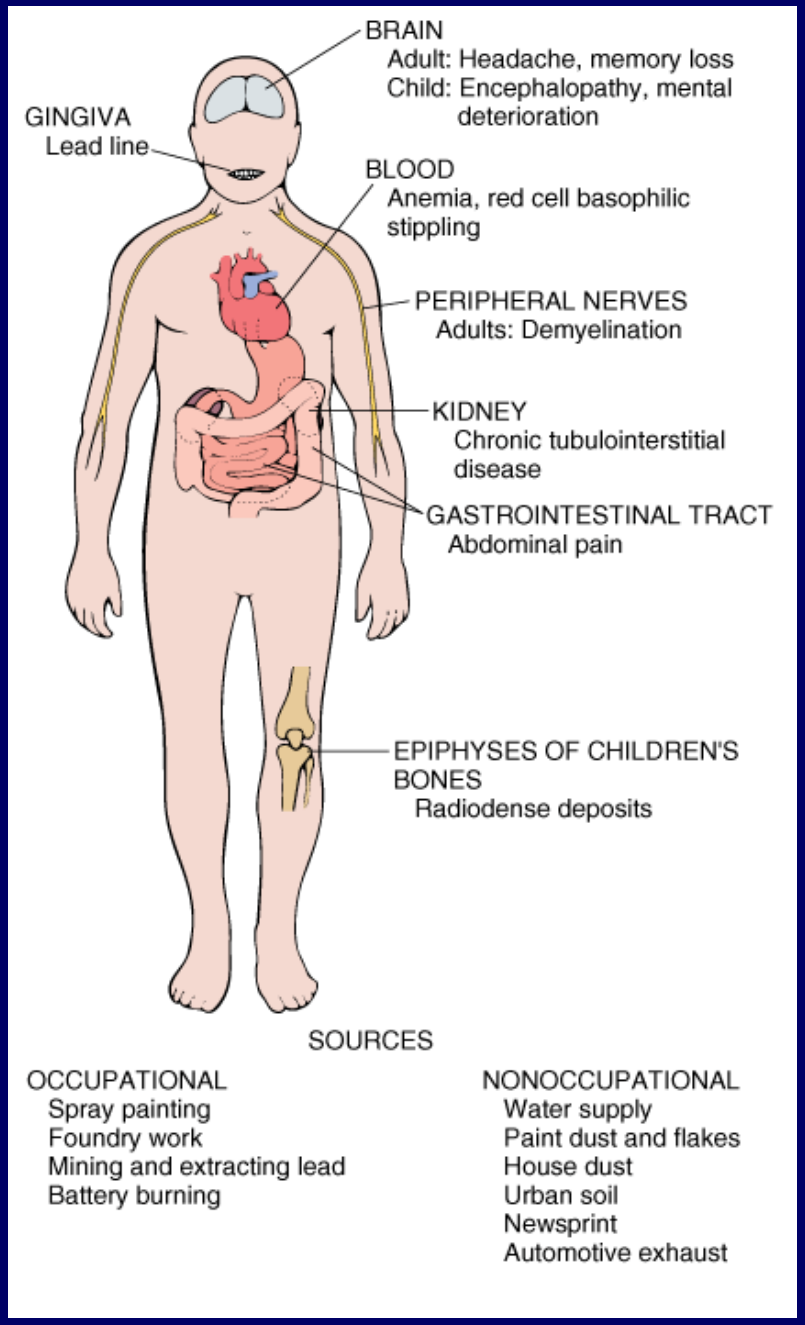
Effects of Lead

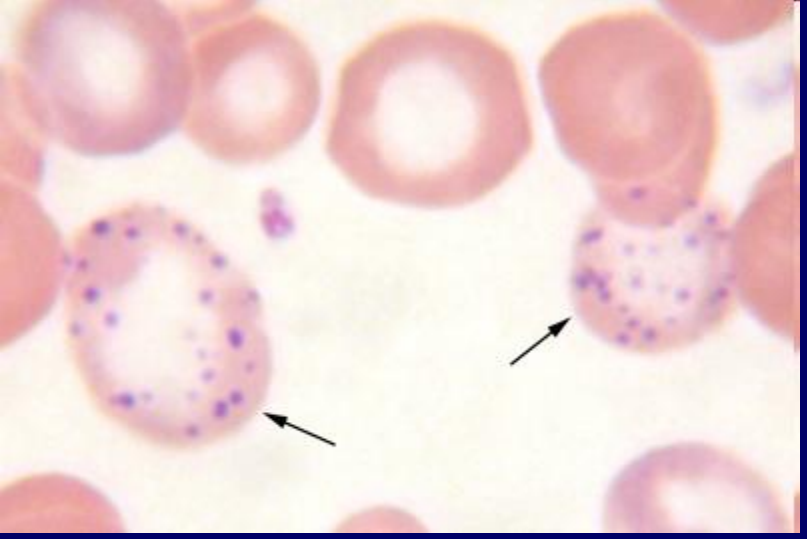
- *High affinity for sulfhydryl groups*
  - inhibition of heme biosynthesis with hypochromic anemia and basophillic stippling of erythrocytes

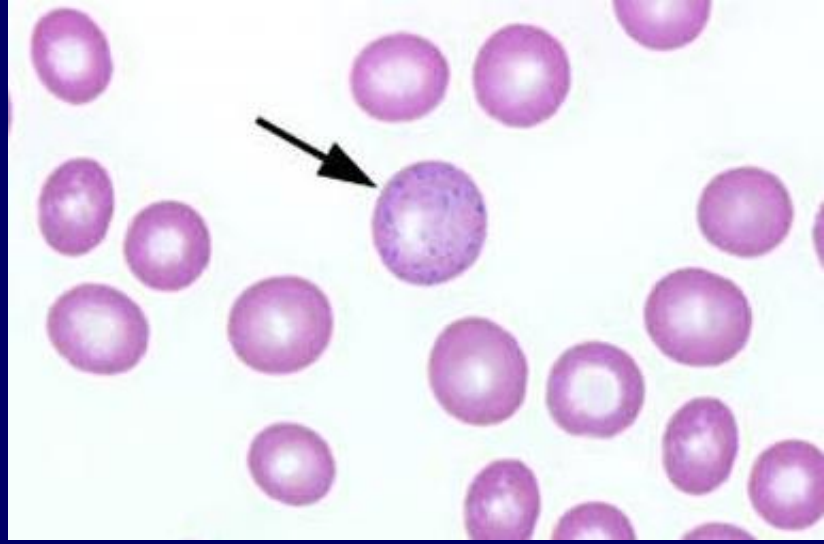


- *Competition with calcium ions*
  - As a divalent cation, lead competes with calcium and is stored in bone.
  - It also interferes with nerve transmission and brain development.
- *Inhibition of membrane-associated enzymes*
  - Lead inhibits 5'-nucleotidase activity and sodium-potassium ion pumps, leading to decreased survival of red blood cells (hemolysis), renal damage, and hypertension.

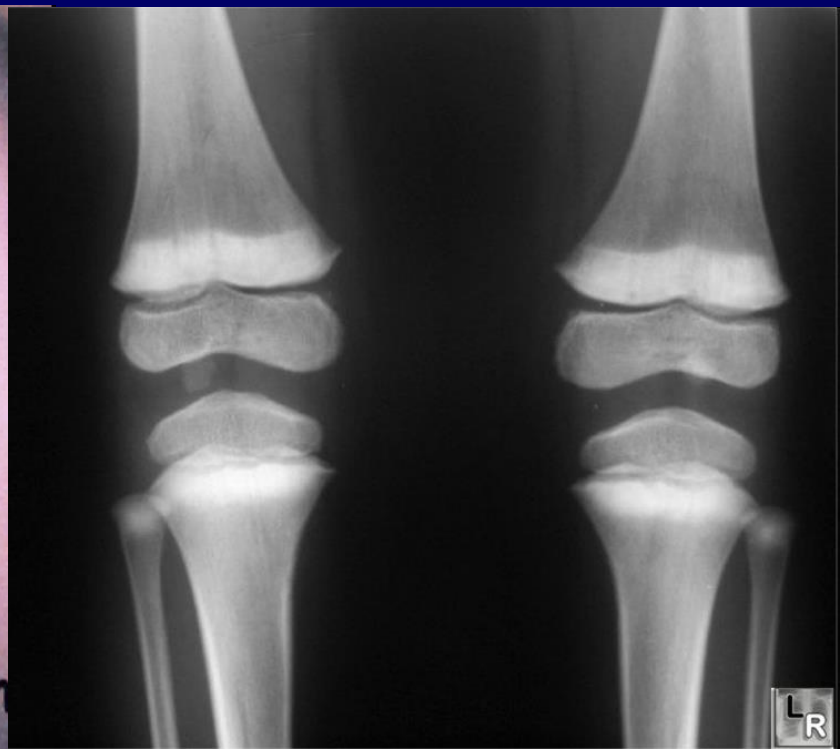
Consequences of lead exposure

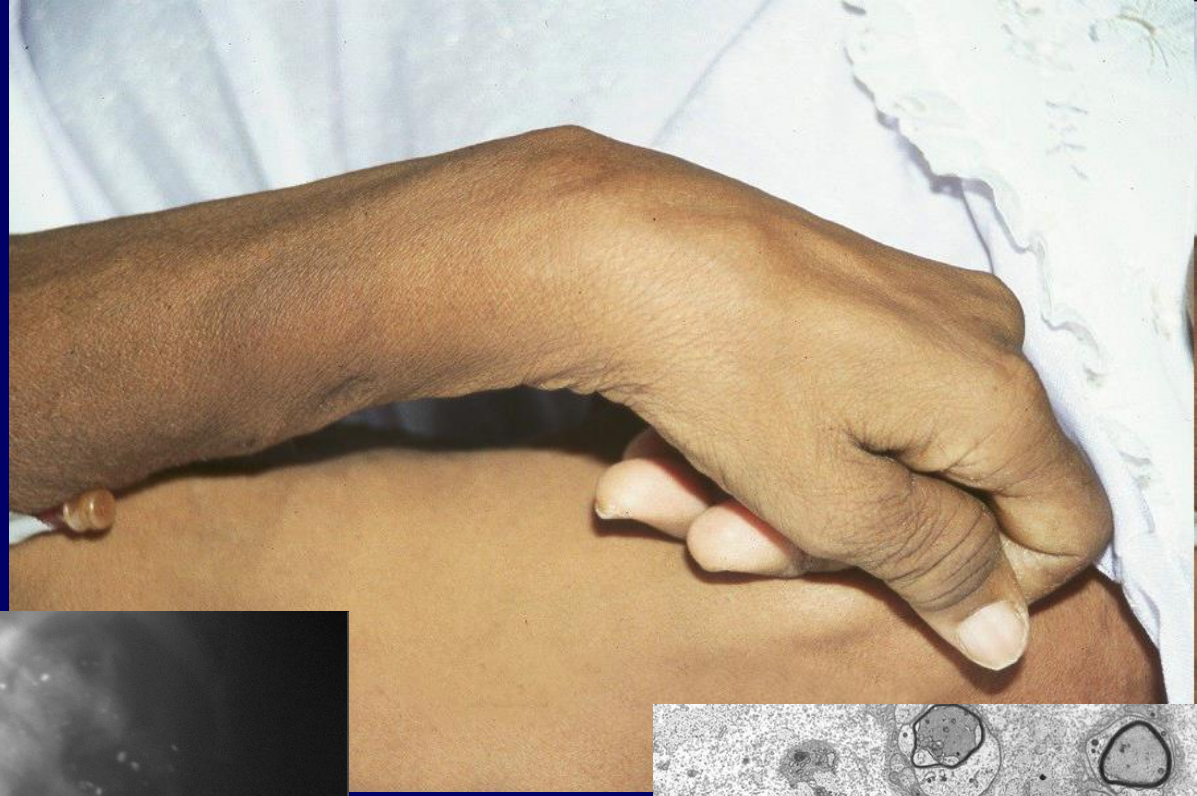


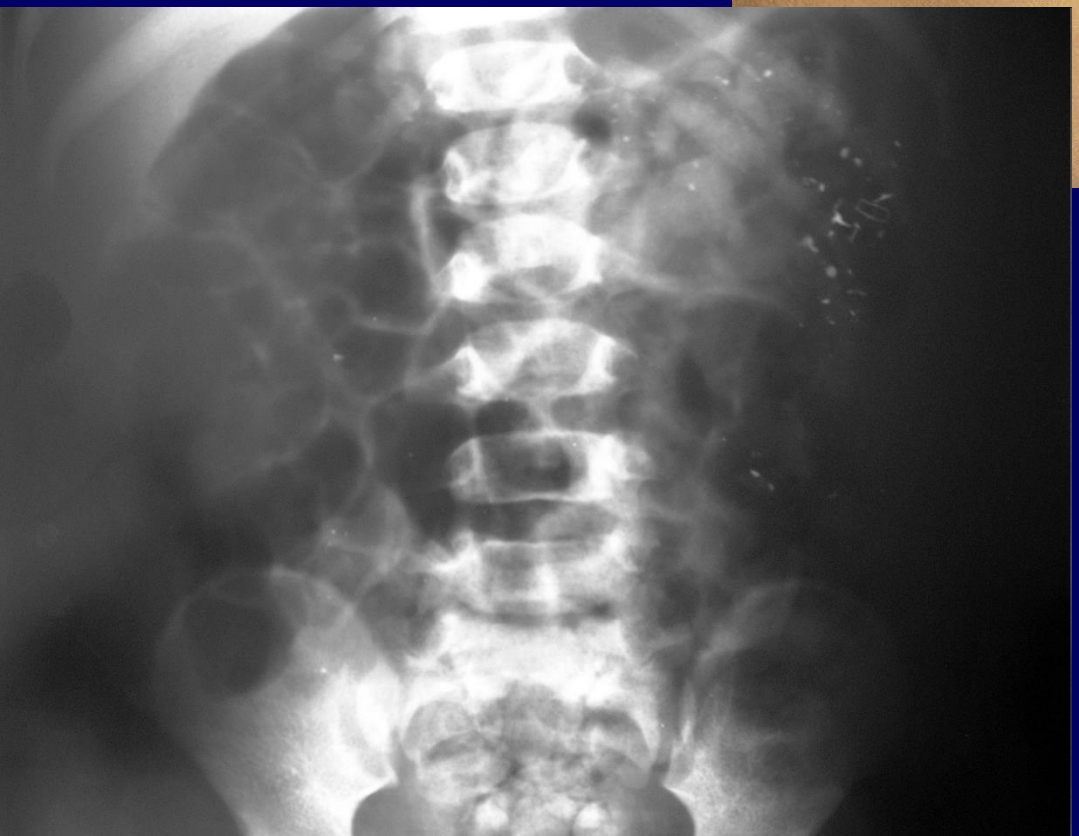


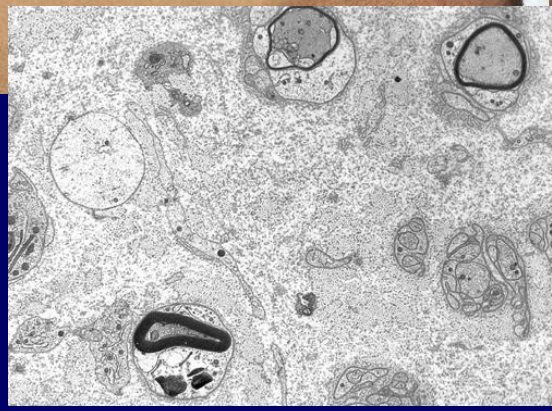



Gingival lead line in adult with lead poisonin



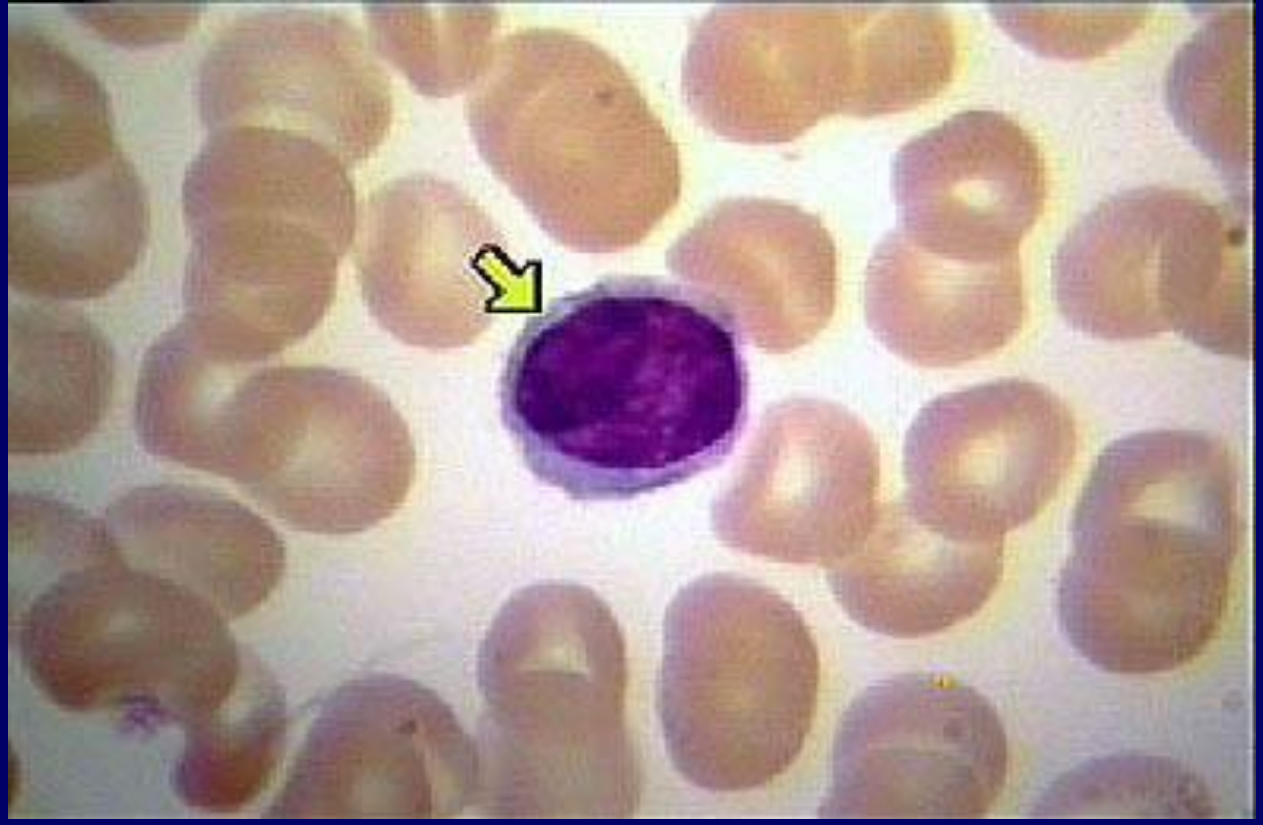






"RADIATION"

- T ½
- (m)Curie vs. (M)Becqerel
- IONIZING vs. NON-IONIZING
- PARTICULATE vs. NON-PARTICULATE (Photons)
- ENERGY: Kev, Mev (~Wavelength)
- Linear Energy Transfer (LET), Relative Biologic Effect (RBE)
- LD50@60d



This is the single most

RADIOSENSITIVE CELL

In your body

Radiation Dosimetry

- *Roentgen:* unit of charge produced by x-rays or gamma rays that ionize a specific volume of air. Physics unit.
- *RAD (radiation absorbed dose):* the dose of radiation that will produce absorption of 100 ergs of energy per gram of tissue; 1 gm of tissue exposed to 1 roentgen of gamma rays is equal to 93 ergs
- *Gray* (Gy): the dose of radiation that will produce absorption of 1 joule of energy per kilogram of tissue; 1 Gy corresponds to 100 rad (SI unit for absorbed dose)
- *REM (radiation equivalent man):* the dose of radiation that causes a biologic effect equivalent to 1 rad of x-rays or gamma rays
- *Sievert* (Sv): the dose of radiation that causes a biologic effect equivalent to 1 Gy of x-rays or gamma rays; 1 Sv corresponds to 100 rem (SI unit)

Acute Effects of Ionizing Radiation

- Free radical generation
  - Ionizing radiation + $H_20 \rightarrow H_30^+ + OH\cdot$
- DNA Damage
  - double-stranded DNA breaks needed to kill cell (mammalian cells can repair single stranded breaks)
  - cross-linking of DNA strands, cleavage of sugar-phosphate bonds
- Tumor-suppressor gene *p53* activation
  - cell cycle arrest in presence of damaged DNA
  - repair of DNA damage or apoptosis

Acute Whole Body Radiation

- LD50 @ 6 wks 2.5 to 4.0 Gy (250 to 400 rad)
- Hematopoietic
  - 200–600 REM
  - Maximum neutrophil and platelet depression in 2 wk
- Gastrointestinal
  - 600–1000 REM
  - Nausea, vomiting, diarrhea
  - Hemorrhage and infection in 1–3 wk
- Central nervous system
  - >1000 REM
  - Intractable nausea and vomiting
  - Confusion, somnolence, convulsions
  - death in 14–36 hr

Therapeutic Radiation

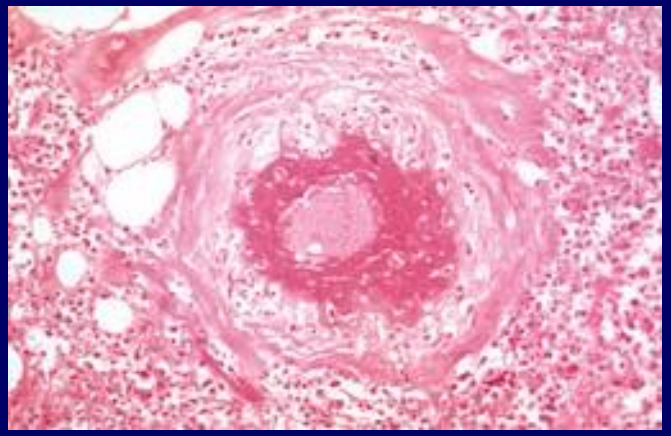
- External radiation is delivered to malignant neoplasms at fractionated doses up to 40 to 70 Gy (4000 to 7000 rad), with shielding of adjacent normal tissues
- Therapeutic radiation alone seems to add little risk of AML but can increase the risk in people exposed to alkylating agents
- Fatigue, nausea and vomiting frequent
- Bone marrow suppression may occur especially with chest or abdominal radiation

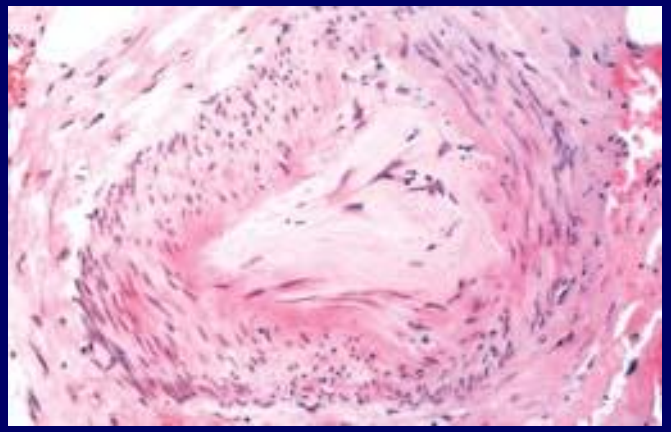
Delayed Radiation Injury

- Carcinogenesis (atom bomb survivors)
  - myeloid leukemias peak 5 to 7 years after exposure
  - breast and thyroid cancers may show greater latency
  - no germline mutations noted in progeny of survivors
- Vascular effects
  - endothelial necrosis followed by intimal and medial fibrosis
  - capillaries may become thrombosed and obliterated or ectatic
- Parenchymal atrophy and fibrosis

Radiation effects on TISSUE

- ACUTE (vasculitis, possibly “fibrinoid” necrosis)
- CHRONIC (fibrosis)





Silicosis

- inhalation of crystalline silica
- crystalline forms (quartz, crystobalite, tridymite)
- most prevalent chronic occupational disease in the world
- very heavy exposure - acute silicosis (generalized accumulation of lipoproteinaceous material within alveoli)
- decades of exposure - coal mining, stone cutting, foundry work, ceramics, sandblasting
- in high risk professions - after 30Y - 10-15% are afflicted
- complicated by TBC, Caplan's syndrome
- pulmonary hypertension, cor pulmonale chronicum
- not increased risk of malignancy (x asbestosis!)

Injury by chemical agents

Injury by chemical agents

- endless list
- inhalation, ingestion, injection, skin absorption
- therapeutic agents, nontherapeutic agents
- accident or intention
- dose
- requirement for metabolic conversion (directly toxic vs. converted compounds)
- site of absorption, accumulation or excretion
- individual variation (tolerance, enzymatic defects)
- capacity to induce immune reaction (penicillin)

Injury by therapeutic agents

- adverse drug reactions - extremely common in practice of medicine
- most frequently antibiotics, antineoplastic agents, immunosuppressive drugs
- adverse reaction - predictable (dose-dependent) - digitalis, streptomycin, cytostatics, sedatives
- - unpredictable - idiosyncrasy - massive necrosis of the liver after paracetamol

Examples

- agranulocytosis, pancytopenia (chloramphenicol, quinine, antituberculotics)
- urticaria, expholiative dermatitis (ATB, barbiturates)
- acute tubular necrosis, necrosis of papillae, renal vasculitis (phenacetine, sulphonamides, analgetics)
- lung edema, fibrosis (bleomycine, busulphan)
- liver steatosis, cholestasis, necrosis of hepatocytes (tetracycline, estrogens, halothan, chlorpromazine)
- cardiomyopathy (anthracyclines - adriamycin)

Therapeutic Drugs (Medications)

- Oral Contraceptives (BCPs)
- Hormone Replacement Therapy (HRT)
- Acetaminophen
- Aspirin

Oral Contraceptives (BCPs)

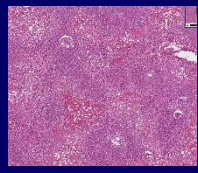
- Breast cancer and other cancers
  - no increase in breast cancer
  - decrease endometrial and ovarian cancers
  - increase in cervical cancer (?lifestyle induced)
- Thromboembolic events
  - DVT and Pulmonary Embolism increased
  - adds to other risk factors (e.g. Factor V Leiden)
- Cardiovascular disease
  - with current low estrogen pills, risk of MI and atherosclerosis not increased in non-smoking women < 45 y
  - ischemic stroke increased regardless of age or smoking
- Liver tumors
  - benign hepatic adenomas
  - older women with prolonged use
  - may rupture and cause intra-abdominal bleeding

Hormone Replacement Therapy (HRT)

- Cancer
  - in women with a uterus combined estrogen and progestin Rx necessary to reduce endometrial cancer
  - WHI showed increased risk of breast cancer in women who used HRT combined therapy for 5 years
- Thromboembolic events
  - elevated approximated twofold in HRT users, especially within the first 2 years
- Cardiovascular disease
  - WHI reported 29% increased risk of myocardial infarction, especially during the first year of combined HRT use

Acetaminophen (Tylenol)

- Does not affect cyclooxygenase so bleeding associated with aspirin does not occur
- Has analgesic and antipyretic actions but no anti-inflammatory action
- Large doses may produce hepatic necrosis
  - patients should not exceed recommended dose (4 grams/day)
  - toxic dose in adults is 15 to 25 gm
  - dose should be reduced in children with fever or dehydration



Analgetics

- aspirin (acetylsalicylic acid)
- - overdose - intoxication - respiratory alkalosis, metabolic acidosis, Reye syndrome (?)
- - chronic toxicity - erosive gastritis, ulcers
- phenacetine - kidney damage (necrotizing papillitis, chronic interstitial nonbacterial nephritis) - phenacetine kidney
- acetaminophen - very large doses - hepatotoxicity

Aspirin

- Overdose
  - respiratory alkalosis followed by metabolic acidosis that may be fatal
- Chronic aspirin toxicity (salicylism)
  - headache, dizziness, ringing in the ears (tinnitus), mental confusion, drowsiness, nausea, vomiting, and diarrhea
- Inhibits cyclooxygenases (COX 1 & 2)
- Erosive gastritis is a major cause of GI bleeding
- May be implicated in Reye syndrome (fatty liver with encephalopathy) in children < 15 years old, especially with influenza and chicken pox

Cox-1 and Cox-2 Inhibitors

- Cyclooxygenase 1 (inhib of COX-1 is BAD)
  - constitutively expressed and active in the normal platelet (thromboxane A2)
  - involved in synthesis of gastro-protective prostaglandins
- Cyclooxygenase 2 (inhib of COX-2 is GOOD)
  - induced, especially in inflamed tissue
  - in vessel wall produces prostacyclin (PGI_2)
- Aspirin and other nonselective NSAIDS inhibit both COX-1 and COX-2

Injury by nontherapeutic toxic agents

Lead

- acute poisoning - colicky abdominal pain, fatigue, headache, encephalopathic crisis
- chronic (professional) exposure - defect of Hb synthesis - anemia, neurological disorders

Tobacco and Cancer

- 70% of all lung cancers
- 30% of all cancers

Organ-Specific Carcinogens in Tobacco Smoke

Organ	Carcinogen
Lung, larynx	Polycyclic aromatic hydrocarbons
	4-(Methylnitrosoamino)-1-(3-pyridyl)-1-buta-none (NNK)
	Polonium 210
Esophagus	*N'*-Nitrosonornicotine (NNN)
Pancreas	NNK (?)
Bladder	4-Aminobiphenyl, 2-naphthylamine
Oral cavity (smoking)	Polycyclic aromatic hydrocarbons, NNK, NNN
Oral cavity (snuff)	NNK, NNN, polonium 210

Data from Szczesny LB, Holbrook JH: Cigarette smoking. In Rom WH (ed): Environmental and Occupational Medicine, 2nd ed. Boston, Little, Brown, 1992, p. 1211.

Cigarettes And The Workplace

- Similar to asbestos exposure, cigarette smoke is "synergistic" with radon decay products in causing lung cancer
- Cigarette smoke exacerbates bronchitis, asthma, and pneumoconiosis associated with exposure to silica, coal dust, grain dust, cotton dust, and welding fumes (DUH)

Alcohol (ethanol)

- worldwide problem - western countries 8-12% of population
- alcohol metabolized mainly in liver (acetaldehyde), minor part (10%) excreted in breath and urine
- acute intoxication
- depression of CNS (following transitory excitation), impairment of intellectual, motoric and vegetative functions - injuries, accidents
- severe intoxication - respiratory arrest, aspiration

Alcohol

- 15 to 20 million alcoholics in the USA
- 100,000 deaths/year due to alcohol abuse
- Economic losses of $100 to $130 billion/year
- One to two drinks/day reduces incidence of coronary artery disease*

*** What kind of person would put this kind of bullet on a powerpoint?**

A) Drinker? B) Non-Drinker? C) Alcoholic in Denial?

Effects of Blood Alcohol Levels in the Absence of Tolerance

Blood Level, mg/dL	Usual Effect
20	Decreased inhibitions, a slight feeling of intoxication
80	Decrease in complex cognitive functions and motor performance
200	Obvious slurred speech, motor incoordination, irritability, and poor judgment
300	Light coma and depressed vital signs
400	Death

Harrison Internal Med, 16th Ed

ALCOHOL IMPAIRMENT CHART

NEVER DRINK AND DRIVE

	APPROXIMATE BLOOD ALCOHOL PERCENTAGE								
Drinks	Body Weight in Pounds								
	100	120	140	160	180	200	220	240	
0	.00	.00	.00	.00	.00	.00	.00	.00	ONLY SAFE DRIVING LIMIT
1	.04	.03	.03	.02	.02	.02	.02	.02	Impairment Begins
2	.08	.06	.05	.05	.04	.04	.03	.03	
3	.11	.09	.08	.07	.06	.06	.05	.05	Driving Skills Affected — Possible Criminal Penalties
4	.15	.12	.11	.09	.08	.08	.07	.06	
5	.19	.16	.13	.12	.11	.09	.09	.08	
6	.23	.19	.16	.14	.13	.11	.10	.09	
7	.26	.22	.19	.16	.15	.13	.12	.11	Legally Intoxicated — Criminal Penalties
8	.30	.25	.21	.19	.17	.15	.14	.13	
9	.34	.28	.24	.21	.19	.17	.15	.14	
10	.38	.31	.27	.23	.21	.19	.17	.16	

MALE

Your body can get rid of one drink per hour.
Each 1½ oz. of 80 proof liquor, 12 oz. of beer or 5 oz. of table wine = 1 drink.

Source: Journal of Studies on Alcohol, Vol. 42, No.7, 1981.

Alcohol and the Liver

- Fatty Change
  - present in over 90% of binge and chronic drinkers
  - liver is enlarged but patient is asymptomatic
  - changes are reversible with cessation of drinking
  - macrosteatosis w/o inflammation or necrosis
- Alcohol hepatitis
  - only between 10 - 15% of alcoholics will develop alcoholic hepatitis
  - may have systemic symptoms and jaundice
  - hepatocellular necrosis with Mallory bodies and PMNs (central hyaline sclerosis)
  - thought to be a precursor of cirrhosis
  - probably more than HALF will go onto cirrhosis if ETOH is not stopped
- Alcoholic cirrhosis
  - shrunken nodular liver with uniform small nodules (micronodular cirrhosis)

Fatty Change Biochemistry

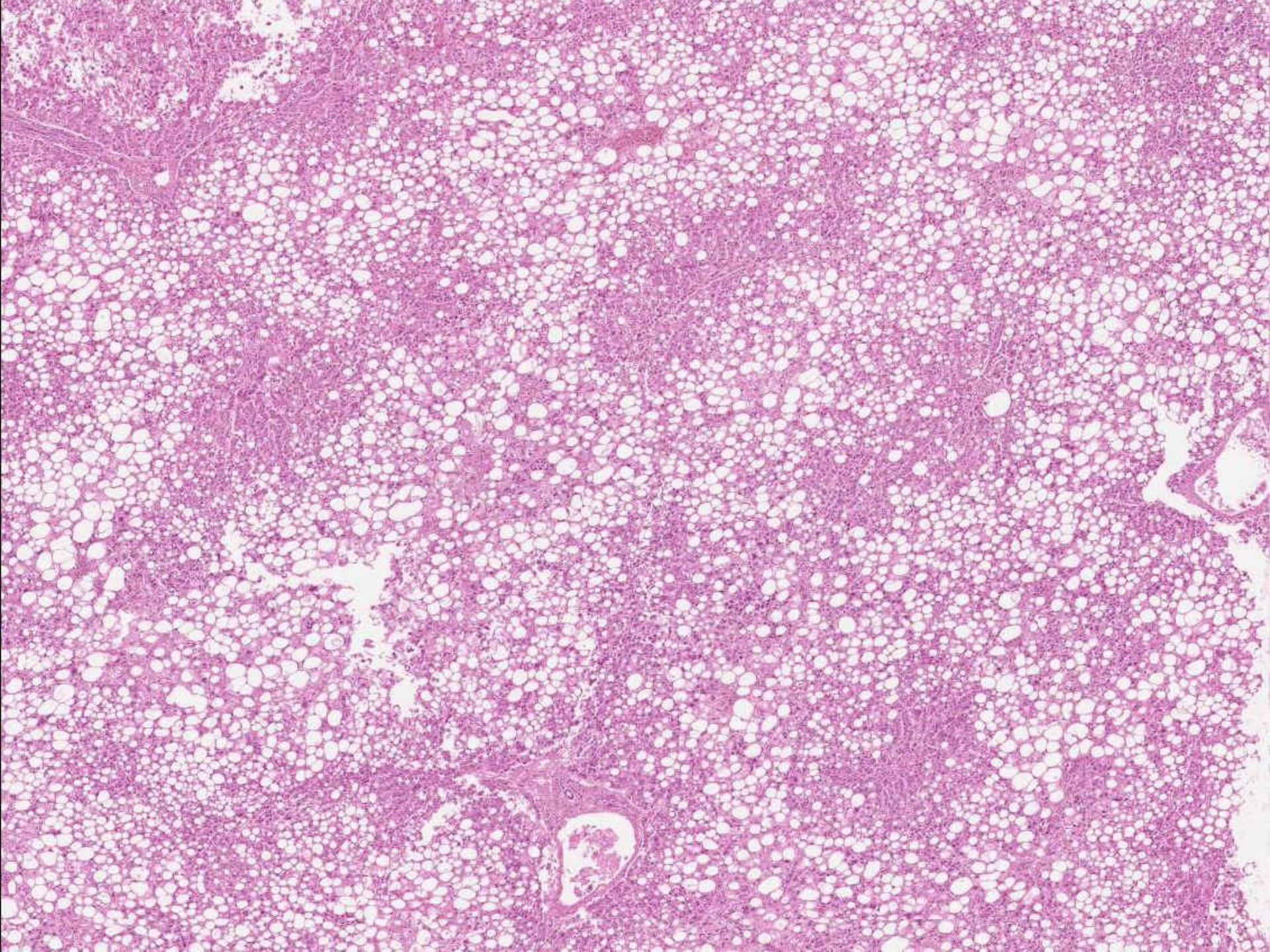
- Catabolism of fat by peripheral tissues is increased, and there is increased delivery of free fatty acids to the liver
- An excess of NADH over NAD stimulates lipid biosynthesis
- Oxidation of fatty acids by mitochondria is decreased
- Acetaldehyde forms adducts with tubulin and impairs function of microtubules, resulting in decreased transport of lipoproteins from the liver

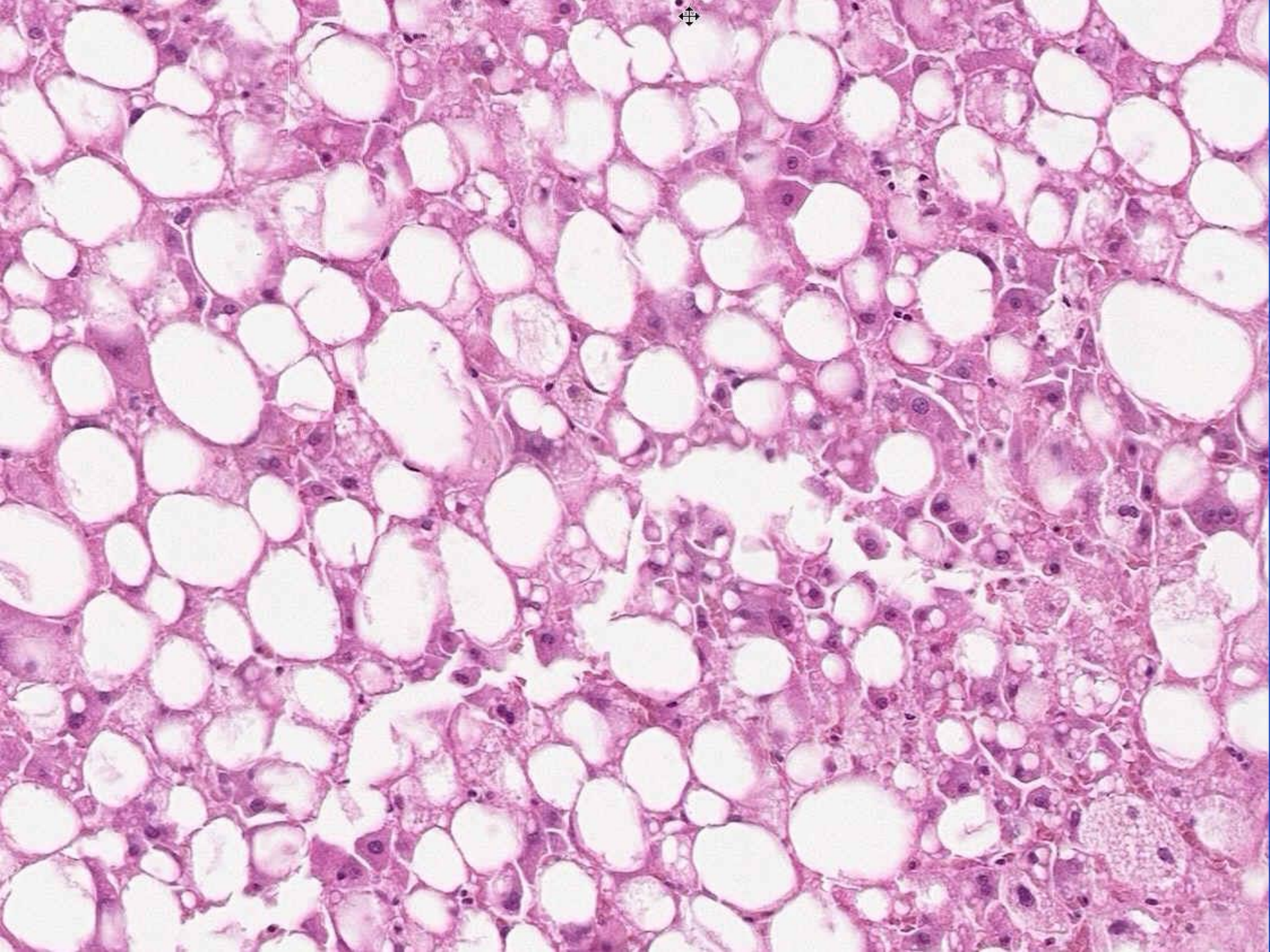
Mechanisms of Disease Caused by Ethanol Abuse

Organ System	Lesion	Mechanism
Liver	Fatty change	Toxicity
	Acute hepatitis	
	Alcoholic cirrhosis	
Nervous system	Wernicke syndrome	Thiamine deficiency
	Korsakoff syndrome	Toxicity and thiamine deficiency
	Cerebellar degeneration	Nutritional deficiency
	Peripheral neuropathy	Thiamine deficiency
Cardiovascular system	Cardiomyopathy	Toxicity
	Hypertension	Vasopressor

Mechanisms of Disease Caused by Ethanol Abuse

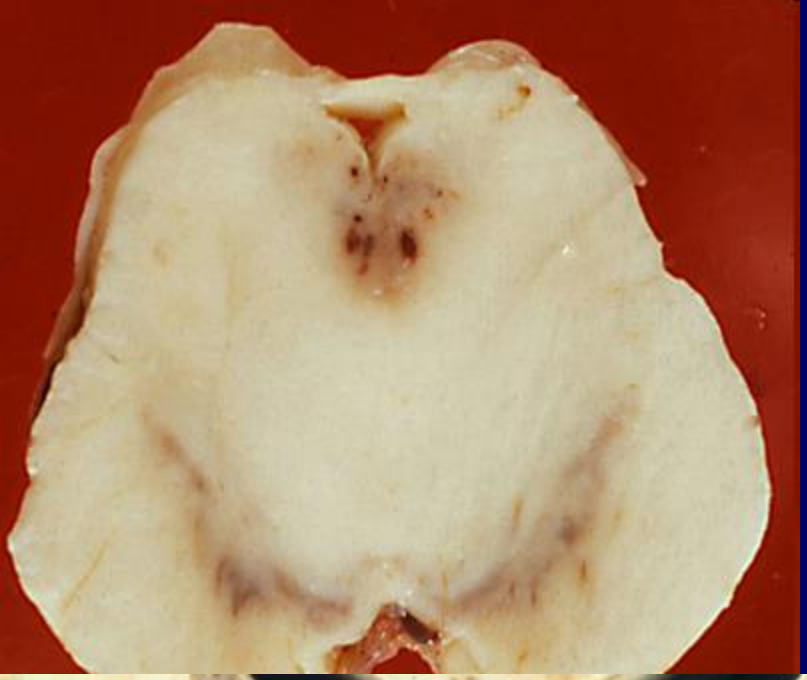
Organ System	Lesion	Mechanism
Gastrointestinal tract	Gastritis	Toxicity
	Pancreatitis	Toxicity
Skeletal muscle	Rhabdomyolysis	Toxicity
Reproductive system	Testicular atrophy	?
	Spontaneous abortion	?
Fetal alcohol syndrome	Growth retardation	Toxicity
	Mental retardation	
	Birth defects	

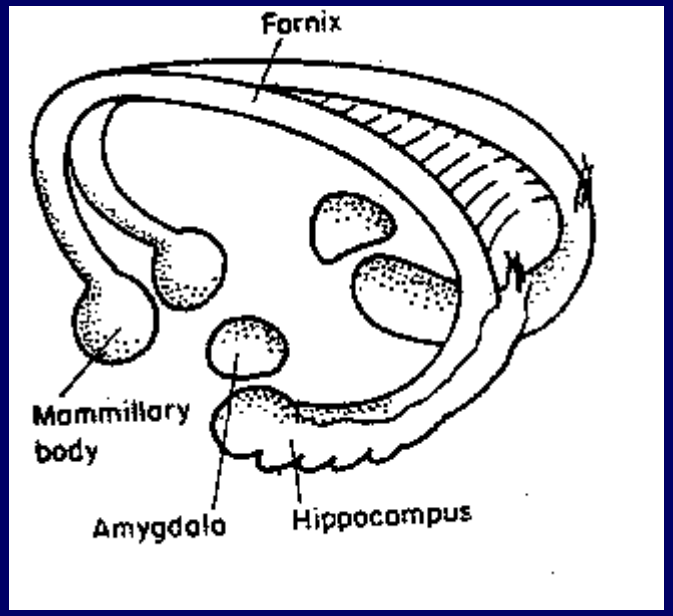


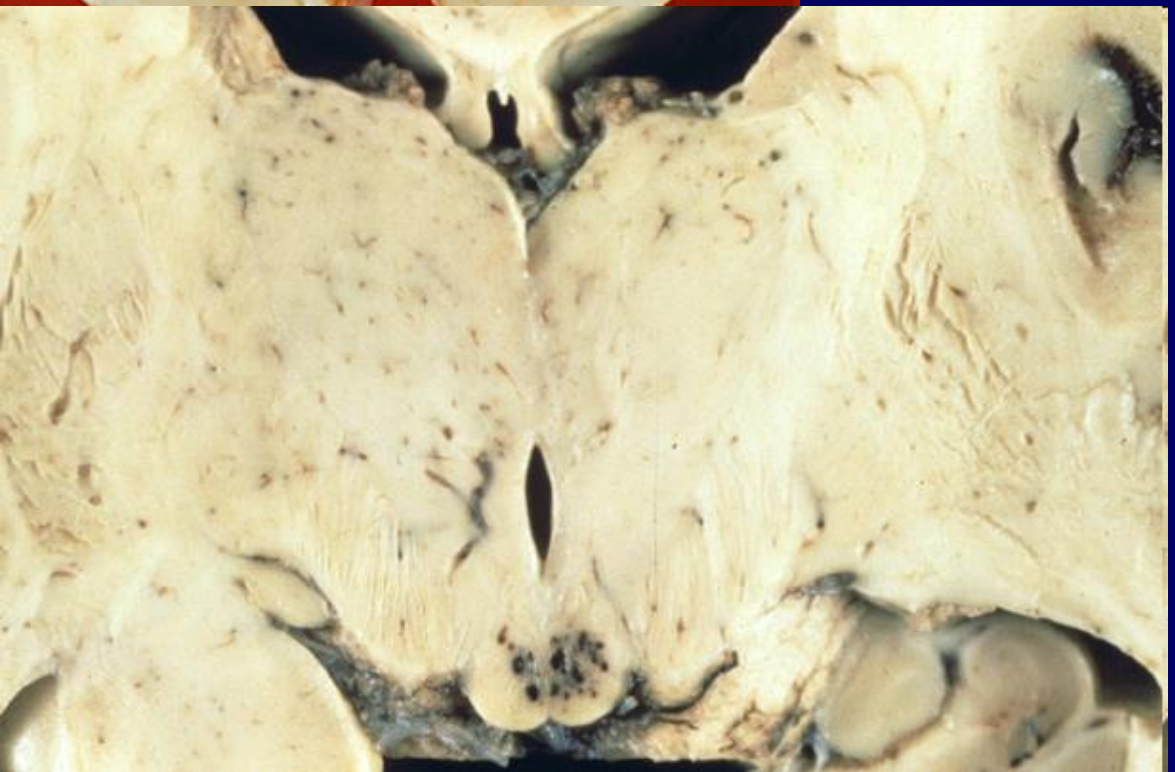


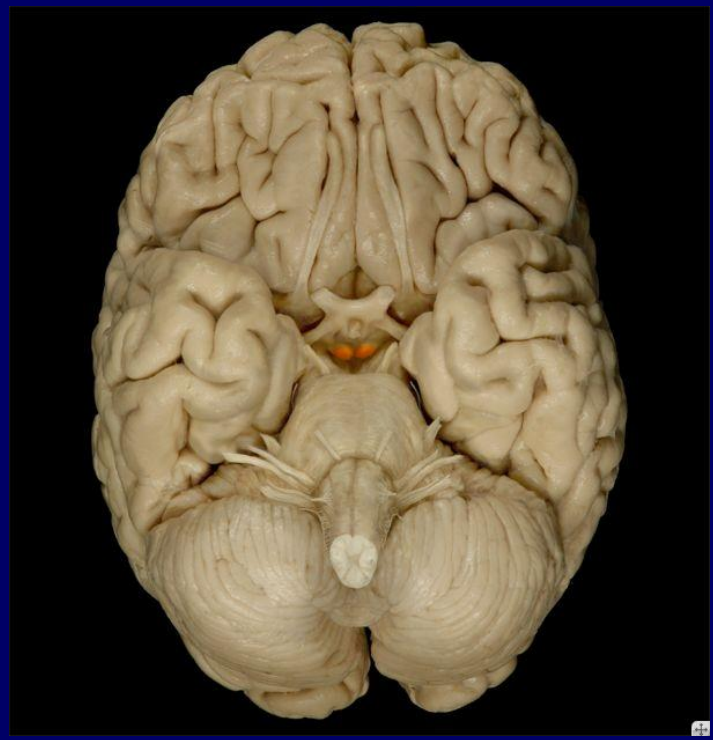
Neurologic Manifestations of Alcoholism

- Wernicke syndrome
  - confusion, ataxia, and diplopia from ophthalmoplegia
  - damage to mammillary bodies, cerebellum and periaqueductal gray matter of the midbrain
  - due to thiamine deficiency
  - may respond to prompt thiamine replacement
- Korsakov syndrome
  - memory loss and confabulation
  - results from thiamine deficiency and direct toxicity




Fornix
Mammillary body
Amygdala
Hippocompus





Chronic alcoholism

- damage of several systems (alcohol and acetaldehyde)
- secondary complications - nutritional disorders, hypovitaminosis B-complex
- toxic injury - liver (steatosis - steatohepatitis - micronodular cirrhosis)
- heart - dilated alcoholic cardiomyopathy, moderate consummation protects against coronary atherosclerosis
- CNS - Wernicke-Korsakov syndrome (hypovitaminosis B) - psychosis, memory defects
- PNS - peripheral neuropathy
- immune system - secondary immunodeficiency
- GIT - oral cavity ca, esophageal ca, esophageal varices, peptic ulcer, acute+chronic pancreatitis

Carbon monoxide (CO)

- nonirritating, colorless, tasteless, odorless gas
- product of imperfect oxidation
- affinity of CO to Hb is 200x higher, than that of O_2 - carboxyhemoglobin - systemic hypoxia
- acute intoxication - cherry red skin, liquid blood (no post-mortal coagulation)

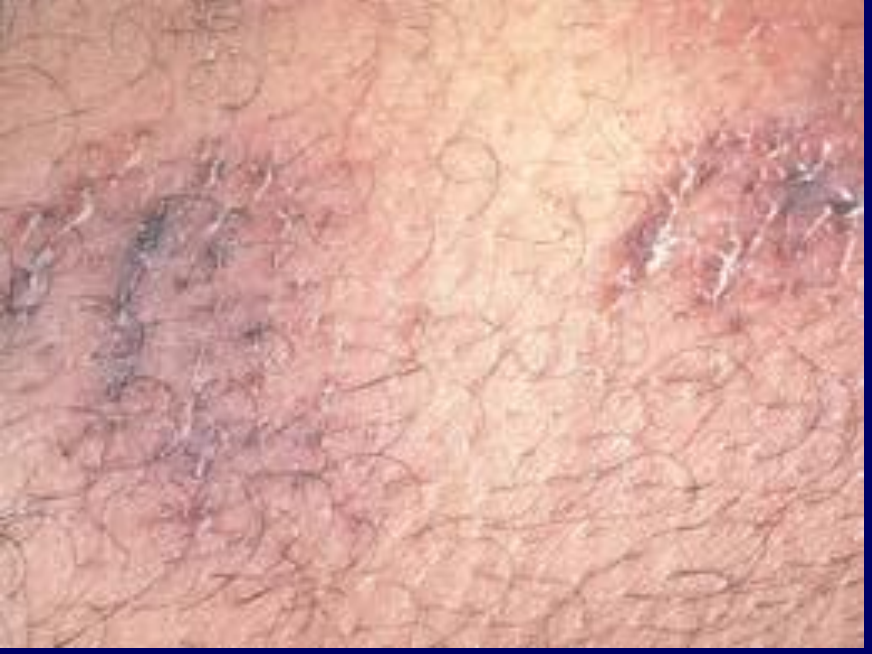
Injury by physical agents

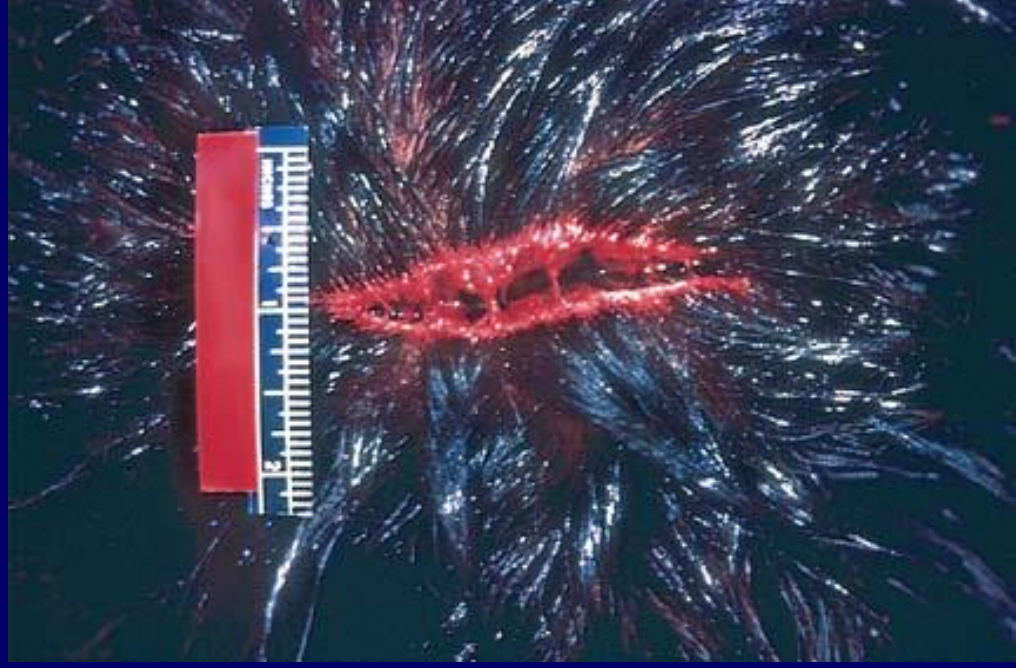
Injury by physical agents

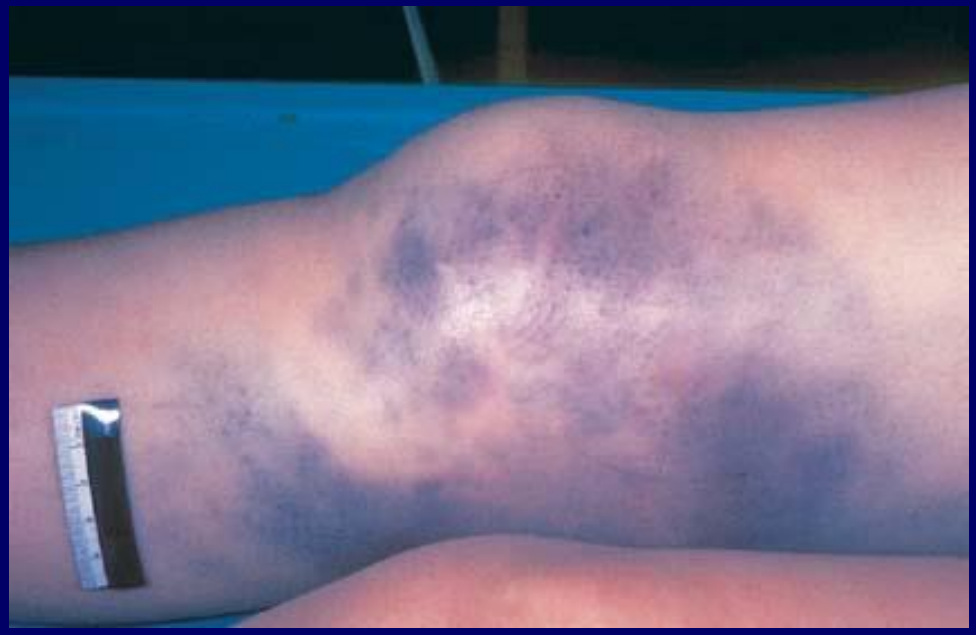
- Mechanical trauma
- car accidents - polytrauma
- abrasion - scraping or rubbing - removal of superficial layer
- contusion - blunt injury, extravasations of blood into tissues - hematoma
- laceration - disruptive stretching of tissue - jagged, irregular edges
- incised wound - by sharp instrument
- puncture wound - long narrow instrument - penetrating (in) or perforating (in+out)
- rupture - hollow organs, large vessels
- fracture - bones - surgery

Physical Injury

- Abrasion
  - basically a scrape
  - superficial epidermis is torn off by friction or force
  - regeneration without scarring usually occurs
- Laceration vs. Incision
  - a laceration is an irregular tear in the skin produced by overstretching. The wound margins are frequently hemorrhagic and traumatized
  - an incision is made by a sharp cutting object. The margins of the incision are usually relatively clean
- Contusion
  - an injury caused by a blunt force that damages small blood vessels and causes interstitial bleeding, usually without disruption of the continuity of the tissue (*cf* ecchymosis)







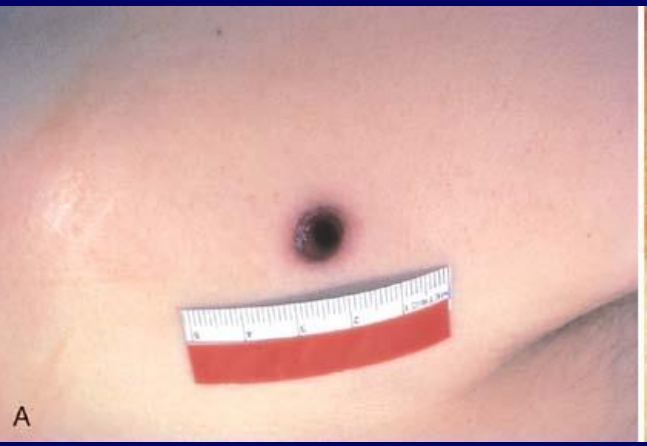
Adult Mortality Rates in the United States, Ages 25–44, in 1998

	Rate per 100,000 population		
Cause	***Hispanic***	***Black***	***White***
Unintentional injuries	33.4	40.1	31.6
Cancer	16.8	38.0	25.3
Homicide	13.1	**36.2**	**4.7**
Human immunodeficiency virus	12.1	**43.3**	**4.8**
Heart disease	10.3	43.5	18.3
Suicide	7.8	—	17.0
Total	130.2	303.7	139.4

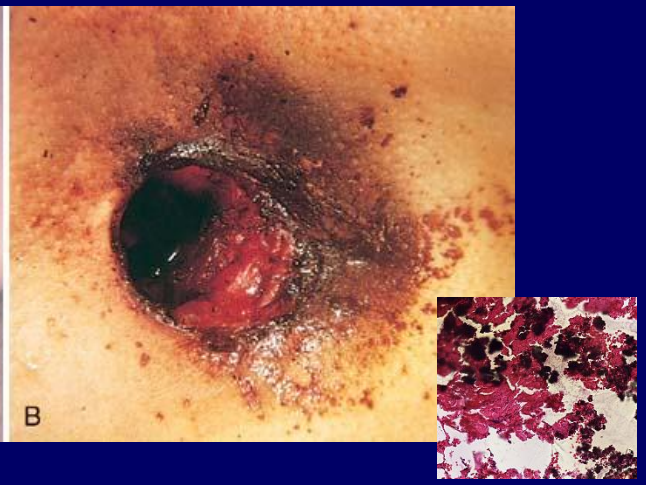
Data from CDC Fact Book, 2000/2001, Department of Health and Human Services, Centers for Disease Control and Prevention.

GUNSHOT WOUND

- Entrance Vs. Exit
- Far range Vs. Close range



NOT CLOSE RANGE



CLOSE RANGE (POWDER BURNS)

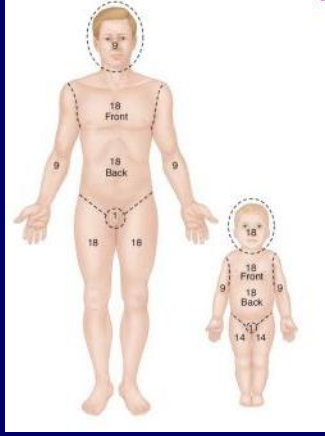
EXIT WOUNDS are generally SLOPPIER than ENTRANCE WOUNDS

Thermal injury

- Burns - frequent, prevention (children!)
- Clinical importance depends on:
- depth of the burn
- percentage of the body surface involved
- presence of internal injuries (inhalation of hot and toxic fumes)
- treatment

- full thickness burn - epidermis, dermis, loss of dermal appendages - skin grafts, pigskin
- partial thickness burn - deep parts of dermal appendages are spared - source of reepitelization
- % of body surface
- in the past - 50% - lethal
- today - 80% can survive
- complications - infection, loss of proteins and fluid (hypovolemic shock) - in patients with >20% of surface, "stress" peptic ulcers, squamous cell ca in the area of scar

BURNS



- 1st, 2nd, 3rd, 4th "Degree"
- FULL vs. PARTIAL Thickness
- Survival
  - PERCENT of body using the rule of NINES
  - DEGREE (i.e., Depth)
  - Respiratory Tract Involvement
  - AGE
  - Speed of access to Burn Unit
  - Immune System (Pseudomonas, S. aureus, Candida), infections are usually the lethal delayed cause of death.

HYPER-THERMIA

- HEAT→
  - CRAMPS: Electrolyte loss via sweat
  - EXHAUSTION: Water depletion and lack of cardiovascular compensation
  - "STROKE": Extensive peripheral vasodilatation, i.e., "shocky", very serious, T>106º, over 110º have been reported, high mortality. In this case true "SHOCK" would be a better term than "STROKE"

Hyperthermia

- heat cramps - due to loss of electrolytes (sweating)
- heat exhaustion - sudden onset, collapse, hypovolemia
- heat stroke - high temperature + high humidity - rise of core body temperature; in severe cases 50% mortality - peripheral vasodilatation, shock, necrosis of muscles, DIC

HYPO-THERMIA

- Often in setting of homelessness or alcoholism or both
  - –< 90º often fatal, assoc. w.
    - BRADYCARDIA
    - ATRIAL FIBRILLATION

Hypothermia

- local reactions
- freezing of cells - crystallinization of water within cells, high salt concentrations
- circulatory changes - vasoconstriction, increased permeability, edema, hyperviscosity of blood - ischemia (e.g. gangrene of toes)

Injury produced by ionizing radiation

- electromagnetic waves (gamma)
- high-energy particles (alpha, beta, neutrons, positrons)
- most important target = DNA (directly by radiation or via free radicals
- radiosensitivity depends on mitotic activity (1906 - Bergonie-Tribondeau)
- the effects may be latent (apparent after very long period)

- effect is dependent on dose (Gy) and type of tissue
- high sensitivity - lymphoid tissue, bone marrow, germ cells, intestinal mucosa, skin appendages, other surface epithelia
- low sensitivity - cartilage, bone, glands, lung, kidney, liver, muscle, neurons

Effect

- skin - erythema (radiodermatitis) - hyperpigmentation, depigmentation, teleangiectasia, atrophy, loss of hair, ulceration, secondary squamous cell ca
- hematopoietic system+LN - lymphopenia, decrease of size of LN and spleen, neutropenia, thrombocytopenia, anemia
- genitals - extinction of germ cells
- lungs - edema, DAD, fibrosis
- GIT - hyperemia, ulceration, fibrosis -> strictures

Total body radiation

- atomic bomb, nuclear power plant accident
- even in very low doses - devastating effect
- effect on hematopoietic system, GIT and brain - as little as 2-3Gy may be lethal!
- nausea, vomiting, fatigue - acute radiation syndrome
- 1-5 Gy - hematopoietic form - nausea, vomiting, lymphopenia, thrombocytopenia, neutropenia, later anemia
- 5-50 Gy - gastrointestinal form - diarrhea, hemorrhage, toxemia (from large bowel) - death in 8-9 days
- ≥50 Gy - cerebral form - drowsiness, listlessness, convulsions, coma (death within hours, max. 3 days)

Late effects

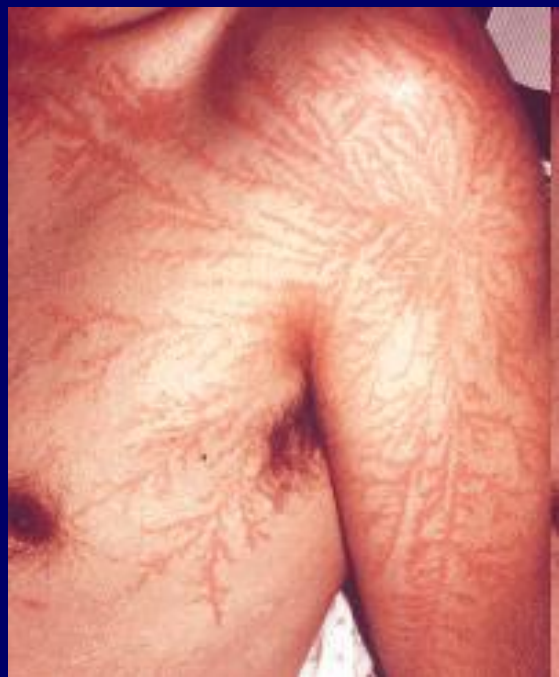
- acute leukaemia - in 5-20Y
- other tumors - thyroid ca, breast ca, ML, lung ca
- genetic defects - descendants

Radiation in treatment of tumors

- much higher doses (up to 40 Gy)
- patient is carefully shielded - selective irradiation of tumor mass
- local reactions - mainly skin
- early - erythema, dry desquamation, wet desquamation, acute postradiation ulcer
- chronic - postradiation poikiloderma - atrophy, teleangiectasia, hyperpigmentation
- late - neoplasms - squamous cell ca

LIGHTNING/ELECTRICAL

- ELECTRIC DISTURBANCES
  - NEURAL (because nerve is such an EXCELLENT conductor of electricity)
  - EKG (like reverse cardioversion)
- THERMAL INJURY, directly proportional to a particular tissue's RESISTANCE to electrical flow
- "LIGHTNING" MARKS



ATMOSPHERIC PRESSURE

- Altitude Illness
- Blast Injuries
- Decompression Injuries

ALTITUDE ILLNESS

- Caused by LOW Oxygen Tension
  - HIGH ALTITUDES (>4000 m [12,000 feet])
  - OBTUNDATION→
  - INCREASED CAPILLARY PERMEABILITY→
  - ACUTE PULMONARY EDEMA (HAPE)

Q: What is the name of the base camp at Mt. Everest

A: Pulmonary Edema

BLAST INJURIES

- RELATED TO RAPID ATMOSPHERIC PRESSURE CHANGES
  - LUNGS
  - VISCERA, especially GAS filled viscera
- Rupture, Hemorrhage, etc.
- IMMERSION BLAST also possible, causing more of a total body compression syndrome

DECOMPRESSION

- Related to GAS SOLUBILITY in divers ascending rapidly, especially the more NON-SOLUBLE gasses, like NITROGEN, and, to a lesser extent, XENON
- AIR EMBOLISM is the common pathology
  - ACUTE:
    - "BENDS" (peri-articular), acute
    - "CHOKES" (lungs), acute
    - "STAGGERS" (inner ear), acute
  - CHRONIC:
    - ASEPTIC NECROSIS: humeri, femurs