Alcohol Metabolism

A 25-year-old male is brought to the emergency room suffering from protracted vomiting, nausea, and severe hypotension. He claims that he only had two glasses of beer when he became lightheaded and started to vomit. He further admits that he has been taking metronidazole for treatment of amebiasis that he contracted a month ago. Which of the following would be the most likely cause of the symptoms in this patient?

A. Re-infection with resistant Entamoeba histolytica
B. Ethanol intoxication
C. Antabuse effect of metronidazole therapy
D. Common side-effect of metronidazole therapy
E. Inappropriate dosage of metronidazole

- Alcohol is metabolized in the liver by a 2-step oxidation to acetaldehyde and acetate, using alcohol and acetaldehyde dehydrogenase enzymes respectively.
- Both steps produce NADH.
- Increased cytosolic NADH facilitates reduction of pyruvate to lactate, and oxaloacetate to malate.
- Oxaloacetate and pyruvate are gluconeogenic substrates. Hence, ethanol decreases gluconeogenic substrates, and glucose synthesis, causing hypoglycemia.

- Hypoglycemic effect of alcohol is more exaggerated in starved individuals who have depleted their hepatic glycogen levels.
- Disulfiram (Antabuse drug) inhibits acetaldehyde dehydrogenase.

- Acetaldehyde accumulation causes nausea, hypotension, tachycardia, flushing, hyperventilation, headache, and vomiting.

Tips from Coach’s Corner

- Alcohol is the most commonly abused drug in this country but it is less commonly blamed for the pathological changes that it causes.
- It is very commonly presented on the exam.
- Your check list of related concepts for alcohol for the exam should minimally include:
  - Alcohol dehydrogenase
  - Mechanism of ketone and lactate formation with alcohol
  - Mechanism of toxicity with ethylene glycol and methanol, and role of ethanol as an antidote.
  - Antabuse effect and Disulfiram reaction
  - Mechanism of hypoglycemia in alcoholism
  - CNS and Non-CNS manifestations of alcoholism.
  - Vitamin deficiencies related to alcoholism: Folate, B1, and B3.
  - Wernicke’s encephalopathy and Korsakoff’s syndrome.
  - Delirium tremens
  - Fetal alcohol syndrome

- This syndrome is a secondary consequence of chronic alcoholism.
- It is manifested by confusion, ataxia and ophthalmoplegia.
- It is due to thiamine (B1) deficiency as a result of alcoholism.
- What is this syndrome? ___________________________
### Tested Bugs, Their DOC and Mechanism of Action

<table>
<thead>
<tr>
<th>Bugs</th>
<th>DOC and Mechanism</th>
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<tbody>
<tr>
<td>H. Influenza</td>
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<tr>
<td>Bacillus anthracis</td>
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<tr>
<td>Candida</td>
<td>Fluconazole ( \text{Inhibits fungal ergosterol} )</td>
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<tr>
<td>Proteus</td>
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<tr>
<td>Salmonella</td>
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<tr>
<td>Rickettsia</td>
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<td>Staphylococcus aureus</td>
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<td>Streptococcus pyogenes</td>
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<td>Vibrio cholerae</td>
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<td>Legionella</td>
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<td>Gardnerella</td>
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<td>Chlamydia trachomatis</td>
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<td>Toxplasmosis</td>
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<td>MRSA</td>
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<td>Klebsiella</td>
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<td>TB</td>
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</tbody>
</table>

### 5 Must-Know Viral Bugs and Their DOC

<table>
<thead>
<tr>
<th>Virus</th>
<th>DOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herpes simplex</td>
<td></td>
</tr>
<tr>
<td>Influenza A &amp; Rubella</td>
<td></td>
</tr>
<tr>
<td>Respiratory Syncytial Virus</td>
<td></td>
</tr>
<tr>
<td>HIV</td>
<td></td>
</tr>
<tr>
<td>CMV (Retinitis)</td>
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</tr>
</tbody>
</table>

### Tips from Coach’s Corner

You must know the unique members of each class of related entities such drugs or microorganism.

#### Non-Anticancer Drugs that Cause Agranulocytosis

- C
- C
- C
- P
- S

#### Unique Anticancer Drugs Without Agranulocytosis

- V
- B

A patient is presented with pain over his shoulder that extends down his arm and radiates to the back. To confirm scalenus anticus syndrome, attending physicians asks the patient to get seated with his head extended and turned to the side of the pain. Then he asked the patient to take in a deep inspiration. At the peak of inspiration the physician observed a total loss of radial pulse on the affected side. Assuming that the patient is really presented with the scalenus anticus syndrome which of the following options is demonstrated by the maneuver?

- A. Proximal nerve root compression
- B. Brachial plexus compression
- C. Subclavian artery compression
- D. Subclavian venous compression
- E. Elevation of the first rib produced by scalene spasm

### Top 12 Tested Syndromes

- Brown-Sequard Syndrome
- Budd-Chiari Syndrome
- Carcinoid Syndrome
- DiGeorge’s Syndrome
- Down’s Syndrome
- Ehlers-Danlos Syndrome
- Reiter’s Syndrome
- Fragile X Syndrome
- Lesch-Nyhan Syndrome
- Sjogren’s Syndrome
- Wernicke-Korsakoff’s Syndrome
- Zollinger-Ellison Syndrome
DiGeorge’s Syndrome

- AKA. Thymic hypoplasia
- Defect of 3rd and 4th pharyngeal pouches
- Lack of thymus and parathyroid development
- Absence of cell-mediated immunity (T-deficiency)
- Hypocalcemia (tetany) and hyperphosphatemia
- Recurrent viral and fungal infections
- Cardiovascular anomaly of great vessels
- Facial abnormalities (e.g. Fish-shaped mouth)

CATCH 22

Cardiac anomalies
Abnormal Facies
Thymic aplasia
Cleft Palate
Hypocalcemia/ Hypoparathyroid
22q11 gene deletion

 pais of DiGeorge’s for "2" & "22"

2 pouches—3rd & 4th
2 organs—thymus plus parathyroid
22nd chromosome!

Immune Deficiency Syndromes

DiGeorge’s Syndrome: Failure of development of 3rd and 4th pouches, thymic hypoplasia. Hypoparathyroidism.

Selective IgA Deficiency: #1 Primary selective deficiency (1/500 rate). Normal IgG and IgM. Giardiasis.

AIDS (HIV, Acquired Immunodeficiency Syndrome): Loss of helper Ts. p24, gp41 and gp120. Western Blot and ELISA.

X-linked (Bruton’s) Agammaglobulinemia: No B-Cells. All immunoglobulins are decreased. Onset at 6-9 months.

Wiskott-Aldrich: X-linked, low IgM and thrombocytopenia


Chronic Granulomatous Dz: Lack of NADPH oxidase in lysosomes of macrophages and Neutrophils. Frequent infections with catalase-positive bugs.


Hereditary Angioedema: C1 esterase deficiency. Uncontrolled complement activation. Pharyngeal and laryngeal edema.

Brown-Sequard Syndrome

- Lateral hemisection of spinal cord
- Ipsilateral paralysis and spasticity (pyramidal tract)
- Ipsilateral tactile and motor loss (dorsal column)
- Contralateral pain and temperature loss caudal to the lesion (spinothalamic tract)

Why does hemisection of the cord at the level T2 produce contralateral loss of pain and temperature sensation at level T4 or below, and not at the level of T2 and below?
A young adult with a childhood history of rheumatic fever develops an infection of the mitral valve shortly after she had an operation to remove one of her molar teeth. The etiologic agent would most probably be

A. Strep pyogenes
B. Strep mutans
C. Strep bovis
D. Strep salivarius
E. Staph aureus

✔ Tips from Coach’s Corner
The top 6 tested gram-positive cocci bugs of the exam in the order that they were remembered:

1. Streptococcus pyogenes
2. Staphylococcus aureus
3. Streptococcus pneumoniae
4. Streptococcus agalactiae
5. Streptococcus viridans
6. Staphylococcus epidermidis

Molar Mutilation Made Mom Mute!
Molar Mutilation for Strep Mutans!

What high-yield bug is gram-positive and catalase-positive like staphylococcus and is transmitted by unpasteurized milk, hotdog, and luncheon or deli meats?

Hint 1: It has lately become a major health hazard in the USA
Hint 2: People at risk are pregnant women, newborns, immunocompromised patients and the elderly

✔ 9 Must Know Complications of Infections*

1. Chlamydia
2. Influenza in children treated with aspirin
3. Varicella in children treated with aspirin
4. Helicobacter pylori
5. Campylobacter
6. Strep pyogenes (group A)
7. Meningococcus
8. EHEC (Entero-hemorrhagic E Coli)
9. Lyme Disease Bell’s Palsy

* There is a strong association between above infections and conditions cited in the right-hand column.

Soft Pains
For Staph aureus Diseases
Skin Infections Pneumonia
Osteomyelitis Acute Endocarditis
Food Poisoning Infective arthritis
Toxic Shock Syndrome Necrotizing Fasciitis
Sepsis

BAG
B(Beta): Best Lysis
A(Alpha): Almost Lyses
G(Gamma): Garbage--no hemolysis

Mixed Excerpts of Various Northwestern Medical Review Publications, 2005
Listeria is Cattle-ase positive!

- I start as multiple pruritic and erythematous lesions mainly on the face.
- I evolve into oozing yellow crusts.
- I am either caused by Staph aureus or Strep pyogenes

I am ____________________________!

- Streptococcus pyogenes is bacitracin sensitive
- Streptococcus pneumoniae is optochin sensitive

Importance of Identifying Impetigo Caused by Streptococcus Pyogenes
- Impetigo contagiosa is caused by S. pyogenes infection
- There is a high risk of post-streptococcal glomerulonephritis

What two antibiotics are used in the treatment of impetigo? __________________ & __________________

Three antibacterials that are used to treat Staph aureus infections are:

P____________;
N____________ and
V____________

✔ Two Must-Know Facts About Strep Pyogenes

1. What is the basis of immunity to Group A Strep?

2. What is the strep component involved in scarlet fever rash?

This bug is a normal skin flora.
The three most prone populations to this bug are: immunocompromised individuals, hospital patients who get a Foley urine catheter or IV line, and patients with prosthetic devises such as valves or joints.
In the prone patients the bug may cause bacteremia.
It is catalase-positive but coagulase-negative.
It is best treated with Vancomycin.
What is this bug?

Which of the following findings of the cerebrospinal fluid is least likely related to the bacterial infections of the CNS?

A. Increased white blood cells
B. Increased neutrophils
C. Elevated proteins
D. Elevated glucose
E. Increased cerebrospinal fluid pressure

✔ Findings in Meningitis

<table>
<thead>
<tr>
<th>Bacterial</th>
<th>Viral</th>
<th>TB &amp; Fungal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocytosis</td>
<td>Lymphocytosis</td>
<td>Lymphocytosis</td>
</tr>
<tr>
<td>↓ Glucose</td>
<td>Normal Glucose</td>
<td>↓ Glucose</td>
</tr>
<tr>
<td>↑ Protein</td>
<td>Slight ↑ Protein</td>
<td>↑ Protein</td>
</tr>
<tr>
<td>Strep agalactiae Group B; E coli (K1); H. Influenza; Neisseria meningitidis Strep pneumoniae</td>
<td>Mumps Coxsackie virus Epstein Barr HSV2 Echo virus</td>
<td>TB Cryptococcus neoformans</td>
</tr>
</tbody>
</table>

CSF Normal Composition: Glucose, 50-75 mg/dL; Protein, 15-50 mg/dL; CSF Pressure, 80 - 180 mmHg.
A 7-year-old boy is admitted to the hospital directly from the school with the complaint of nausea, and severe headache. He has fever, and nuchal rigidity. To confirm meningitis, attending physician would like to analyze the CSF contents of the patient.

The most desired location for a spinal tap would be between:

A. L1 and L2  
B. L1 and L3  
C. L1 and L5  
D. L2 and S1  
E. L2 and S2

Lab results of the CSF confirm presence of numerous polymorphonuclear leukocytes, decreased glucose level and increased protein levels. Assuming no history of immunization, which of the following would be the most likely cause of the patient’s nuchal rigidity?

A. Escherichia coli (K1)  
B. Haemophilus influenzae  
C. S. Pneumoniae  
D. Mumps virus  
E. Herpes simplex type 2

Cisterns; the expanded subarachnoidal space, is between L2 and S2. Crest of iliac is the landmark to identify the center of the lumbar cistern.

TAP Two!  
Spinal  
Tap between the two--L2 and S2!

Explaining Hot Neck Stiffness! For big (key) bugs of each non-vaccinated bracket of patients.

Explaining (E. coli)  
Hot (Haemphilus)  
Neck (Neisseria)  
Stiffness! (Strep pneumonia)

What three big bacterial causes of meningitis are treatable by Chloramphenicol?  
H_____________________________  
N_____________________________  
S_____________________________

Is Chloramphenicol the primary DOC for them?  
____________________________________

Under what conditions Chloramphenicol is indicated for the above three causes of meningitis?  
____________________________________

What would be the prophylactic drug of choice for people who are exposed to contagious meningitic patients or the choice drugs when you deal with unidentified causes of meningitis?

Hint 1: RNA polymerase  
Hint 2: Acts on both intra and extracellular bugs  
Hint 3: Red excreta  
Hint 4: Effective against H flu, Neisseria and TB  
____________________________________

Do not Mistake the site of epidural anesthesia with spinal tap.

Below S2!  
Site of epidural (saddle block) anesthesia  
2S2addle!
**ABCD of Hepatitis for the Exam**

Serology of hepatitis B and to some extent A is very high-yield. You must master the following facts on Hepatitis A, B, C and D before you step into the exam session.

---

**Course & Serology of HBV**

<table>
<thead>
<tr>
<th>Ag &amp; Ab Levels</th>
<th>Anti-HBsAg (IgG)</th>
<th>HBeAg</th>
<th>Anti-HBcAg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mo</td>
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<tr>
<td>3 mo</td>
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<td>6 mo</td>
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</tbody>
</table>

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**Window Period in Hepatitis B!**

- During “Window” Period both surface Ag and antibody are negative.
- Anti-HBcAg is the only marker of infection during “window” period and before Anti-HBsAg appears.

Thru window u C!

---

**Case 1: Hepatitis B**

**Question 1:** At point A above, a patient with jaundice and liver pain visits your office. You are suspicious of hepatitis B. You order a lab serology for HBV. Results come back to you that there is evidence of HBSAg, HBeAg and anti-HBcAg. Your diagnosis would be:

---

**Question 2:** At point B above, and after about 3 months the patient comes. He claims that he is doing better but still he does not feel as good. You order another serology panel on him. Results come back to you that there is evidence of only anti-HBcAg. Your diagnosis would be:

---

**Question 3:** At point C above, and after another 2 months, the patient comes back for a follow-up. You order a 3rd serology panel on him. Results come back that there is evidence of Anti-HbsAg, Anti-HBeAg and Anti-HBcAg. What would you tell the patient?

---

**Chronic Hepatitis B**

<table>
<thead>
<tr>
<th>Ag &amp; Ab Levels</th>
<th>Anti-HBsAg (IgG)</th>
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<tbody>
<tr>
<td>1 mo</td>
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<tr>
<td>3 mo</td>
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<tr>
<td>6 mo</td>
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<tr>
<td>12 mo</td>
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**Question 4:** You are examining the lab report on another hepatitis B patient. Results of the patient’s serology at point D, about 9 months post-exposure, are positive for HBsAg, Anti-HBcAg (IgG) and HBeAg. Your interpretation would be

---

**Question 5:** The patient (in question 4) asks you “Doc. I peeked at the report. It says I have antibody to c antigen; does it mean I am cured?” What would you answer?

---

**Question 6:** About 4 weeks after exposure, HBV multiplies in the hepatocytes and damaged cells undergo apoptosis. Result of apoptosis is fragmented debris that is called: ________________

---

**Question 7:** One of your Hepatitis B female patients asks you. “Doc, I’ve heard that this bug is transmitted to people by blood and sexual intercourse, and to infants by sliding through the infected birth canal during delivery and by crossing the placenta; is this correct?”

Your answer would be: ____________________
**Top 5 Indigenes**

GI Congenital Anomalies

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**GULF**

Predisposing Causes of Colon & Rectum Cancer

1. Granuloma inguinale
2. Ulcerative Colitis
3. Lymphogranuloma venereum
4. Familial polyposis
5. Lymphogranuloma venereum

Infections with Chlamydia trachomatis (Types L1-L3). Ulcers, lymphadenopathy (swollen inguinal nodes) and rectal strictures.

- This is a hereditary condition.
- Many small lumps (AKA. Juvenile polyps) appear in the stomach, small and large intestine.
- Patients are borne with them or get them during childhood.
- Polyps do not have risk of intestinal cancer. But pose a higher risk for pancreas, breast and ovary cancers.
- Patients have a characteristic brown skin and mucus membranes—especially on the lips and gums.
- Familial Polyposis is the dyad of this disease.
- This disease is: ______________________

**Meckel's Diverticulum**

Most common congenital anomaly of GI. Persistent vitelinite duct (yolk stalk). Antimesenteric border of the bowel.

Complications:
- Bleeding and peptic ulceration;
- intussusceptions and volvulus.

Symptoms mimic acute appendicitis.

**Fragile X Syndrome**

X-Linked Recessive

Macro-orchidism

Second most common cause of hereditary mental retardation

**Top 8 Indigenes**

Enzyme Deficiencies

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</tbody>
</table>

A 7-month male Ashkenazi Jewish child with gradual mental, motor, and visual deterioration has a pale color retina. Which of the following micro-pathological findings is most accurately related to this case?

A. Dermatan sulfate in connective tissues
B. Glucocerebrosides in mononuclear phagocytes
C. Sphingomyelin in mononuclear phagocytes
D. Gangliosides in brain cells
E. Heparan sulfate in tissues

Which of the following enzymatic deficiencies is the most likely cause of the above problems?

A. Hexosaminidase A
B. Hexosaminidase A and B
C. Aryl sulfatase
D. α-galactosidase A
E. Glucocerebrosidase

Mixed Excerpts of Various Northwestern Medical Review Publications, 2005
### Autonomic Control On Different Organs

<table>
<thead>
<tr>
<th>Organ</th>
<th>Sympathetic</th>
<th>Parasympathetic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heart</strong></td>
<td>• Increases rate (chronotropic)</td>
<td>• Decreases rate (chronotropic)</td>
</tr>
<tr>
<td></td>
<td>• Increases contractility of ventricles (inotropic)</td>
<td>• No effect on contractility (there is no PSN innervations on ventricles)</td>
</tr>
<tr>
<td></td>
<td>• Constriction of vessels via $\alpha$ receptors</td>
<td>• No effect on blood vessels (no PSN innervations)</td>
</tr>
<tr>
<td></td>
<td>• Dilation of smooth muscles via $\beta$ receptors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Dominant system</td>
<td></td>
</tr>
<tr>
<td><strong>Blood Vessels</strong></td>
<td>• Decreases motility</td>
<td>• Increases motility</td>
</tr>
<tr>
<td></td>
<td>• Contracts sphincters</td>
<td>• Relaxes sphincters</td>
</tr>
<tr>
<td></td>
<td>• Inhibition of exocrine glands</td>
<td>• Stimulates exocrine glands</td>
</tr>
<tr>
<td></td>
<td>• Liver: Glycogenolysis, and gluconeogenesis</td>
<td>• Liver: Glycogen synthesis</td>
</tr>
<tr>
<td></td>
<td>• Dominant system</td>
<td>• Dominant system</td>
</tr>
<tr>
<td><strong>GI Tract</strong></td>
<td>• Relaxes bronchioles</td>
<td>• Contracts bronchioles</td>
</tr>
<tr>
<td></td>
<td>• Decreases mucus secretion</td>
<td>• Increases mucus secretion</td>
</tr>
<tr>
<td></td>
<td>• Urinary retention and decreased flow</td>
<td></td>
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<tr>
<td><strong>Lung and Bronchi</strong></td>
<td>• Urination and increased bladder activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Urination and increased bladder activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Dominant system</td>
<td></td>
</tr>
<tr>
<td><strong>Eye</strong></td>
<td>• Contracts radial muscle (pupillary dilation)</td>
<td>• Contracts circular and ciliary muscles</td>
</tr>
<tr>
<td></td>
<td>• Elevates upper eyelid</td>
<td>(pupillary constriction)</td>
</tr>
<tr>
<td></td>
<td>• Dominant tone</td>
<td>• Dominant tone</td>
</tr>
<tr>
<td><strong>Urinary System</strong></td>
<td>• Ejaculation</td>
<td>• Penile/Clitoral erection</td>
</tr>
<tr>
<td><strong>Sex Organ</strong></td>
<td>• Contract piloerector muscles (causes piloerection)</td>
<td>• No direct effect on sweating or vessels</td>
</tr>
<tr>
<td></td>
<td>• Stimulate sweat secretion (a cholinergic reaction)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Dominant System</td>
<td></td>
</tr>
<tr>
<td><strong>Skin</strong></td>
<td>• Dominant System</td>
<td></td>
</tr>
</tbody>
</table>

### Erection and Ejaculation:

**Sympathetics versus Parasympathetics**

- **Sympathetic**: Shoot.
- **Parasympathetic**: Point.

Please see the chapter on Respiratory System for information on Kartagener’s Syndrome.

### Secretory Functions Are All Parasympathetic

- Secretary glands no matter irrespective of their location are all activated by the cholinergic system (acetylcholine).
- Lacrimation, salivary GI acid and enzyme, bronchial and sweat secretions are all stimulated (mediated) by the acetylcholine!

**Full activation of sympathetic nervous system, as in maximal exercise, can produce all of the following responses EXCEPT:**

- A. Mydriasis
- B. Increased renal flow
- C. Decreased intestinal motility
- D. Bronchodilation
- E. Tachycardia
About Mnemonic of LPN for β-Blockers

LPNs (Licensed Practical Nurses) are the least specialized nurse practitioners. Quite often, as opposed to Registered Nurses, they are assigned to non-specialty jobs at various hospital units. Following mnemonic relates to the fact that LPNs may perform all sorts of low-level jobs at many hospital wards including cardiac and pulmonary wards!

Note: Non-selective beta-blockers (like LPNs) not only can work on the heart but can also affect the lung function.

Non-Selective β-Blockers

Contraindication of Non-Selective β-Blockers

COPD

Asthma

More on Propranolol

- Epinephrine stimulates K⁺ entry into the cells. Propranolol blocks epinephrine’s effect. Hence, it causes hyperkalemia.
- Propranolol causes vasodilation, decreases blood pressure and heart rate. Activity of β-adrenergics increases glucagon level. Glucagon increases blood sugar. Hence, propranolol reverses this function and causes hypoglycemia.
- Propranolol increases PR interval.

A 55-year-old man is brought to the emergency room. He is dyspneic; heart rate, 45; and blood pressure 85/40 mmHg. Lab results confirm hyperkalemia, and hypoglycemia. ECG indicated a normal QRS, and prolonged PR interval. His wife explains that he has been taking a medication for his hypertension. Of the following, the most likely cause of this patient’s condition would be:

(A) Atenolol overdose
(B) Captopril toxicity
(C) Phenylephrine toxicity
(D) Prazocin overdose
(E) Propranolol toxicity

Why it is important to monitor diabetics on non-selective β-blockers?

Name 4 antihypertensive autonomic medications that cause depression?

R
M
C
P

Answer to “Why it is important to monitor diabetics on non-selective β-blockers?”

- These blockers decrease glycogenolysis and drop glucose level. May exacerbate hypoglycemic effect of insulin.
- May cause bradycardia
- Bradycardia may cover the very important sign of diabetic hypoglycemia—tachycardia.
**Hydroxysteroid Dehydrogenase Deficiency**
- Congenital adrenal hyperplasia. Lack of steroids removes negative feedback over the pituitary and ACTH production stays uninhibited. Constant ACTH production causes hypertrophy of the adrenal cortex.
- Pregnenolone is not converted to all other subsequent steroids.
- All pathways; mineralosteroids, corticosteroids and glucocorticoids are affected.
- The most lethal of all steroid deficiencies after desmolase. Early death.

**17-Hydroxylase Deficiency**
- Congenital adrenal hyperplasia.
- Conversion of progesterone to subsequent sex hormones and glucocorticoids is affected.
- Female characteristics are preserved, but secondary developments are affected.
- Pathways are shunted to further mineralocorticoids production.
- Increased mineralocorticoids leads to sodium and water retention and Hypertension

**21-Hydroxylase Deficiency**
- The most common deficiency.
- Congenital adrenal hyperplasia.
- Deficiency of mineralocorticoids and glucocorticoids.
- Due to lack of feedback over pituitary there is a high level of ACTH in the plasma.
- Pathways are shunted to sex steroid production
- Adrenal virilism. Male phenotype exaggerated.
- Salt loss and hypotension

**11-Hydroxylase Deficiency**
- Deficiency of corticosterone, aldosterone and cortisol.
- Increased level deoxycorticosterone leads to salt and fluid retention, and hypertension.
- Hypertension is due to accumulation of deoxycorticosterone that has a mineralocorticoids effect.
- Like 21-α-hydroxylase leads to adrenal virilism (AKA. adrenogenital syndrome) and masculinization. More common in males.
- Less common than 21-hydroxylase deficiency
• This antibiotic is both bacteriostatic and bactericidal.
• It acts on both gram-positive and negative bacteria except proteus and pseudomonas.
• It is effective against E. coli and enterococci UTIs.
• It is filtered into the kidneys.
• Side effects are: polyneuritis, brown urine and hemolytic anemia in glucose-6-phosphate dehydrogenase deficient persons.
• Hint: It is not sulfonamide!
• This drug is: _______________________

<table>
<thead>
<tr>
<th>Urinary Tract Drugs</th>
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<tbody>
<tr>
<td>QUinolones</td>
<td>Urinary infection!</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>In-to-Uran!</td>
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<tr>
<td>Methenamine</td>
<td>At low pH is converted to formaldehyde.</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td>E.g. E coli &amp; Neisseria</td>
</tr>
<tr>
<td>TMP/SMX</td>
<td>E.g. E coli &amp; Proteus</td>
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Top Three Causes of Red Cell Sickling
- Hypoxia
- Dehydration
- Acidosis

Normal fetal hemoglobin is $\alpha_2\gamma_2$ called HbF
Normal adult hemoglobin is $\alpha_2\beta_2$ called HbA
Sickle Cell: Defect is in $\beta$ chain results in HbS

Hereditary Spherocytosis (HS)
- Most common hereditary hemolytic anemia of whites (1:5000). Autosomal dominant pattern.
- Deficiency and defect of spectrin; a membrane protein that maintains cytoskeleton. Defective cells have less surface area and tend to assume a spherical shape.
- Hallmark: Spherical and small RBCs, osmotic fragility, and autohemolysis.
- Indirect bilirubinemia, splenic sequestration, splenomegaly, gallstones, and cholecystitis.
- Note: Hemolysis is confined to the spleen and results from the interplay of intact spleen and red cells with defective spectrin. The outcome is sequestration of the RBCs in the spleen.
- Folic acids and splenectomy are helpful.
- Splenectomized children are susceptible to encapsulated bacteria—like streptococcus pneumoniae. Avoid splenectomy, if anemia is mild, to after age 10 or, give pneumococcus, and diplococcus vaccine pre-and post surgery supplanted with penicillin.

Mixed Excerpts of Various Northwestern Medical Review Publications, 2005
### Brachial Plexus

Stories That Happen Below the Neck

<table>
<thead>
<tr>
<th>Dr. AMCU! AMCU Drive!</th>
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<tbody>
<tr>
<td>Dr. Drop-hand, Radial</td>
</tr>
<tr>
<td>Ape-hand, Median</td>
</tr>
<tr>
<td>Claw-hand, Ulnar</td>
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</tbody>
</table>

### Brachial Plexus Nerves: Lateral to Medial

<table>
<thead>
<tr>
<th>MAM RU by any chance Lona Thorci?</th>
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</thead>
<tbody>
<tr>
<td>I'm Lora Turner! Can't you tell by looking at me?</td>
</tr>
</tbody>
</table>

#### Waiter's Tip Injury

AKA. Erb-Duchenne Palsy. Upper brachial plexus (C5-C6) damage. Extended, internally rotated, and adducted arm, with phonated forearm.

**Classic Case:** A newly born infant who had excessive stretching of the neck during delivery.

#### Erb's Palsy

- **Waiter's Tip**
- **Adduction Injury**
- **Upper Brachial Plexus:** C5 - C6
- **Major Cause:** Violent stretch (pull) of neck in delivery
- **Complication:** Phrenic nerve damage (ipsilateral diaphragmatic hernia)

#### Klumpke's Palsy

- **Claw Hand**
- **Abduction Injury**
- **Lower Brachial Plexus:** C7 - T1
- **Major Cause:** Forceful pull of the upper limb
- **Complication:** Horner's syndrome (ipsilateral damage to sympathetic T1)

### KLaw hand!

"KL" = K-Klumpke; L-Lower trunk

#### Winging of Scapula

- **Long thoracic (LT) nerve lesion usually close to vertebral column leads to serratus anterior palsy.**
- **LT supplies serratus anterior (which holds scapula to the rib cage).**
- **Scapula protrudes when patient pushes against wall.**
- **Radical mastectomy may injure LT in the thorax.**

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Make a match between the following patients (descriptions 1 through 5) and conditions (A through E):  

1. A patient with recent history of elbow injury is presented with inability to flex or oppose thumb, inability to flex wrist to the radial side.  
2. A female patient with recent history of radical mastectomy who has difficulty in abducting her arm.  
3. A patient with recent history of middle third humeral fracture and inability to dorsiflex his hand over forearm.  
4. A patient with recent history of fall on shoulder with biceps brachial weakness.  
5. A patient with recent history of fracture of humeral head, and deltoid weakness.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Condition</th>
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<tbody>
<tr>
<td>1.</td>
<td>A. Long thoracic nerve injury</td>
</tr>
<tr>
<td>2.</td>
<td>B. Median nerve palsy</td>
</tr>
<tr>
<td>3.</td>
<td>C. Musculocutaneous nerve injury</td>
</tr>
<tr>
<td>4.</td>
<td>D. Radial nerve palsy</td>
</tr>
<tr>
<td>5.</td>
<td>E. Axillary nerve lesion</td>
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