Evidence Based Adult Treatment Guidelines 2010: Clinical Practice Strategies for the Retail Clinician

Developed and Presented by:

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Facilitated by Partners In Healthcare Education, LLC
Part 2 of 2

Evidence Based Adult Treatment Guidelines 2010:
CARDIOVASCULAR
Insulin resistance

- Defined clinically as a state in which a given increase in plasma insulin in an individual causes less of an effect in lowering the plasma glucose than it does in a normal population.

Insulin resistance

- Primarily, the plasma glucose level regulates the physiologic secretion of insulin
- When an individual develops insulin resistance, the normal amount of insulin is not able to maintain normal plasma glucose levels
- To compensate, insulin secretion is increased until the plasma glucose levels return to normal


Interrelation between atherosclerosis and insulin resistance

- Hypertension
- Obesity
- Hyperinsulinemia
- Diabetes
- Hypertriglyceridemia
- Small, dense LDL
- Low HDL
- Hypercoagulability

Slide source: Lipidsonline.org
Factors which increase suspicion for presence of insulin resistance

- Cerebrovascular disease
- Hypertension
- PCOS
- NAFLD (NASH)
- Acanthosis nigricans
- Numerous skin tags
- Central adiposity

- Sedentary lifestyle
- Age > 40 years
- History of gestational diabetes
- Family history of diabetes
Insulin resistance: Early diagnosis

- **Dyslipidemia**
  - Increase in VLDL, Small LDL
  - Decrease in HDL (number and size)
- **NASH** (hepatic manifestation of IR)
  - ALT/AST > 1
- **Increased Insulin levels**
  - > 30 microU/ml
- **Hypertension**
  - Systolic > 130 mmHg; Diastolic > 80 mmHg
Insulin resistance – Early diagnosis

- Central Obesity
  - Waist circumference
    - > 35 inches in women
    - > 40 inches in men

- Glucose
  - Normal glucose yet with multiple risk factors
  - $\geq 100$ mg/dL
  - A1C $\geq 6.5\%$

First World Congress on Insulin Resistant Syndrome; 2003, Nov20-23, Los Angeles, California, USA

Earliest markers of target organ damage...

- **NASH**
  - ALT/AST > 1
    - Ultrasound to verify absence of lesions
    - Hepatitis ABC screen

- **Proteinuria**
  - Spot urine for microalbuminuria > 30

First World Congress on Insulin Resistant Syndrome; 2003, Nov 20-23, Los Angeles, California, USA
Therapies for diabetes

- Beta-cell dysfunction
- Sulfonylureas
- Meglitinides
- DPP-4 inhibitors
- Incretin Mimetics
- Hepatic glucose overproduction
- ↓Glucose level
- Insulin resistance
- Muscle and fat
- Pancreas
- Liver
- Muscles and fat
- Biguanides
- TZDs
- DPP-4 inhibitors
- Incretin Mimetics
- Glucose absorption
- Alpha-glucosidase inhibitors
- Biguanides
- Bile acid sequestrants

DPP-4 = dipeptidyl peptidase-4; TZDs = thiazolidinediones.
Hypertension
Due out in fall of 2010

– Thought to consider ACE/ARB as therapeutically interchangeable
– Thought to change treatment of more compelling indications with ARB
– Thought to change the blood pressure recommendations for elderly to higher systolic than mid 130’s
### JNC 7 Compelling Indications

<table>
<thead>
<tr>
<th></th>
<th>Diuretic</th>
<th>BB</th>
<th>ACEI</th>
<th>ARB</th>
<th>CCB</th>
<th>AA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Failure</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Post MI</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>CAD risk</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Renal disease</td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Recurrent stroke</td>
<td>✓</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BB, beta blocker; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; AA, aldosterone antagonist; HF, Heart Failure; MI, myocardial infarction; CAD, coronary artery disease; DM, diabetes mellitus

JNC 7 Algorithm for Treatment of Hypertension

Lifestyle Modifications

Not at Goal Blood Pressure (<140/90 mmHg) (<130/80 mmHg for those with diabetes or chronic kidney disease)

Initial Drug Choices

Without Compelling Indications

**Stage 1 Hypertension**
(SBP 140–159 or DBP 90–99 mmHg)
Thiazide-type diuretics for most.
May consider ACEI, ARB, BB, CCB, or combination.

With Compelling Indications

**Stage 2 Hypertension**
(SBP ≥160 or DBP ≥100 mmHg)
2-drug combination for most (usually thiazide-type diuretic and ACEI, or ARB, or BB, or CCB)

Drug(s) for the compelling indications
Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.

Not at Goal Blood Pressure

Optimize dosages or add additional drugs until goal blood pressure is achieved.
Consider consultation with hypertension specialist.

Assessed 5-1-08
### JNC 7: Classification and Management of Blood Pressure

<table>
<thead>
<tr>
<th>Category</th>
<th>SBP* mm Hg</th>
<th>DBP* mm Hg</th>
<th>Lifestyle modification</th>
<th>Considerations for Initial Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Without Compelling Indications</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
<td>Encourage</td>
<td></td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
<td>Yes</td>
<td>No antihypertensive drug indicated</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140-159</td>
<td>or 90-99</td>
<td>Yes</td>
<td>Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>≥160</td>
<td>or ≥100</td>
<td>Yes</td>
<td>2-drug combination for most** (usually thiazide-type diuretic and ACEI or ARB or BB or CCB)</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; DBP, diastolic blood pressure; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; CCB, calcium channel blocker.

*Treatment determined by highest BP category.

**Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.

†Treat patients with chronic kidney disease or diabetes to BP goal of <130/80 mm Hg.
Hypertension and Management: Old School

Hypertension = Systemic disease

Hemodynamics altered

Treat the blood pressure

Therapeutic options

- Beta Blockers
- ACE
- ARB
- Diuretics
- CCB
- Others

Adapted from Vascular Biology Working Group, University of Florida College of Medicine, Carl Pepine, MD, Director
Hypertension and Management: **New School**

Hypertension = Disease of the blood vessels

Vascular biology altered

Treat the vasculature

Therapeutic options

- Beta Blockers
- ACE
- ARB
- Diuretics
- CCB
- Others

Adapted from Vascular Biology Working Group, University of Florida College of Medicine, Carl Pepine, MD, Director
Chest Pain/Discomfort
Acute Coronary Syndrome

• Consists of
  – Unstable Angina
  – Non ST segment elevation MI
  – ST segment elevation MI

• Change in standards of practice published by AHA/ACC in 2000

• Updated in 2002
  – Circulation 2002;106:1893-900
Coronary Artery Disease -- 2010

- In 2005, CHD caused 1 in 5 deaths in the United States
  - Single largest killer of American males and females
- About every 25 seconds, an American will suffer a coronary event
  - In the US, one person dies every 1 minute from CHD
- In 2009, estimated direct and indirect costs of CHD are 165.4 billion dollars.

Heart Disease and Stroke Facts – 2009 Update, American Heart Association, Dallas Texas.
CAD prevention.....

- The process of retarding the natural progression of vascular obstruction that leads to myocardial ischemia, injury or infarction.
  - Anticipating
  - Thwarting
  - Forethought of cause

- What are the goals of prevention?
Endothelium: The root cause?

- Injury to the endothelium appears to be the key event in:
  - The origin
  - The progression
  - The clinical manifestation of atherosclerotic plaques

- Endothelial dysfunction increases the likelihood for the presence of other risk factors

Risk factors for the development of endothelial dysfunction

- Catalysts of **endothelial dysfunction**
  - Aging
  - Insulin resistance
  - Impaired glucose tolerance/Impaired fasting glucose
  - Hypertension
  - Dyslipidemia
  - Tobacco use
  - Sedentary lifestyle
  - Diet

Heart Disease and Stroke Facts – 2009 Update, American Heart Association, Dallas Texas.
Endothelium

Dysfunctional endothelial cells in dyslipidemia and atherosclerosis

The endothelium

Inner-most lining of the vessel

The endothelium is the largest organ in the body!

- **Total Surface Area:** About 6 tennis courts
- **Total Mass:** About 5 normal hearts
- **Total Weight:** Approx. 1800 grams (> liver)
- **Total # Cells:** About 1 trillion cells

Endothelium: Early diagnosis

- Early diagnosis of endothelial issues is very similar to Class I classification of heart failure. “Patients at risk for development”
- Patients at risk for developing endothelial dysfunction
  - Insulin resistance
  - Dyslipidemia
  - Hypertension
  - Tobacco user

The endothelium maintains vascular health

Dilatation
Growth inhibition
Antithrombotic
Anti-inflammatory
Antioxidant

Constriction
Growth promotion
Prothrombotic
Proinflammatory
Pro-oxidant

Vascular Biology Working Group, University of Florida
College of Medicine, Carl Pepine, MD, Director
Risk factors for endothelial dysfunction

- Dyslipidemia
- Hypertension
- Diabetes
- Smoking
- Sedentary Lifestyle
- Heart Failure
- Diet

Oxidative Stress

Endothelial Dysfunction

Adapted from Vascular Biology Working Group, University of Florida College of Medicine, Carl Pepine, MD, Director
What pathology are we working on?
Acute Coronary Syndrome

- Consists of
  - Unstable Angina
  - Non ST segment elevation MI
  - ST segment elevation MI

- Change in standards of practice published by AHA/ACC in 2000

- Updated in 2002
  - *Circulation* 2002;106:1893-900
Acute Coronary Syndrome

• Results from an interaction between a vulnerable atherosclerotic plaque and thrombus formation – atherothrombosis
• Two major players – atherothrombosis
  – Lipoprotein accumulation
  – Chronic inflammation
  • Chronic inflammation being intimately involved in plaque rupture and thrombosis

Evolution of the Acute MI
Treatment of Suspected Acute Coronary Syndrome

- Activate 911
- Administer aspirin 81 mg – 325 mg – preferably chewed
- Administer nitroglycerin as directed
- Monitor closely until EMS arrives
Lipid Disorders
# NCEP Interim Report:
LDL-C Goals and Drug Cut Points for High-Risk Patients

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Risk Category</th>
<th>LDL-C Goal (mg/dL)</th>
<th>LDL-C to Consider Drug Therapy* (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderately High</td>
<td>≥2 Risk Factors; 10-Year Risk 10%-20%</td>
<td>&lt;130 &lt;100†</td>
<td>≥130** 100-129‡</td>
</tr>
<tr>
<td>High Risk</td>
<td>CHD or CHD Risk Equivalents; 10-Year Risk &gt;20%</td>
<td>&lt;100</td>
<td>≥100**</td>
</tr>
<tr>
<td>Very High Risk</td>
<td>Established CVD Plus:</td>
<td>&lt;100</td>
<td>≥100**</td>
</tr>
<tr>
<td></td>
<td>• Multiple Major Risk Factors</td>
<td>&lt;70†</td>
<td>&lt;100‡</td>
</tr>
<tr>
<td></td>
<td>• Severe and Poorly Controlled Risk Factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Multiple Risk Factors of the Metabolic Syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Acute Coronary Syndromes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*When LDL-C-lowering drug therapy is used, the intensity of therapy should be sufficient to achieve a 30%-40% reduction in LDL-C; **Therapeutic lifestyle changes (TLC) should be initiated when LDL-C is at or above goal; any high-risk or moderately high-risk patient who has lifestyle-related risk factors is a candidate for TLC regardless of LDL-C level; †Optional LDL-C goal; ‡Consider drug options.


[www.lipidsonline.org](http://www.lipidsonline.org)
## Key Lipid Lowering Agents

<table>
<thead>
<tr>
<th>Agent</th>
<th>Target of Impact</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>HMG-CoA reductase inhibitors</td>
<td>Liver</td>
<td>Decrease manufacturing LDL – lower LDL</td>
</tr>
<tr>
<td>Bile acid sequestrants</td>
<td>Gut</td>
<td>Absorb bile acid – Lower LDL</td>
</tr>
<tr>
<td>Cholesterol absorption inhibitors</td>
<td>Brush border small intestine</td>
<td>Block re-absorption of bile acid – Lower LDL</td>
</tr>
<tr>
<td>Nicotinic acid derivative</td>
<td>Liver</td>
<td>Increase HDL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Decrease triglycerides</td>
</tr>
<tr>
<td>Fibrates</td>
<td>Liver</td>
<td>Decrease triglycerides</td>
</tr>
<tr>
<td>Omega-3 acid ethyl esters</td>
<td>Liver</td>
<td>Decreased triglycerides</td>
</tr>
</tbody>
</table>
Percentage Change in LDL-C: 4 leading statins

**The STELLAR Trial**

Change in LDL-C From Baseline (%)

- **Rosuvastatin**: 10 mg *, 20 mg **, 40 mg †
- **Atorvastatin**: 10 mg, 20 mg, 40 mg, 80 mg
- **Simvastatin**: 10 mg, 20 mg, 40 mg, 80 mg
- **Pravastatin**: 10 mg, 20 mg, 40 mg

STELLAR = Statin Therapies for Elevated Lipid Levels Compared Across Doses to Rosuvastatin.


www.lipidsonline.org
PROVE IT-22

Cumulative Incidence of Recurrent MI or CHD Death by Achieved Levels of LDL-C and CRP

PROVE IT = PRavastatin or AtOrVastatin Evaluation and Infection Therapy  N=3745; pravastatin 40 mg vs atorvastatin 80 mg


www.lipidsonline.org
Treatment Changes

- Accent of use of polypharmacy
- Emphasis on treatment of HDL
  - 4 genotypes of HDL have been identified
  - Some genotypes respond excellently to fish oil and should be tried initially
  - If the HDL goal is not reached either change to nicotinic acid or add nicotinic acid to fish oil treatment
Polypharmacy is Emphasized

• Single agent treatment of dyslipidemias has not been as successful as polypharmacy to achieve LDL, HDL, and triglyceride goals
  – Statin + bile acid sequesterant
  – Statin + Niacin
  – Statin + fish oil (+/-) Niacin
  – Addition of fenofibrate to above
# Proposed Algorithm for Treatment

<table>
<thead>
<tr>
<th>Issue</th>
<th>Statins</th>
<th>BAS</th>
<th>CAI</th>
<th>Niacin</th>
<th>Fibrates</th>
<th>Omega 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL #</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>LDL Size</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>++++</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>HDL #</td>
<td>+++</td>
<td>+</td>
<td>+/-</td>
<td>++++</td>
<td>+++</td>
<td>+/-</td>
</tr>
<tr>
<td>VLDL #</td>
<td>+++</td>
<td>+/-</td>
<td>+/-</td>
<td>+++</td>
<td>++++</td>
<td>+++</td>
</tr>
<tr>
<td>Lp(a) #</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>+++</td>
<td>+</td>
<td>0</td>
</tr>
</tbody>
</table>
Peripheral Vascular Disease
Common Sites of Claudication

Obstruction in:
- Aorta or iliac artery
- Femoral artery or branches
- Popliteal artery

Ischemia in:
- Buttock, hip, thigh
- Thigh, calf
- Calf, ankle, foot
Clinical Treatment Goals for Patients With PAD

- Improve functional status
- Preserve the limb
- Prevent progression of atherosclerosis
- Reduce cardiac and cerebrovascular mortality
- Decrease the need for revascularization
- Reduce nonfatal events such as MI and stroke

# Medications Currently Indicated for Intermittent Claudication

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Pentoxifylline- Trental</th>
<th>Cilostazol -Pletal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug Class</td>
<td>Nonselective phosphodiesterase inhibitor</td>
<td>Phosphodiesterase III inhibitor</td>
</tr>
<tr>
<td></td>
<td>Methylxanthine derivative</td>
<td>Quinolinone derivative</td>
</tr>
<tr>
<td>Dosing</td>
<td>400 mg tid</td>
<td>100 mg bid</td>
</tr>
<tr>
<td>Pharmacologic properties</td>
<td>Hemorrheologic agent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Weak antiplatelet activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Some vasodilation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Platelet aggregation inhibitor</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Vasodilation</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ HDL-C (10%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Triglycerides (15%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inhibits smooth muscle cell proliferation in vitro</td>
<td></td>
</tr>
</tbody>
</table>

Walking

- Cornerstone of conservative therapy to improve the patient’s functional capacity is an exercise program.
- 3 times a week for 1 hour
- Walk until mild or moderate amount of pain
- Rest until pain subsides
- Resume walking
- Increase the pace to 1.5 or 2 mph; increase workload by increasing grade or speed

*J Vasc Surg. 2000;31:S1-S296*
Summary: Walking Programs

• Consistency of these findings suggests that exercise training programs have a clinically important impact on functional capacity in patients with PAD

J Vasc Surg. 2000;31:S1-S296
Deep Venous Thrombosis
## Risk Factors

<table>
<thead>
<tr>
<th>Primary Risk Factors</th>
<th>Secondary Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major surgery</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>Previous DVT</td>
</tr>
<tr>
<td>Major trauma</td>
<td>Immobilization</td>
</tr>
<tr>
<td>Paralytic stroke</td>
<td>Obesity</td>
</tr>
<tr>
<td>Cancer</td>
<td>Chronic respiratory failure</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>Increasing age</td>
</tr>
<tr>
<td>Pelvic fracture</td>
<td>Hematologic disorders</td>
</tr>
<tr>
<td></td>
<td>Central venous catheter</td>
</tr>
<tr>
<td></td>
<td>Varicose veins</td>
</tr>
<tr>
<td></td>
<td>Pregnancy</td>
</tr>
<tr>
<td></td>
<td>Estrogen treatment</td>
</tr>
</tbody>
</table>

Hypercoagulability

- Protein C deficiency
- Protein S deficiency
- Antithrombin III deficiency
- Activated Protein C resistance
- TPA deficiency
- Plasminogen activator inhibitor
- Leiden Factor V mutation

Diagnosis

• Diagnosis is difficult at best and may be based on probable risk factors
• Typical signs and symptoms are not always present
• Classic Signs
  – Unilateral limb swelling not resolving with elevation
  – Calf pain
  – Redness and / or palpable cord
• Diagnosis – ultrasound is most cost effective

Treatment DVT

- Heparin to coumadin bridge
- Low molecular weight heparin to coumadin bridge
- Inferior vena cava filter for candidates where risk benefit ratio is not in favor of anticoagulation
- Care in placement of IVC filter to avoid SCV syndrome – hard to remove!
GASTROENTEROLOGY
Diarrhea
Diarrhea

• Sudden onset of increased watery stools
• Biggest risk: issues with dehydration, electrolyte imbalance
• Signs of dehydration
  – Thirst, decreased urination, dark urine, fatigue
  lightheadedness, confusion
• Categories
  – Acute – sudden onset; > 2 days
  – Persistent – lasting for 2 weeks or longer
  – Chronic – lasting for > 1 month

Diarrhea accessed 7-26-08 at
http://digestive.niddk.nih.gov/ddiseases/pubs/diarrhea/#cause
Causes

• Acute Diarrhea
  – Bacteria (contaminated food or water)
    • Campylobacter, Salmonella, Shigella, E. Coli
  – Virus
    • Rotavirus, Norwalk virus, cytomegalovirus, viral hepatitis
  – Food intolerances
    • Artificial sweeteners, lactose
    • Ingestion of fiber, bran, fructose, fruit

Diarrhea accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/diarrhea/#cause
Causes

• Reaction to medications
  – Antibiotics, hypertensive medications, diabetic medications (metformin), antacids containing magnesium, antiarrhythmics, prokinetics (macrolides)

• Intestinal diseases
  – Inflammatory bowel disease (Ulcerative colitis, Crohn’s disease), Celiac disease

• Functional Bowel Disorders
  – IBS-D or IBS - M
Causes

– Antibiotic associated diarrhea
  • Oral antibiotics – few hours to 2 months after use of antibiotics
  • *C. Difficile* must be entertained

– Parasites (food and water)
  • *Giardia lamblia, Entamoeba histolytica, Cryptosporidium*

Diarrhea accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/diarrhea/#cause
Diagnosis & Treatment

• Comprehensive history and physical
• Stool cultures
• CBC – wbc count and eosinophils
• Sigmoidoscopy, colonoscopy, imaging tests
• Treatment:
  – Treat the cause (bacteria, virus, structural, food ingestion)
  – Rehydration and electrolyte balance

Diarrhea accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/diarrhea/#cause
Nausea & Vomiting
Nausea & Vomiting

• Significant causes
  – Iatrogenic causes
    • Chemotherapy
  – Infectious causes
    • Viral and bacterial
  – Gastrointestinal disorders
    • Appendicitis, cholecystitis, pancreatitis, obstruction or motility issues
  – Central nervous system issues
    • Migraine or increased intracranial pressure

Nausea & Vomiting

- **Endocrine**
  - Pregnancy

- **Psychiatric issues**
  - Stress, anxiety, physical stressors

- **Hypotension**
  - Cardiac
  - Bleeding
  - Sepsis

AGA Guidelines: Nausea & Vomiting

• Recognize and correct symptoms
  – Dehydration or electrolyte imbalance

• Identify underlying cause
  – History and physical
    • Abdominal pain suggests organic causes
    • Warning signs for immediate intervention
      – Chest pain, CNS symptoms, high fever, older age, immunosuppressed patients, severe dehydration
  – Treat the specific pathophysiology

AGA Guidelines: Nausea & Vomiting

• Empiric therapy, if no cause can be identified
  – Phenothiazines (prochlorperazine = compazine)
  – Prokinetic agents (metoclopramide = reglan)
  – Serotonin antagonists (ondansetron = zofran)

Appendicitis
Appendicitis

• Small tube-like structure attached to the first part of the large intestine with no known function
• Most often occurs: ages 10 – 30 years
• Inflammation related to blockage of the lumen of the appendix
  – Increased pressure, impaired blood flow, inflammation, and potential rupture with peritonitis

Appendicitis accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/appendicitis/
Appendicitis

• Causes:
  – Feces
  – Bacterial or viral infections in the GI tract result in swelling of lymph nodes
  – Traumatic abdominal injury

Appendicitis accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/appendicitis/
Appendicitis

• Symptoms
  – Pain in the abdomen, first at umbilicus then moving to the RLQ
  – Loss of appetite
  – Nausea
  – Vomiting
  – Constipation or diarrhea
  – Inability to pass gas
  – Low fever that begins after other symptoms
  – Abdominal swelling

Appendicitis accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/appendicitis/
Specialized Maneuvers

• Markle’s test
  – Heel jarring test; tapping on heel produces pain in the RLQ

• Psoas sign
  – Inactive elongation of the patient’s thigh by stretching the knees. In this test, the patient has to be positioned on the left side of his or her body while the right leg is stretched toward the back of the patient.
  – This is considered positive if pain is produced in the RLQ.

• Obturator sign
  – Considered positive if the patient experiences RLQ pain while you flex the hip and knee and internally rotate the leg
Diagnosis and Treatment

• CBC with differential
  – Left shift (leukocytosis, neutrophilia and bandemia)
• Helical CT scan
• Surgical removal

Appendicitis accessed 7-26-08 at http://digestive.niddk.nih.gov/ddiseases/pubs/appendicitis/
Acute Abdomen
The Acute Abdomen

• Differential Diagnosis
  – Ectopic Pregnancy/Miscarriage
  – Appendicitis or perforation (see prior section)
  – Infectious diarrhea
  – Gastrointestinal bleed

The Acute Abdomen

• Differential Diagnosis
  – Diverticulitis
    • Attacks of localized abdominal pain, LLQ, signs of inflammation, rigidity to rebound noted in severe cases
  – Ulcer perforation
    • Abrupt onset, epigastrium, absent bowel sounds, abdominal rigidity
  – Pancreatitis
    • Nausea, vomiting, dehydration, mid-epigastric pain that is constant; pain may radiate to back

The Acute Abdomen

Cholecystitis
- Unrelenting and intensifying pain at the right side of the abdomen; fever, vomiting, Murphy’s sign

- Small bowel obstruction
  - Diffuse colicky pain, nausea, vomiting, altered bowel sounds, distention, dehydration, diffuse tenderness, possibly ill defined mass

- Large bowel obstruction
  - Malignancy is most common cause; insidious onset; similar to SBO; feculent emesis, weight loss and anemia

- AAA
  - Frequently asymptomatic even in large aneurysms; abdominal pain, backache, claudication Triad = back pain, pulsatile mass and hypotension

Lyon, C. Clark, D. Diagnosis of Acute Abdominal Pain in Older Patients Am Fam Physician 2006;74:1537-44.
The Acute Abdomen

• Mesenteric artery occlusion/ischemia
  – Celiac, superior and inferior mesenteric arteries
  – Very often missed
  – Severe poorly localized abdominal pain that is out of proportion for findings
  – 1/3rd will have nausea, vomiting and diarrhea
  – Often have BRB per rectum

• Trauma (organ rupture)

Exam

- Abdominal pain > 5 on 10 with sudden onset or rapidly worsening pain
- Guarding
- Rigidity
- Rebound tenderness (Blumberg sign)
- Leukocytosis
- Ecchymosis on abdomen (Cullen’s sign)
- Hematuria

GU
UTI’s in Women
UTI’s in Women

• Significantly more common in women than men
  – Proximity of urethra to anus
  – Shorter urethra than men

• Inoculation is with gram negative aerobic bacilli from the gut with *Escherichia coli* being the most common offending organism

Most Frequent Causes in Women

- New sex partner or multiple partners
- More frequent intercourse
- Diabetes
- Pregnancy
- Use of irritating products such as
  - Harsh skin cleansers, diaphragms, spermicides
- Use of birth control pills
- Heavy use of antibiotics

Presentation

- Urgency and frequency of urination
- Hematuria
- Dysuria
- Chills, fever
- Altered mental status in older women
- Pain during intercourse
- Strong, foul smelling urine
- Back pain

Complicated vs Uncomplicated

• Cystitis
  – Uncomplicated
    • Young women, non pregnant, normal anatomy, nonresistant organism, not recurrent
  – Complicated
    • Pregnant, very young or old, diabetic, immunocompromised, anatomically abnormal, catheter related, etc

Complicated vs Uncomplicated

• Pyelonephritis
  – Uncomplicated
    • Upper urinary tract infection
  – Complicated
    • Progression to involve corticomedullary abscess, perinephritic abscess, emphysematous pyelonephritis or papillary necrosis
Treatment

- Urinalysis (may perform urine dipstick in uncomplicated cases)
- C & S
- Antibiotics; consider phenazopyridine
- Increased fluids
- Prevention
  - Urination after intercourse
  - Lubricants during intercourse if atrophic vaginitis is present

Sanford Guide Recommendations

• Treatment
  – If local *E. coli* resistance to TMP/SMX < 20% and no allergy, then TMP/SMX-DS BID x 3 days
  – If local *E. coli* resistance to TMP/SMX >20% or sulfa allergy, nitrofurantoin X 7 d or fosfomycin X 1 dose

• Additional Options
  – If local *E. coli* resistance to TMP/SMX >20% or sulfa allergy, ciprofloxacin 250 mg BID, ciprofloxacin ER 500 mg qd, levofloxacin 250 mg QD, - 3 days of treatment
UTI’s in Men
UTI’s in Men

• Low incidence of UTI’s in men from 3 months to 50 years old
  – Frequent UTI’s in this age range, must consider anatomical abnormalities

• Inoculation is with gram negative aerobic bacilli from the gut with *Escherichia coli* being the most common offending organism

Presentation

• Most frequent complaint is dysuria
• Accompanied by
  – Urgency, frequency, nocturia, gross hematuria, suprapubic pain, nausea and vomiting in some
• Associated symptoms
  – Fever, chills, back/flank pain
• Differentiate UTI from urethritis
  – Sexual history and urethral swabs (*chlamydia*)

Complications in Older Men

- Prostatitis
- Pyelonephritis
  - CVA tenderness, fever, chills
    - 30-50% of pyelonephritis cases may be silent

Differentials and Concomitant Issues

- Consider differentials and concomitant issues:
  - Back pain, mechanical in origin
  - Epididymitis
  - *Chlamydia*
  - *Gonorrhea*
  - Orchitis
  - Prostatitis
  - Testicular torsion in younger patients
UTI Males: Recommendations

- 10-14 day course of antibiotics
- TMP-SMZ, nitrofurantoin or one of the fluoroquinolones, such as ciprofloxacin
- If resistance >20% locally to TMP-SMZ or nitrofurantoin, use fluoroquinolones (such as ciprofloxacin)

Sexually Transmitted Infections
Sexually Transmitted Infections

• Individuals with STI’s are often asymptomatic

• Most common:
  – *Chlamydia*
    • Most common in US
    • 13% women affected
  – *Gonorrhea*
  – *Syphilis*
  – *Trichomoniasis*
  – Genital herpes

*Sex Transm Dis. 2001;28(6):321-325*
Screening

• As the majority of these infections are asymptomatic, screening is vitally important

• Each year 2 million cases of asymptomatic Chlamydia go undiagnosed and untreated

• ACOG, CDC, U.S. Preventive Services Task Force recommend annual screening in all sexually active women age ≤ 25 years

CDC Treatment Guidelines 2006

• Syphilis or Syphilitic Chancroid
  – Azithromycin 1 g orally in a single dose
  – Ceftriaxone 250 mg IM in a single dose
  – Ciprofloxacin 500 mg orally bid for 3 days
  – Erythromycin base 500 mg orally tid for 7 days

• Genital Herpes (HSV 1 or HSV 2)
  – Acyclovir 400 tid X 7-10 days
  – Famciclovir 500 mg tid X 7-10 days
  – Valacyclovir 1 gm bid for 7-10 days

CDC Treatment Guidelines 2006

- **Chlamydia trachomatis**
  - Azithromycin 1 gram as a single dosage or
  - Doxycycline 100 mg one pill two times daily x 7 days

- **Nongonococcal urethritis**
  - Azithromycin 1 gm orally in single dose or
  - Doxycycline 100 mg bid for 7 days

- **Gonococcal Infections**
  - Ceftriaxone 250 mg IM single dosage + chlamydia treatment
  - Cefixime 400 mg as a single dosage plus treatment for chlamydia infections, if not ruled out
CDC Treatment Guidelines 2006

• Bacterial Vaginosis
  – Metronidazole 500 mg po bid X 7 days or
  – Clindamycin cream 2%, full applicator PV X 7 days or
    metronidazole gel 1 applicator PV two times daily x 5 days

• Candidiasis
  – Butoconazole 2% PV X 3 days or
  – Clotrimazole 1% cream PV 7-14 days

• Trichomononiasis
  – Metronidazole 2 gm orally single dose or
  – Tinidazole 2 gm orally single dose

Cellulitis
Cellulitis

• Acute infection of skin and or soft tissues
• Characterized:
  – Localized pain
  – Tenderness
  – Swelling
  – Redness
  – Warmth
  – Generalized malaise, fever, chills may be associated

Micali, G. Cellulitis. E Medicine July 15, 2008 accessed 7-28-08 at
http://www.emedicine.com/derm/TOPIC464.HTM
Infectious Organisms

• MRSA vs. MSSA—regionally varies in frequency
  – *Staphylococcal* cellulitis may be due to nasal carriage of *staphlococci*

• *S. pyogenes*

• *S. pneumoniae* uncommon in adults
**Treatment**

- Dicloxacillin
- Cephalexin
- Cefuroxime
- Ceftriaxone
- MRSA
  - trimethoprim/sulfamethoxazole
  - clindamycin

Contact Dermatitis
Clinical Pearls

- Poison ivy is not spread by scratching.
- No oleoresin is found in the vesicles and therefore, cannot be spread by scratching.
- Lesions will appear where initial contact with the plant occurred.
- Resin needed to be washed from the skin within 15 minutes of exposure to decrease the risk of the condition.
Clinical Presentation

- Clinical presentation
  - Characteristic linear appearing vesicles are likely to appear first
  - Often surrounded by erythema
  - Intensely itchy
  - Lesions often erupt for a period of 1 week and will last for up to 2 weeks
  - More extensive and widespread presentation can occur with animal exposures or burning of the plants / smoke exposure

Shy, BD. Contact Dermatitis. E medicine. Feb 28,2008 accessed 7-28-08
http://www.emedicine.com/emerg/TOPIC131.HTM
Contact Dermatitis
Contact Dermatitis
Treatment

• Cool compresses 15 – 30 minutes three times daily
• Topical calamine or caladryl lotions
• Zanfel (OTC) wash – binds urushiol oil and removes from body/blisters
  – 75% decrease in itching and rash within 24 hours per package
• Colloidal oatmeal baths (AVEENO) once daily

Shy, BD. Contact Dermatitis. E medicine. Feb 28, 2008 accessed 7-28-08
http://www.emedicine.com/emerg/TOPIC131.HTM
Treatment

• Oral antihistamines
  – May wish to use sedating antihistamines at bedtime

• Topical corticosteroids
  – Avoid usage on the face

• Oral prednisone vs. injectable Kenalog or similar
  – 20 mg two times daily x 7 days
  – Kenalog 40 mg injection (IM)

Shy, BD. Contact Dermatitis. E medicine. Feb 28, 2008 accessed 7-28-08
http://www.emedicine.com/emerg/TOPIC131.HTM
Follow-Up

• Monitor for secondary infections
• Impetigo
  – Staph vs. strep
  – MRSA
• Education:
  – Lesions will decrease over a 2 week period
  – May continue to erupt over 48 hours despite steroid administration
  – Not spreading lesions with rubbing or scratching

Shy, BD. Contact Dermatitis. E medicine. Feb 28, 2008 accessed 7-28-08
http://www.emedicine.com/emerg/TOPIC131.HTM
Folliculitis Furunculosis
Folliculitis

• Follicular based pustule
• Formed by presence of inflammatory cells within the wall and ostea of the hair follicle
• Acute onset
  – Papules and pustules
  – Pruritus or mild discomfort

Satter, EK. E medicine. April 17,2008 accessed 7-28-08
http://www.emedicine.com/derm/TOPIC159.HTM
Folliculitis

• Superficial vs. Deep
  – Most common cause of superficial type is *Staphylococcus aureus*
  – Superficial can turn into deep
    • Follicular centered abscess forms
      – On face – *vulgaris*
      – Occurs elsewhere – *furuncle* or boil
      – A confluence of several furuncles - *carbuncle*

Satter, EK. E medicine. April 17,2008 accessed 7-28-08
http://www.emedicine.com/derm/TOPIC159.HTM
Treatment

• Recurrent, uncomplicated folliculitis
  – Good skin care
  – Good handwashing
• If systemic antibiotics are indicated
  – Oral antibiotics that cover gram-positive organisms
    • S aureus should be covered as this is one of the most common organisms found
    • Consideration to MRSA

Satter, EK. E medicine. April 17, 2008 accessed 7-28-08
http://www.emedicine.com/derm/TOPIC159.HTM
Candidiasis
Tinea Infection
Candidiasis

• Caused by *Candida albicans*

• Ubiquitous yeast like fungi – very common fungal pathogen in humans

• Opportunistic pathogens
  – May be local
  – May be systemic

Hidalgo, JA. E medicine. July 14, 2008 accessed 7-28-08  
http://www.emedicine.com/med/TOPIC264.HTM
Candidiasis

• Oropharyngeal
• Esophageal
• Non-esophageal gastrointestinal candidiasis
• Respiratory tract
• Genitourinary tract
• Systemic

Hidalgo, JA.  E medicine. July 14, 2008 accessed 7-28-08
http://www.emedicine.com/med/TOPIC264.HTM
Presentation

• Cutaneous candidiasis
  – History
    • Generalized pruritis
    • Increased severity in the genitocrural folds, anal region, axillae, hands and feet
  – Diffuse eruption
  – Exam
    • Papules or vesicles that spread into large confluent areas
    • Trunk, thorax or extremities

Hidalgo, JA. E medicine. July 14, 2008 accessed 7-28-08
http://www.emedicine.com/med/TOPIC264.HTM
Oral Candidiasis

• Found in history of
  – HIV, dentures, diabetes, exposed to broad spectrum antibiotics or inhaled steroids

• Frequently asymptomatic but have:
  – Sore and painful mouth
  – Burning of mouth or tongue
  – Dysphagia
  – White-thick patches on the oral mucosa

Hidalgo, JA. E medicine. July 14, 2008 accessed 7-28-08
http://www.emedicine.com/med/TOPIC264.HTM
Treatment

• Cutaneous
  – Topical antifungal agents
    • Clotrimazole, miconazole, ketoconazole, nystatin
  – Oral – systemic involvement
    • Oral itraconazole (sporanox)

Hidalgo, JA. E medicine. July 14, 2008 accessed 7-28-08
http://www.emedicine.com/med/TOPIC264.HTM
Treatment

• Oropharyngeal
  – Topical antifungal agents
    • Nystatin or clotrimazole
  – Oral antifungal agents
    • Fluconazole or itraconazole

Hidalgo, JA. E medicine. July 14, 2008 accessed 7-28-08
http://www.emedicine.com/med/TOPIC264.HTM
Herpes Simplex
Herpetic Lesions

• Herpes Simplex$^1$
  – DNA viruses that cause acute skin infections
  – Recurrent; often appear in same location

1 Schinstine, M. Torres, G. Herpes Simplex. E medicine. Accessed 7-20-08
http://www.emedicine.com/derm/topic179.htm
Herpes Simplex

- HSV-1 (70-90% above the waist)
  - Face
  - Oropharyngeal
  - Ocular mucosa

- HSV-2 (70-90% below the waist)
  - Hips
  - Buttocks
  - Genitalia
  - Lower extremities

http://www.emedicine.com/derm/topic179.htm
Herpes Simplex
Simplex

• Intimate contact with a susceptible person and an individual who is actively shedding the virus
• Body fluids containing virus are required for HSV infection to occur

http://www.emedicine.com/derm/topic179.htm
Simplex

• Systemic symptoms (often dismissed by patient)
  – Fever, malaise, pain (especially primary infection)
  – HSV-2: occurs within 2 days to 2 weeks after exposure

• Clustered vesicles on an erythematous base

• Progress to ulcerated lesions which eventually form a crust

http://www.emedicine.com/derm/topic179.htm
Simplex

• Differentials
  – Aphthous Stomatitis
  – Chancroid
  – Chickenpox
  – Erythema Multiforme
  – Zoster
  – Syphilis

http://www.emedicine.com/derm/topic179.htm
Simplex

• Treatment
  – Usually self limiting (2-3 weeks)
  – Use of antiviral medications tend to shorten the course
    • Acyclovir, valacyclovir, famciclovir
  – Use of chronic, low dose antiviral medications has been shown decrease asymptomatic shedding

http://www.emedicine.com/derm/topic179.htm
Herpes Zoster
Herpes Zoster

• Highly contagious DNA virus which during the varicella infection (primary infection) gains access into the dorsal root ganglia

• Virus remains dormant for decades and is reactivated when an insult occurs to the individual’s immune system
  – Examples: HIV, chemotherapy, illness, stress, corticosteroid usage
Incidence and Prevalence

- 3 million cases of chickenpox yearly
  - Disease of childhood
- 600,000 - 1 million cases of herpes zoster each year in the United States
  - Tends to be more of a disease of aging
  - By age 80, 20% of us will have zoster at some point in our lifetime
  - Men = Women

www.niaid.nih.gov/shingles/cq.htm
Risk Factors

• Increasing age (50-60 years and beyond)
• Varicella infection when < 2 years of age
• Immunosuppression
• Stress (controversial)
• Trauma
• Malignancies
  – 25% of patients with Hodgkin’s will develop zoster¹

Goals of Treatment

- Treat acute viral infection
  - Shorten course
  - Reduce lesions
- Treat acute pain
- Prevent complications
  - Postherpetic neuralgia
Acute Treatment Options

• Antiviral
  – Goal: Reduce viral reproduction

• Corticosteroids
  – Initially postulated that these reduce viral replication; recent studies have not found this to be true
  – However, they do decrease pain

• Pain Management
  – Topical agents
  – Anti-inflammatory agents
  – Narcotics

• Postherpetic neuralgia prevention

www.aad.org/pamphlets/herpesZoster.html
Antiviral Treatment Options

• Ideally, want to begin within the first 72 hours of the eruption as benefits may be reduced if started after that

• These medications decrease duration of the rash and severity of the pain
  – Studies vary as to how much these products actually reduce the incidence of post-herpetic neuralgia

**Controlled Trials of Antiviral Agents in Herpes Zoster**

<table>
<thead>
<tr>
<th>Percentage of Patients with PHN at:</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acyclovir vs. Placebo</td>
<td>25% vs. 54%</td>
<td>15% vs. 35%</td>
</tr>
<tr>
<td>Valacyclovir vs. Acyclovir</td>
<td>31% vs. 38%</td>
<td>19.9% vs. 25.7%</td>
</tr>
<tr>
<td>Famciclovir vs. Placebo</td>
<td>34.9% vs. 49.2%</td>
<td>19.5% vs. 40.3%</td>
</tr>
</tbody>
</table>

Corticosteroids

- Often utilized despite mixed results in trials
- Prednisone, when used with acyclovir, in one study reduced pain associated with herpes zoster
- Corticosteroids are currently recommended for individuals over 50 years of age with HZ
- Dosage:
  - 30 mg bid x 7 days; 15 mg bid x 7 days; 7.5 mg bid x 7 days

Pain

• Pain associated with herpes zoster can range from mild – severe

• Clinician must tailor pain medication options based upon individual presentation
Pain Management

• Topical Agents
  – Calamine lotion to lesions 2 – 3x/day
  – Betadine to lesions qd
  – Capsaicin cream once lesions crusted 3 – 5x/day
  – Topical lidocaine 5% patch for 12 hours at a time once lesions are crusted

Acute Pain Management

• Oral Agents
  – Acetaminophen
    • Has not been shown to be effective in trials
  – Ibuprofen or similar
    • Not likely to be effective with neuropathic pain

• Nerve Blocks
  – Have been shown to be effective for many individuals with severe pain in some trials; other trials - ineffective
And…the use of medications such as TCA’s, gabapentin, pregabalin, oxycodone and tramadol during the acute phase of HZ decrease pain but also may also reduce the risk of PHN.
Follow-up

- Monitor for secondary infections
- Monitor for evidence of postherpetic neuralgia
- Monitor for adverse impact on quality of life
Paronychia
Paronychia

• Definition: A common nail infection of eponychium associated with a collection of pus between the eponychium and the nail root or lateral nail fold.

• Epidemiology and Etiology
  – 1 out of 10 people will have during a lifetime
  – Caused by a break in skin or injury to the cuticle
  – Symptoms include: redness, swelling, pressure and significant pain
  – Signs: pus pocket may be visible

http://www.emedicine.com/derm/TOPIC798.HTM
Paronychia

• Indications
  – If small with mild pain and no pustule, can frequently be managed by hot soaks and bacitracin
  – If much pain, edema, or pustule, drainage of lesion is treatment of choice

http://www.emedicine.com/derm/TOPIC798.HTM
Paronychia

• Anatomy Overview

Anatomy of the distal finger and nail components.
Paronychia

• Procedure
  – Soak digit
  – Insert #11 blade or 18g needle between eponychium and nail plate
  – Gently sweep to separate surfaces and drain pustule
  – Gentle massage
  – Soak digit again
  – Bacitracin
Technique for draining simple paronychia. Note that the #11 blade is brought between the nail and the eponychium parallel to the nail plate. This simple maneuver will drain the vast majority of paronychiae.
Paronychia

• Follow-up Care
  – Soak digit
  – Replace bacitracin and bandage
  – Oral antibiotics are usually unnecessary

• Red Flags
  – Pain, swelling or erythema concentrated on the palmar surface (This is a felon NOT a paronychia)
Psoriasis
Psoriasis

• Etiology
  – 1-3% of the population worldwide
  – Transmitted genetically
  – Disease is lifelong; often beginning in childhood
  – Characterized by chronic, recurrent exacerbations and remissions
  – Stress can precipitate an episode
  – Strep pharyngitis has been known to precipitate the onset

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

• Etiology
  – Physically and emotionally disabling
  – Erodes self esteem and often forces the patient into a life of concealment
  – Medications can precipitate (Beta blockers, lithium)

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

- Symptoms
  - Red, scaling papules that coalesce to form round-oval plaques
  - Scale is silvery white and is adherent
  - When removed, bleeding occurs (Auspitz’s sign)
  - May begin at a site of a sunburn or surgery
    - This is called Koebner’s phenomenon
  - Elbows, knees, scalp, gluteal cleft, toenails, fingernails
    - Extensor surfaces

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis
Guttate Psoriasis
Psoriasis

• Diagnosis
  – History and physical examination
  – Biopsy if uncertain

• Plan
  – Diagnostic: None

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

- Therapeutic
  - Topical corticosteroids
    - Pulse therapy
    - Two weeks on/ two weeks off
    - Caution: side effects
  - Dovonex
    - Vitamin D3 analogue
    - Works by inhibiting epidermal cell proliferation
    - Can be used long-term and is very safe
    - Dovonex ointment two times daily x 8 weeks
    - May see about a 70% improvement

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

- Therapeutic
  - Tar: newer preparations are more pleasant
  - Intralelional steroids

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

- Therapeutic
  - Ultraviolet light B
  - Retinoids
- Systemic Treatments
  - Methotrexate
  - Plaquenil
  - Enbrel

http://www.emedicine.com/derm/TOPIC365.HTM
Psoriasis

• Plan
  – Educational
    • Moisturize
    • Consider psychological therapy
    • Review the nature of this chronic disease

http://www.emedicine.com/derm/TOPIC365.HTM
Acne
Acne Vulgaris

• Etiology
  – Disease involving the pilosebaceous unit
  – Most frequent and intense where sebaceous glands are the largest
  – Acne begins when sebum production increases
  – Propionibacterium acnes proliferates in the sebum
  – P. acnes is a normal skin resident but can cause significant inflammatory lesions when trapped in skin

http://www.emedicine.com/derm/TOPIC2.HTM
Acne Vulgaris

- Noninflammatory lesions
  - Open and closed comedones
- Inflammatory lesions
  - Papules, pustules and nodules (cysts)

http://www.emedicine.com/derm/TOPIC2.HTM
Acne Vulgaris

• Symptoms
  – Papular lesions on the face, chest and back
  – White heads
  – Black heads

• Signs
  – Papular lesions
  – Closed and open comedones

http://www.emedicine.com/derm/TOPIC2.HTM
Closed Comedones
Closed Comedones
Open Comedones
Cystic Acne
Acne Vulgaris

• Diagnosis
  – History and physical examination

• Plan
  – Diagnostic: None
Acne Vulgaris

- **Therapeutic**
  - **Benzyl Peroxide (2.5%, 5% and 10%)**
    - Effective as initial medication
    - Begin early on in the disease process
  - **Tretinoin**
    - Very effective agent
    - Start with 0.05% - 0.1% cream
    - Reduces and minimizes scarring
  - **Topical Antibiotics**
    - Initial medication or can be combined with benzyl peroxide
    - Erygel, clindamycin are most commonly utilized

http://www.emedicine.com/derm/TOPIC2.HTM
Acne Vulgaris

• Therapeutic
  – Oral Antibiotics
    • Tetracycline
    • Minocycline
    • Erythromycin
    • Cephalosporins
    • Should only be used when topicals are ineffective or when patient has moderate disease at presentation
  – OCP’s
    • Women desiring contraception who also have acne
  – Accutane
    • Cystic acne or mod-severe disease

http://www.emedicine.com/derm/TOPIC2.HTM
Acne Vulgaris

• Plan
  – Educational
    • 6 weeks for improvement to be seen
    • Avoid antibacterial soaps
    • Dove soap or similar is recommended
    • Avoid hats
    • Foods have not been implicated as a cause
    • Caramel products may worsen situation
    • Avoid picking at the lesions
    • Review side effects of the medications

http://www.emedicine.com/derm/TOPIC2.HTM
Rosacea
Rosacea

- Very common dermatological condition characterized by:
  - Facial flushing
  - Erythema
  - Telangiectasias
  - Coarseness of skin
  - Inflammatory papulopustular eruption resembling acne

Treatments

• Avoid possible triggers:
  – Hot or cold temperatures, wind, hot drinks, caffeine, exercise, spicy food, alcohol, emotions, topical products

• Sunscreen
  – Daily use of broad spectrum sunscreen is recommended for all patients
    • Ultraviolet A & B protection

Treatments

• Laser
  – Nonablative laser effective against rosacea by remodeling of the dermal connective tissue
  – Improving the dermal layer
    • Expensive
    • Not covered by insurance
    • Mainstay of rosacea therapy

Treatments

• Medications
  – Acne products
    • Benzyl peroxide, azelaic acid, sodium sulacetamide and sulfur
  – Immunosuppressants
    • Tacrolimus
  – Antibiotics
    • Azithromycin, metronidazole, erythromycin, fusidic acid (ocular rosacea), clindamycin, tetracycline, minocycline, doxycyline, clarithromycin

Treatment

– Retinoids
  • Tretinoin, isotretinoin

– Prognosis:
  • Most patients receive a stable state with variable residual symptomatology
  • Disease takes a chronic relapsing state in some patients

Bite Wound
Human Bite Wounds

• Three general types:
  – Closed fist injury
  – Chomping injury to the finger
  – Puncture-type wound about the head caused by clashing with a tooth

• General principles of contaminated wound management apply to human bite wounds

Types of Human Bites

• Closed fist injury
  – Often inoculate the extensor tendon and sheath
  – Bacterial load is high with flexed fist
  – Contamination not removed easily through normal cleaning

• Finger
  – Chomping type injury – watch for tendon and overlying sheaths – careful inspection needed

• Tooth striking injury
  – Deep puncture would may appear innocuous
  – Deep, bacterial contamination is possible

Infected Human Bite Wound

• Infectious Disease Society of America recommendations:
  – Amoxicillin/clavulanate or ampicillin/sulbactam
  – TMP/SMX plus clindamycin is acceptable alternative in penicillin allergic patients

• Update Td/Tdap status
Burns
First Degree Burn

Only the epidermis

- Tissue blanches with pressure
- Tissue is erythematous and often painful
- Tissue damage is minimal
- Typical = sunburn

Usual symptomatic care

1st Degree Burns

- ASA or NSAID’s
- Fluid replacement
- Topical care
  - Cold soaks with water or aluminum acetate solution

Second Degree Burn

Immediate Emergency Care

Partial thickness burn

• Epidermis and portions of the dermis are involved
• Adnexal structures
  – Sweat glands, hair follicles
• Enough of the structure is preserved for function
• Blistered and very painful
• If deep 2\textsuperscript{nd} degree, edema, deceased blood flow can result in conversion to full thickness

2nd Degree Burns, Initial Treatment

- Remove charred clothing
- Cool tissues with saline or clean water
- Once the burn has been cooled, place the patient in dry, sterile sheets
- Maintain adequate hydration
- Prevent infection
  - Do not rupture blisters
  - For ruptured blisters, apply bactroban or silvadene cream

Third Degree Burn

Full Thickness Burn

- Charring skin
- Translucent white color
- Coagulated vessels
- No pain, but pain in surrounding 1\textsuperscript{st} and 2\textsuperscript{nd} burns
- All tissue and structures destroyed

Immediate Emergency Care

Malignant Melanoma
Malignant Melanoma

- Very dangerous cancer that arises from the cells of the melanocytic system
- Can metastasize to any organ including the brain
- Epidemic proportions - Lifetime risk: 1:90
- Risk factors
  - Sun exposure
  - Family history of melanoma
  - Immunosuppression

Malignant Melanoma

- Asymmetry
- Borders
- Color
- Diameter enlargement
- Enlarging or evolving

Malignant Melanoma

• Characteristics
  – Can be black, brown, red, white or blue

• Types
  – Superficial spreading
  – Lentigo maligna
  – Nodular melanoma
  – Acrallentiginous melanoma

Malignant Melanoma
Malignant Melanoma
Malignant Melanoma

• Treatment
  – Biopsy with elliptical excision only
  – Shave excision and punch biopsy are NOT recommended
  – Referral to dermatology/general surgeon/plastics depending upon access
  – Surgical excision with margin clearing
    • 1-2 cm margin
    • Recent evidence that a 3 cm margin may improve survival rates

Insect Bite Sting
Insect Bites

• Usually a minor nuisance
• Bites from Hymenoptera species (bees, wasps, yellow jackets, ants) can be severe and result in anaphylaxis
• Usual presentation:
  – Pruritis
  – Erythematous papules
  – Excoriation is common from scratching
  – Vesicular and bullous reactions are not uncommon

Insect Bites

- Arthropods commonly serve as vectors to spread disease
  - Viral encephalitis – mosquito
    • Many parts of the US
  - Lyme Disease – ticks
    • Northeastern US 90% ticks carry Lyme disease spirochete
  - West Nile Virus, Dengue fever - mosquito
    • Now found in parts of US
  - Rocky Mountain Spotted Fever – ticks
    • East Coast US – North Carolina

Brown Recluse Spider

• Toxins introduced by this spider can cause significant endothelial tissue damage – tissue necrosis

Presentation

• Commonly appears as:
  – Pruritic papules
  – Grouped in area where the bite occurs
  – Vesicular and bullous bite reactions are common
  – Intense pruritis and a distribution in exposed areas suggest the diagnosis of a bullous bite

Management

• Typically managed with topical antipruritics
  – Camphor and menthol
• Topical corticosteroids
• Antibiotics: Lyme disease
• Be mindful of the brown recluse spider and educate patient to come in if changes or gets worse
  – Ice initially
  • May need surgical debridement

Scabies
Scabies

• Etiology
  – Contagious disease caused by a mite
  – Common amongst school children
  – Adult mite is 1/3 mm long
  – Front two pairs of legs bear claw-shaped suckers
Scabies

• Etiology
  – Infestation begins when a female mite arrives on the skin surface
  – Within an hour, it burrows into the stratum corneum
    • Lives for 30 days
    • Eggs are laid at the rate of 2-3 each day
    • Fecal pellets are deposited in the burrow behind the advancing female mite
    • (Scybala)-feces are dark oval masses that are irritating and often responsible for itching
Scabies

• Etiology
  – Transmitted by direct skin contact with infested person either through clothing or bed linen
  – Eruption generally begins within 4 – 6 weeks after initial contact
  – Can live for days in home after leaving skin
Scabies

• Symptoms
  – Minor itching at first which progresses
  – Itching is worse at night (this is characteristic of scabies)

• Signs
  – Erythematous papules and vesicles
  – Often on the hands, wrists, extensor surfaces of the elbows and knees, buttocks
  – Burrows are often present; May see a black dot at the end of the burrow
  – Infants: wide spread involvement
Scabies
Scabies
Scabies

• Diagnosis
  – Scraping to look for mite, eggs or feces

• Plan
  – Diagnostic: Scraping
  – Therapeutic
    • Permethrin 5% cream
Scabies

• Plan
  – Therapeutic
    • Sulfur (6% in petroleum or cold cream qd x 3 days)
    • Antihistamine
  – Educational
    • Cut nails short
    • Scratching spreads the mites
    • Itching can last for weeks
    • Treat all family members
Scabies

• Plan
  – Educational
    • Wash all clothing, towels and bed linen
    • Do not need to wash carpeting
    • Consider animal bathing
    • Bag stuffed animals x 1-2 weeks
Lyme Disease
Lyme Disease

• A SYSTEMIC infection caused by *Borrelia burgdorferi*

• Introduced into the skin by a tick bite

Lyme Disease - Stages

- Stage 1: early localized – undifferentiated febrile illness
- Stage 2: Early disseminated disease
  - Extracutaneous manifestations

Stages

- Stage 3: late Lyme disease refers to rheumatologic and neurologic manifestations
- Months to years after initial infection

Diagnosis

• History extremely important
  – Work, live, vacation
  – May-September highest months
  – Many individuals have no recollection of tick bite

• Systemic:
  – Low grade fever
  – Flu-like illness
  – Fatigue, headache, myalgias, arthralgias may be early in the presentation

Labs and Treatment

- Laboratory testing
  - Western Blot & ELISA

- Treatment
  - Antibiotics
    - Amoxicillin 500 mg tid x 21 days – 28 days
    - Doxycycline 100 mg 1 two times daily x 21 – 28 days
    - Cefuroxime
    - Erythromycin
    - Azithromycin

Mumps
Mumps

- Systemic illness
- Nationally reportable
- Spread by respiratory droplets
- Incubation period 14-25 days
- Symptoms last from 3-5 days
- Parotitis occurs in 30-40% of patients

Presentation

• Prodromal phase
  – Nonspecific viral symptoms, low grade fever, malaise, myalgias and headache
• Considered infectious from about 3 days before the onset and up to 4 days into active parotitis
• Age of onset is changing:
  – Resurgence in the late 1980’s
  – 30-40% cases in persons older than 15 years

Treatment

- Mandatory report to Department of Public Health
- Supportive care
- Complications – males
  - Orchitis
  - Ice packs to scrotal area
  - Scrotal support and NSAID’s

MUSCULOSKELETAL JOINT AND EXTREMITY INJURY
Ankle Injury
Soft Tissue Injuries

• Frequent sports injuries
  – Basketball, soccer, volleyball, activities on uneven surfaces
• Inversion of ankle during extension of ankle – 3 specific ligaments
  – Anterior talofibular ligament (65% inversion)
  – Calcaneofibular ligament
  – Posterior talofabular ligament

Types of Sprains

• Grade 1
  – Stretch, microscopic tearing only
  – Minimal swelling or reduction in function
  – Full weight bearing

• Grade 2
  – Stretch, partial tearing
  – Moderate to severe swelling and ecchymosis
  – Moderate functional loss, instability
  – Unable to weight bear

Types of Sprains

• Grade 3
  – Complete rupture of the ligament
  – Immediate and severe swelling
  – Ecchymosis with inability to bear weight
  – Moderate to severe instability of joint

• Chronicity
  – Up to 50% of individuals with ankle sprains experience some type of chronic sequelae.
    • Functional instability, mechanical instability, chronic pain, stiffness or chronic swelling

Treatment of Ankle Sprains

- Grade I: ice, elevation, NSAIDs, ankle brace, weight bearing may begin immediately. D/C brace in 1 month
- Grade II: ice, elevation, NSAIDs, ankle brace, no weight bearing x 7 days
- Grade III: walking cast x 3 – 4 weeks, PT, ankle brace

Knee Injury
Assessment

- Swelling
- Ecchymosis
- Diminished range of motion
- Joint instability
- Diminished weight bearing ability
- Consider imaging
  - X-rays – fractures, effusions
  - MRI – ligamentous or meniscus injuries

Treatment

- **Grade 1 Sprain**
  - Rest, ice, elevation, NSAID’s
  - Range of motion exercises
  - Compression dressing or stirrup splint
  - Strengthening with PT

- **Grade 2 & 3 Sprains**
  - Ice, elevation, transportation for further evaluation (sports medicine, orthopedic) and imaging

ACL injuries

• Anterior Cruciate Ligament injuries
  – Low velocity
  – Non-contact
  – Deceleration

• Contact or collision injury ACL
  – Contact injuries with a rotational component
  – Twisting
  – Valgus stress
  – Hyperextension

Presentation

• Non Contact injury
  – Audible pop during change of direction, cutting or landing from a jump (hyperextension/pivot combination)
  – Large hemarthrosis – few hours
  – Inability to play secondary to pain, swelling or instability of knee

• Contact or high energy trauma
  – Terrible Triad (ACL, MCL, medial meniscus)
  – Valgus stress to the knee

Presentation

- Gross effusion or bony abnormality
- Immediate effusion correlates up to 72% of time to an ACL injury of some degree
- Lack of complete extension
- Ligamentous laxity difficult to detect in acute presentation

Reconstruction

- Stability in running, cutting, kicking
- ACL deficit knee has been linked to
  - Increased risk of degenerative changes
  - Increased meniscal injuries
  - Reconstruction effective in restoring stability in 75-95% of cases
  - 8% failure due to continued instability, graft rejection or arthrofibrosis

Elbow injury
Elbow Injuries

- Overuse injuries common in athletes
  - Elbow capsule
  - Olecranon
- Repetitive flexion-extension or wrist motion
  - Throwing and racket sports
- Lateral and medial epicondylitis
  - Overuse injuries

Disabella VN. Elbow and Forearm Overuse Injuries E Medicine. 2-12-08. accessed 7-30-08 http://www.emedicine.com/sports/TOPIC30.HTM
Functional Anatomy

• 3 true joints in the elbow
  – Humeroulnar – modified hinge joint
    • Flexion and extension
  – Humeroradial – hinge joint
    • Flexion and extension
    • Pivot allowing rotation of the radial head
  – Radioulnar
    • Supination and pronation

Disabella VN. Elbow and Forearm Overuse Injuries E Medicine. 2-12-08. accessed 7-30-08 http://www.emedicine.com/sports/TOPIC30.HTM
Functional Anatomy

• Ligamentous structures
  – Lateral and medial ligaments
  – Thickening of the capsule (rather than true ligaments)
  – Anterior medial collateral ligament is the most important
    • 70% of the valgus stability of the elbow medially
    • Laterally – ulnar collateral ligament is the strongest

Disabella VN. Elbow and Forearm Overuse Injuries E Medicine. 2-12-08. accessed 7-30-08 http://www.emedicine.com/sports/TOPIC30.HTM
Overuse Injuries

• Repetitive elbow flexion can result in:
  – Biceps tendinitis
  – Anterior capsule strain

• Forceful elbow extension
  – Triceps tendinitis
  – Posterior impingement syndrome

• Increased valgus stress
  – Ulnar nerve injury
  – Posterior impingement syndrome
  – Olecranon stress fractures

Disabella VN. Elbow and Forearm Overuse Injuries E Medicine. 2-12-08. accessed 7-30-08 http://www.emedicine.com/sports/TOPIX30.HTM
Tennis Elbow – Lateral Epicondylitis

• Excessive wrist extension
• Maximal tenderness - area distal to the origin of the extensor muscle of the forearm at the lateral epicondyle
• Reproduction of pain at site reasonably diagnostic
• Interventions:
  – Watchful waiting and modification of activities
  – NSAID’s, tennis elbow strap
  – Corticosteroid injections

Walrod, RJ. Lateral Epicondylitis E Medicine. 5-28-08. accessed 7-30-08  
http://www.emedicine.com/sports/topic59.htm
Rehabilitation Programs

• Very useful in overuse injuries
  – Protection
    • Modify activity to prevent further injury
  – Rest
    • Modified activity; not deconditioning
  – Ice
    • Pain, decrease venous stasis at injury site
  – Compression
    • Prevent swelling (not with nerve involvement)
  – Elevation
    • Reduction of swelling
  – NSAID’s

Disabella VN. Elbow and Forearm Overuse Injuries E Medicine. 2-12-08. accessed 7-30-08 [http://www.emedicine.com/sports/TOPIC30.HTM](http://www.emedicine.com/sports/TOPIC30.HTM)
Gout
Gout

- Common disorder
- Uric acid overproduction or underexcretion of urate crystals
  - Joint inflammation
  - Tissue deposition of uric acid crystals
  - Joint destruction, if left untreated
- Definitive Diagnosis (rarely done)
  - Joint aspiration and synovial fluid analysis

Diagnosis

• Serum uric acid
  – 5-8% of population has elevated serum uric acid levels (>7mg/dL)
  – Only 5-20% of patients with hyperuricemia develop gout
  – Official diagnosis
    • Urate crystals in synovial fluid

Presentation

• 1-2 joints involved at first
• Inflammation of the first metatarsophalangeal joint in 50% initial cases; 90% of cases eventually
• Attack begins abruptly
• Maximal intensity 8-12 hours
• Joint
  – Red, hot, exquisitely tender – bed sheets can be very uncomfortable

Pseudogout

- Pseudogout is inflammation caused by calcium pyrophosphate crystals
  - Many cases idiopathic
  - Associated with aging, trauma, hyperparathyroidism and hemochromatosis
- May be clinically indistinguishable from gout
- Treatment of the acute phase of pseudogout is the same as gout
- No prophylactic therapy exists

Pseudogout

• Most common locations
  – Knee, wrist, shoulder
  – Doesn’t involve the 1st MTP as with gout

• Presentation
  – Gout – rapid onset
  – Pseudogout - insidious presentation
  – Otherwise, clinically can not distinguish
Treatment

• Treat the acute attack
  - NSAID’s, corticosteroids, colchicine

• Prophylaxis to prevent acute flares
  - Lowering uric acid
    • Allopurinol

• Lowering excess stores of uric acid
  - Prevent flares of gouty arthritis
  - Prevent tissue deposition of uric acid crystals

Hand Injuries
Hand Injuries

• Most common mechanism is trauma
  – Crush injury, contusions, abrasions, lacerations, avulsion, ring avulsion, burns

• Nerve Injuries
  – Blunt penetrating and crush injuries
    • Bruised but intact
    • Axonal core of the nerve is damaged but the myelin sheath intact
      – Regenerate at 1-3 mm per day
    • Complete disruption
      – Surgical reapproximation is necessary

Hand Injury

• Sprains
  – Stretching or partial tearing of ligaments
  – Classified as 1\textsuperscript{st}, 2\textsuperscript{nd}, 3\textsuperscript{rd} degree

• Dislocations
  – DIP, PIP, MCP, 1\textsuperscript{st} digit

• Ligamentous injuries
  – Often misdiagnosed as sprains
  – Serious impact of missed ligamentous injuries
    • Chronic pain, unstable or chronically deformed joints

Hand Injury

• Tendon injuries
  – Extensor tendon’s location (superficial) predisposes injury from avulsions, crushes, burns
  – Forced hyperflexion or forced flexion of digit

• Fractures
  – May not be obvious; get X-rays, if any doubt

Hand Injury

• Due to complexity of hand and often non-distinguishable types of injury, referral to a hand specialist should be strongly considered.
Wrist Pain
Wrist Pain

• Fracture
  – Scaphoid is most frequently fractured carpal bone – 71% of all carpal fractures
  – 90% of acute scaphoid fractures heal completely, if treated early
Wrist Fracture

• Issues with untreated or misdiagnosed scaphoid fractures
  – Malunion
  – Delayed union
  – Nonunion
  – Avascular necrosis
  – Osteonecrosis (more than any other bone due to circulation to this bone)

Acute Low Back Pain

Spinal Column with Vertebrae

- Cervical Vertebrae (7) C₁ - C₇
- Thoracic Vertebrae (12) T₁ - T₁₂
- Lumbar Vertebrae (5) L₁ - L₅
- Sacrum (5 - fused)
- Coccyx (4 - fused)
Acute (Mechanical) Low Back Pain

• Low back pain occurs at least once in 85% of adults ≤ 50 years of age

• Most common etiologies
  – Age related degenerative disks
  – Age related facet processes
  – Muscle or ligament related injuries
    • Nerve root syndromes
    • Musculoskeletal pain syndromes
    • Skeletal causes

Acute Back Pain

• Musculoskeletal Pain Syndromes
  – Myofascial pain syndromes
  – Tenderness over localized areas
  – Loss of range of motion in involved muscle groups

Acute Back Pain

• Classic nerve root syndrome
  – Radicular pain arising from nerve root impingement, inflammation or irritation due to herniated disks
  – Clinical
    • Impingement Pain: Sharp, well localized pain associated with paresthesias
    • Irritation Pain: dull, poorly localized, and without paresthesias; no radiation, - SLR
    • Impingement = + straight leg raising; neurological deficits, pain radiating below knee

Treatment of Acute Back Pain

• Early mobilization and gentle range of motion with strengthening exercises – nonsciatic back pain
• Anti-inflammatory medications
• Muscle relaxants may be helpful
• Gentle flexion/extension exercises are helpful
• Consider physical therapy

Orthopedic Emergencies

Cauda Equina
Cauda Equina

• Collection of intradural nerve roots located at the termination of the spinal cord
  – Sensory innervation to the saddle area
  – Motor innervation to the sphincters
  – Parasympathetic innervation – bladder and lower bowel

• Cauda equina syndrome is caused by loss of function of 2 or more of the 18 nerve roots that constitute the CE

Presentation
Cauda Equina

• Complex of symptoms
  – Lower back pain
  – Unilateral or bilateral sciatica
  – Saddle sensory disturbances
  – Variable lower extremity motor/sensory loss
  – Loss of control of bladder or bowel
  – Erectile dysfunction

• Onset
  – May be acute or chronic

Causes

- Disc herniation
- Intradural disc rupture
- Spinal stenosis (multiple etiologies)
- Traumatic injury
- Primary tumors
- Metastatic tumors
- Infectious conditions
- AV malformations
- Hemorrhage
- Iatrogenic causes

Clinical Presentation of Cauda Equina

• Radicular pain with radicular sensory loss (saddle anesthesia)
• Asymmetric paraplegia with loss of deep tendon reflexes
• Muscle atrophy
• Loss of bowel or bladder dysfunction
Diagnosis of Cauda Equina

- Plain radiographs should be obtained to search for destructive changes, disc-space narrowing, or loss of spinal alignment.
- A CT scan provides additional details about bone density and integrity.
- MRI delineates the soft tissues, including neuronal structures and the offending pathologic condition.
- Radionuclide scanning if consideration is given to osteomyelitis or infection.

Treatment of Cauda Equina

Therapy is directed at the cause

• Considered a Neurologic Emergency
  – Immediate neurosurgery evaluation

• Medical Therapy
  – Infections - antibiotics
  – Tumors, lesions - chemotherapy

• Surgical Therapy
  – Relief of pressure from offending component

NEUROLOGY
Bell’s Palsy
Bell’s Palsy

- Edema and ischemia resulting in compression of the facial nerve
  - Affects muscles of facial expression
  - All muscles of the facial nerve are derived from the second brachial arch
  - Injury to the 7th cranial nerve (Facial)

Monnell, K et al. Bell’s Palsy. E Medicine. 11-14-08. accessed 7-30-08 [http://www.emedicine.com/neuro/TOPIC413.HTM](http://www.emedicine.com/neuro/TOPIC413.HTM)
Causes

- Cause of the edema and ischemia continues to be uncertain and debated
- Many sources think that HSV is most likely cause
  - Consider HerpeSelect test
- 1/3 of all cases of Bell’s Palsy is caused by Lyme disease
  - Must check Lyme Western Blot

Monnell, K et al. Bell’s Palsy. E Medicine. 11-14-08. accessed 7-30-08 [http://www.emedicine.com/neuro/TOPIC413.HTM](http://www.emedicine.com/neuro/TOPIC413.HTM)
Symptoms

• Acute onset of unilateral (mostly) upper and lower facial paralysis
  – Occurs over a 48 hour period
  – Must include forehead and lower aspect of face
  – Mostly (68%) right side of face
  – Inability to close eye or smile on affected side

• Posterior auricular pain (50%)
• Decreased tearing (1/6th of patients)
• Taste disturbances

Monnell, K et al. Bell’s Palsy. E Medicine. 11-14-08. accessed 7-30-08 http://www.emedicine.com/neuro/TOPIC413.HTM
Treatment

- Corticosteroids
  1mg/kg or 60mg for 6 days
  – Controversial
- Acyclovir
  – Acyclovir 400mg 5 times per day X7 d
- Eye care
  – Prevent corneal dryness

Monnell, K et al. Bell’s Palsy. E Medicine. 11-14-08. accessed 7-30-08 [http://www.emedicine.com/neuro/TOPIC413.HTM](http://www.emedicine.com/neuro/TOPIC413.HTM)
Headaches

- Ophthalmic
- Maxillary
- Mandibular

Trigeminal nucleus caudalis
Dorsal horn for C2-C4
C2, C3
C3, C4, C5
Primary Headache
Versus Secondary Headache

Primary HA
- Migraine
- Tension-type HA (TTH)
- Cluster
- Rebound
- Other HA (eg, benign cough HA)

Secondary HA
- Brain tumor
- Hemorrhage
- Increased ICP
- Infection

Generally, >90% of patients have a primary headache disorder and <10% have a secondary headache disorder

Headache Classification Subcommittee of the International Headache Society. *Cephalalgia* 2004;24(suppl 1):24-43
IHS Criteria for Migraine
Episodic, recurrent HA lasting 4 to 72 hours

- At least 2 pain qualities
  - Unilateral
  - Throbbing
  - Moderate to severe intensity
  - Worsened by movement

plus

- At least 1 associated symptom
  - Nausea and/or vomiting
  - Photophobia and phonophobia

In a person with a normal history and physical exam

Headache Classification Subcommittee of the International Headache Society. *Cephalalgia* 2004;24(suppl 1):24-43
Nasal Symptoms and HA: Nasal Stuffiness Is Often a Sign of Migraine
IHS Criteria for Tension-Type HA (TTH)

At least 10 episodes occurring on <1 day per month on average, with attacks lasting 30 minutes to 7 days

- At least 2 pain qualities
  - Bilateral
  - Pressing (nonpulsating)
  - Mild to moderate intensity
  - Not worsened by movement

- Both of the following
  - Absence of nausea and vomiting
  - Either photophobia or phonophobia

And not attributable to another disorder

Headache Classification Subcommittee of the International Headache Society. *Cephalalgia* 2004;24(suppl 1):24-43
Acute Migraine Management

Evidence-Based Guidelines

- Adopted by AAFP, ACP-ASIM, AAN
  - NSAIDs as first-line therapy
  - Triptans (or dihydroergotamine) indicated for those who fail to tolerate or respond to NSAIDs
  - No evidence to support the use of butalbital compounds in acute migraine
  - Little evidence to support the use of isomethoephtene compounds in migraine
  - Opioids “reserved for use when other medications cannot be used”

Acute/Abortive Therapy of Migraine

$5-HT_{1B/1D}$ Agonists: The Triptans

- Almotriptan
- Eletriptan
- Frovatriptan
- Naratriptan
- Rizatriptan
  - Tablet
  - Orally disintegrating tablet

- Sumatriptan
  - Injectable
  - Nasal spray
  - Tablet
  - Sumatriptan & Naproxen

- Zolmitriptan
  - Tablet
  - ODT
  - Nasal spray
Headaches - Secondary
Red Flags for Secondary HA

1. Detailed Patient History and Examination
   - RED FLAG
   - YES
   - Secondary HA
     - Diagnostic Testing
     - Atypical Features
   - NO
   - Primary HA
Addressing Secondary Headache
Red Flags: “SNOOP” Assessment

**S**  Systemic symptoms/signs or systemic disease

**N**  Neurologic symptoms/signs

**O**  Onset that is sudden

**O**  Onset after age 50 or under 5 years of age

**P**  Pattern change

When in doubt, investigate the atypical

CVA
Broad Based Term

- Sudden loss of circulation to an area of the brain, resulting in compromised neurological function
  - Ischemic (thromboembolism = 85%)
  - Hemorrhagic
    - 2nd leading cause of death worldwide in 1990

Etiologies

• Cardiogenic
  – Valvular emboli, mural emboli, endocarditis, prosthetic valves, MI (2-3%), Atrial fibrillation

• Less common etiologies
  – Polycythemia, sickle cell anemia, Protein C deficiency, fibromuscular dysplasia of the cerebral arteries, prolonged vasoconstriction from migraine headaches, trauma, pelvic surgery, orthopedic surgery

Presentation

• Symptoms of ischemic vs hemorrhagic difficult to distinguish
  – Nausea, vomiting, headache, change in level of consciousness are more common in hemorrhagic stroke
  – Acute, neurologic deficit– focal or global
  – Altered level of consciousness

CVA

• Stroke considered with abrupt onset of
  – Hemiparesis
  – Monoparesis
  – Quadriparesis
  – Monocular binocular visual loss
  – Visual field deficits
  – Diplopia
  – Dysarthria
  – Ataxia or vertigo

Conditions Which Can Mimic a CVA

• Seizure - 17%
• Systemic infection - 17%
• Brain tumor - 15%
• Toxic metabolic cause
  – i.e. hyponatremia - 13%
• Positional vertigo - 6%

Treatment

• Urgent presentation
  – Possible thrombolysis
  – Stabilization
  – Identification of etiology of incident

• Immediate referral to an Emergency Department
  – Minutes can make difference in overall outcome

Syncope
Syncope

• Syncope is the result of “self-terminating inadequacy of global cerebral nutrient perfusion”
  – Most often the result of transient systemic hypotension
  – Can be acute global cerebral oxygen deprivation (high altitude)

Syncope

• Syncope vs. non-syncope
  – Key – total loss of consciousness (TLOC)
  – Then, differentiate between syncopal and non-syncopal causes of the TLOC

• Only circumstances in which TLOC can reasonably be attributed to transient cerebral hypoperfusion should be considered syncopal

Probable Causes of Syncope

• Neurally-Mediated
  – Vasovagal
    • Nauseated and sweaty before fainting
    • May feel pain before and appear clammy
  – Carotid sinus syndrome
  – Situational syncope (blood draws)

• Orthostatic
  – Movement associated
    • Lightheadedness, near syncope vs. TLOC

Probable Causes of Syncope

- Cardiac arrhythmias
- Structural cardiopulmonary disease
  - Acute MI
  - Ischemic event
  - Aortic stenosis
- Cerebrovascular Disease
  - Rarely cause of syncope
Hypovolemia vs Dysautonoma

- Elderly patients with ACE/ARB + diuretic are commonly pre-syncopal or syncopal
- Differentiate true hypovolemia from dysautonoma in elderly
- True orthostatic testing
  - Position change for 1 minute
  - Evaluate both blood pressure and pulse
Abnormal Orthostatic Testing

**Hypovolemia**
- Decrease in blood pressure by 20 mm Hg in any positional change
- Increase in pulse with decrease in blood pressure
  - 146/76 67
  - 138/70 76
  - 120/68 86

**Dysautonauamia**
- Decrease in blood pressure by 20 mm Hg in any positional change
- No change in heart rate
  - 146/76 67
  - 138/70 66
  - 120/68 67
Evaluation

• History most important
  – Define and clarify TLOC

• Establish cause to syncope
  – Work-up varies based upon proposed etiology

• Treat accordingly

PSYCHIATRIC
Depression
Depression

- Leading cause of disability worldwide
- Most common psychiatric diagnosis
- Less than 1/3 of adults with depression will obtain appropriate professional treatment
- Epidemiology
  - 121 million worldwide
  - Major depression 8-16% population worldwide
    - Females 10-25%
    - Males 5%-12%

Medical Disorders

• Depression often accompanies other concomitant medical disorders

• If presents as late onset (> 45 years of age) in the absence of situational or personal history of depression, look for underlying medical conditions

# Medical Disorders and Depression

## Disease Category Specific Issues

<table>
<thead>
<tr>
<th>Disease Category</th>
<th>Specific Issues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral Illnesses</td>
<td>Mononucleosis, HIV</td>
</tr>
<tr>
<td>Malignancies</td>
<td>GI, pancreatic</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Thyroid, diabetic, adrenal dysfunction</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Anemia (decreased B12, folate)</td>
</tr>
<tr>
<td>Cerebrovascular</td>
<td>S/P CVA</td>
</tr>
<tr>
<td>Collagen-vascular</td>
<td>SLE, rheumatoid arthritis</td>
</tr>
<tr>
<td>Degenerative CNS</td>
<td>Parkinson's, Huntington's</td>
</tr>
<tr>
<td>Drugs/Toxins</td>
<td>Corticosteroids, antihypertensives</td>
</tr>
<tr>
<td>Sleep Disorders</td>
<td>Obstructive sleep apnea</td>
</tr>
</tbody>
</table>

Medications that Cause of Worsen Depression

- Beta blockers
- Calcium channel blockers
- Interferon
- Histamine-2 blockers
- Clonidine and other antihypertensives
- Procainamide
- Barbiturates
- Phenytoin
- Corticosteroids
- Narcotics
- Anabolic Steroids
DSM IV-TR Diagnostic Criteria for MDD

- 5 or more symptoms in the same 2-week period on most days (see next slide)
- 1 of these symptoms must include:
  - Depressed mood, lack of interest or pleasure in most activities (anhedonia)
Pneumonic SIG E CAPS for the Diagnosis of MDD

- Sleep (or Sex)
- Interest
- Guilt
- Energy
- Concentration
- Appetite
- Psychomotor
- Suicidal thoughts
Tools Available for the Primary Care Provider

- **Beck Depression Inventory, Primary Care (BDI-PC)** http://harcourtassessment.com/haiweb/cultures/en-us/productdetail.htm?pid=015-8018-370
- **Zung Depression Scale**
  http://www.neurotransmitter.net/depressionscales.html
- **Hamilton Rating Scale for Depression** (HAM-D) http://www.neurotransmitter.net/depressionscales.html
- **Hamilton Rating Scale for Anxiety** (HAM-A)
  http://www.anxietyhelp.org/information/hama.html
Treatment of Depression

Goals of Treatment:

• **Reduce/eliminate symptoms**
• **Restore function**
• **Prevent relapse and recurrence**

Drug Therapy
- SSRI/SNRIs
- TCAs

Psychotherapy
- Cognitive Behavioral
- Interpersonal
- Psychodynamic

Other Therapy
- ECT
- EMDR
- Photolight Therapy
# SSRIs

## Dosing and Time to Effect

<table>
<thead>
<tr>
<th></th>
<th>Citalopram (Celexa)</th>
<th>Escitalopram (Lexapro)</th>
<th>Fluoxetine (Prozac)</th>
<th>Paroxetine (Paxil)</th>
<th>Sertraline (Zoloft)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Start dose</strong>*</td>
<td>20 mg</td>
<td>10 mg</td>
<td>10-20 mg</td>
<td>20 mg</td>
<td>25-50 mg</td>
</tr>
<tr>
<td><strong>Max dose</strong></td>
<td>40 mg</td>
<td>20 mg</td>
<td>80 mg</td>
<td>50 mg</td>
<td>200 mg</td>
</tr>
<tr>
<td><strong>Time to effect</strong></td>
<td>4-6 wks</td>
<td>1-2 wks</td>
<td>4-6 wks</td>
<td>4-6 wks</td>
<td>4-6 wks</td>
</tr>
<tr>
<td><strong>Titration Increment</strong></td>
<td>1 week</td>
<td>1 week</td>
<td>3-4 weeks</td>
<td>1 week</td>
<td>1 week</td>
</tr>
</tbody>
</table>

*In clinical practice, based on patient symptoms, starting doses are sometimes lower than that recommended by the drug manufacturer.*
## Medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Classification</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline, nortriptyline, imipramine, desipramine</td>
<td>Tricyclic antidepressants</td>
<td>Block the reuptake of both serotonin and norepinephrine</td>
</tr>
<tr>
<td>Fluoxetine, paroxetine, fluvoxamine, sertaline, citalopram, escitalopram</td>
<td>Selective serotonin reuptake inhibitors</td>
<td>Relatively selective inhibition of reuptake of serotonin (through some effects on other neurotransmitters)</td>
</tr>
<tr>
<td>Bupropion</td>
<td>Norepinephrine and dopamine reuptake inhibitor</td>
<td>Inhibits the reuptake of norepinephrine and dopamine</td>
</tr>
<tr>
<td>Trazodone, nefazodone</td>
<td>Serotonin antagonist reuptake inhibitor</td>
<td>Mainly antagonize 5-HT2 receptors: nefazodone also modestly inhibits the reuptake of serotonin, no epinephrine, and dopamine</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>Noradrenergic and specific serotonergic agent</td>
<td>Antagonizes alpha 2 autoreceptors and heteroreceptors;</td>
</tr>
<tr>
<td>Venlafaxine, duloxetine</td>
<td>Serotonin/norepinephrine reuptake inhibitors</td>
<td>Inhibit the reuptake of serotonin and norepinephrine</td>
</tr>
</tbody>
</table>

Therapy for Depression

• When utilized alone, antidepressant therapy effectively resolves symptoms in 40% of the individuals.

• Medication and psychotherapy combined are effective in resolving the symptoms in 60% of the individuals.
# SSRI Side Effects

<table>
<thead>
<tr>
<th></th>
<th>Citalopram (Celexa)</th>
<th>Escitalopram (Lexapro)</th>
<th>Fluoxetine (Prozac)</th>
<th>Paroxetine (Paxil/CR)</th>
<th>Sertraline (Zoloft)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Headache</strong></td>
<td></td>
<td></td>
<td>+++</td>
<td>++</td>
<td>++++</td>
</tr>
<tr>
<td><strong>Insomnia</strong></td>
<td>++</td>
<td>++</td>
<td>+++</td>
<td>++++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Somnolence</strong></td>
<td>+++</td>
<td>+++</td>
<td>++</td>
<td>++++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Nervousness</strong></td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Anxiety</strong></td>
<td>+++</td>
<td>+++</td>
<td>++++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>↓ Libido</strong></td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+++</td>
<td>++++</td>
</tr>
<tr>
<td><strong>Fatigue</strong></td>
<td>+++</td>
<td>+++</td>
<td>++</td>
<td>++++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Constipation</strong></td>
<td></td>
<td></td>
<td>++</td>
<td>+++</td>
<td>++++</td>
</tr>
<tr>
<td><strong>↓ Appetite</strong></td>
<td></td>
<td></td>
<td></td>
<td>+++</td>
<td>+++</td>
</tr>
</tbody>
</table>
Suicide Potential
Assessing Suicidality

• Proactive screening essential in all environments
  – Strongest risk factors for suicidal behavior
    • History of previous suicide attempts
    • Presence of current severe depression
    • Presence or history of bipolar disorder
    • Schizophrenia
    • Active or recurrent substance abuse
    • Aggressive/impulsive personality traits
Full List of Risk Factors
(in addition to strongest risks)

- Victim of physical or sexual abuse
- Active medical illness, esp with incapacity or pain
- Hopeless, helpless feeling
- Strong sense of shame
- Agitated, severely anxious
- Confused, delirious
- Current severe insomnia
- Socially isolated, lacks supports or living alone
- Easy access to lethal means
- Recent major loss or personal crisis

Full List of Risk Factors
(in addition to strongest risks)

- Recent exposure to highly publicized suicide
  - Especially in adolescents
- Giving away possessions, stockpiling pills, preparing for death
- Well organized, detailed suicide plan
- Divorced, never married, widowed
- Unemployed
- White
- Male
- Age 15-25 or older than 65 years
This concludes Adult Refresher, part II
Please go on line and take the post test.

Thank you for participating in this update