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Overcoming Challenges in the Diagnosis and Management of Axial Spondyloarthritis: New Insights and Implications for Clinical Practice

Presented by:
Joerg Ermann, MD

Approved for 1.0 Prescribed CME

Saturday, July 31, 2021
8:00—9:00am

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
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NOTES

Overcoming Challenges in the Diagnosis and Management of Axial Spondyloarthritis

New Insights and Implications for Clinical Practice

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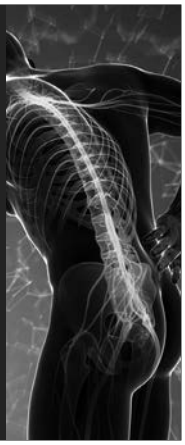
PeerView Live



Disclosures

Joerg Ermann, MD, has a financial interest/relationship or affiliation in the form of:
Consultant and/or Advisor for Novartis; Eli Lilly; Pfizer; and UCB.
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Housekeeping Notes

Thank you to Medical Learning Institute, Inc. and PVI, PeerView Institute for Medical Education for providing this session, and to Novartis Pharmaceuticals Corporation for providing the educational grant for this activity.

You should have received a link to an online program evaluation or a printed copy of the program evaluation.

Evaluation: PeerView.com/axSpA-Eval-WXC

Your evaluation of the program is very important in helping us to better meet your current and future medical education needs. We welcome your opinions and comments.

Please feel free to ask questions at the end of the presentation

Goals of Today's Discussion

- **Identify axSpA in patients with chronic back pain and spondyloarthritis features**
- **Identify the optimal approach towards early diagnosis of axSpA**
- **Recognize the importance of early referral of patients with suspected axSpA to rheumatology**
- **Discuss evidence-based management approaches to axSpA**

A Closer Look at Strategies to Improve the Timely Recognition of Axial Spondyloarthritis

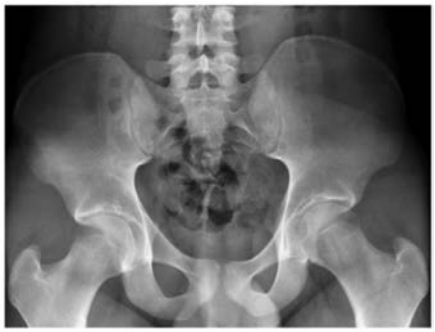
PeerView Live

Patient Case

44-year-old woman with chronic low back pain for 6-8 years

- Back pain is worse with prolonged rest and improves with exercise and activity
- Wakes up because of back pain; NSAIDs provide significant relief
- Needs to visit her podiatrist for recurrent plantar fasciitis
- Normal ROM in L spine, mild tenderness at insertion of plantar fascia on calcaneus; otherwise, normal joint examination
- Labs show normal ESR and CRP, but positive for *HLA-B27*

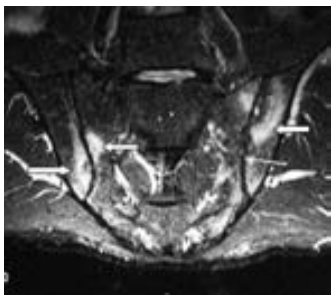
Sacroiliitis Grade 0 (Normal)[a]



[a] Image courtesy of Dr. Abhijeet Danve.

Patient Case[a] (Cont'd)

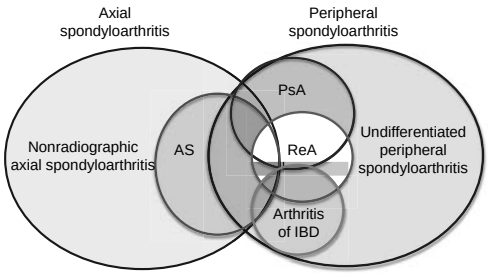
Patient referred to a rheumatologist who had high suspicion of spondyloarthritis[b]



[a] Image courtesy of Dr. Abhijeet Danve.
[b] MRI of pelvis confirmed sacroiliitis; subchondral marrow inflammation shown by increased MRI signal on fat suppressed T2 weighted image (STIR; as shown by white arrows) and joint cavity (as shown by yellow arrows).

What is the diagnosis?

Spectrum of Spondyloarthritis[1]



1. Raychaudhuri SP, Deodhar A. *J Autoimmun.* 2014;48-49:128-133.

Nomenclature[1]

- axSpA became the standard nomenclature in 2009 based on a multinational study by Assessment of Spondyloarthritis International Society (ASAS)[1,2]
- ASAS divided axSpA into two main subtypes
  - Radiographic (r-axSpA) = ankylosing spondylitis (AS)
  - Nonradiographic (nr-axSpA)
- Different, validated ASAS classification criteria are also available for patients with peripheral SpA[3]
  - Patients whose symptoms are not predominantly back pain but PsA, reactive arthritis, and arthritis-associated IBD

1. Rudwaleit M et al. *Ann Rheum Dis.* 2009;68:770-776. 2. Rudwaleit M et al. *Ann Rheum Dis.* 2009;68:777-783. 3. Rudwaleit M et al. *Ann Rheum Dis.* 2011;70:25-31.

What is axSpA?[1]

- Immune-mediated chronic inflammatory disease affecting the axial and peripheral skeleton, as well as extra-articular organs such as skin, gut, and eyes
- Mainly affects young individuals in their third or fourth decade
- Frequently leads to significant pain, loss of function, work disability, and impaired quality of life
- Heterogeneous presentation and progression

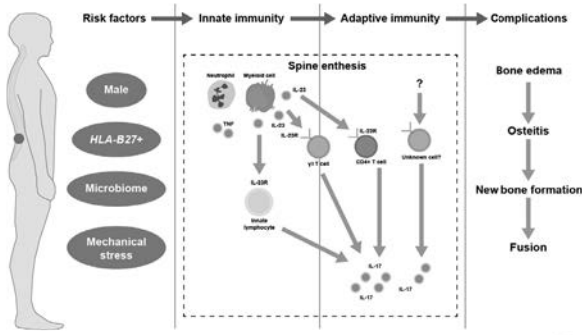
1. Danve A, Deodhar A. *Curr Rheumatol Rep.* 2017;19:22.

Typical Clinical Features of axSpA

Musculoskeletal	Nonmusculoskeletal
• IBP • Peripheral arthritis • Dactylitis • Enthesitis • Anterior chest wall pain	• Uveitis • Inflammatory bowel disease • Psoriasis

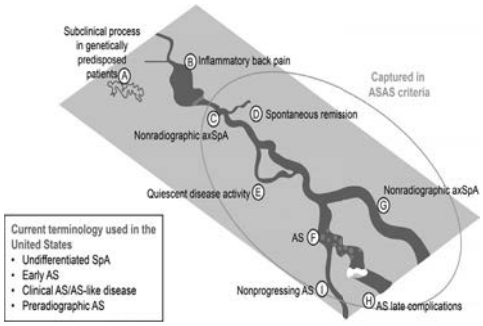
Other features specific to spondyloarthritis
- Strong association with *HLA-B27*
- Familial aggregation
- Negative RF

Pathogenesis of axSpA[1]



1. Watad A et al. *Front Immunol.* 2018;9:2668.

Natural History of axSpA: Newer Trends[1]



1. https://www.the-rheumatologist.org/article/rheumatologists-make-progress-defining-spectrum-of-axial-spondyloarthritis/3/.

Defining AS and nr-axSpA[1,2]

AS (r-axSpA)	nr-axSpA
• Unequivocal evidence of sacroiliitis on plain radiographs (x-rays) • Syndesmophyte formation may lead to vertebral fusion • Functional impairment worse	• No unequivocal radiographic evidence of sacroiliitis • Inflammation of sacroiliac joints may be detected by MRI • Severity of symptoms usually similar

- Emerging concept: axSpA is a single disease continuum with radiographic severity changing over time
- Radiographic sacroiliitis is now considered a late finding in the disease course.

1. van der Heijde D et al. *Ann Rheum Dis.* 2017;76:978-991. 2. Sieper J et al. *Arthritis Rheum.* 2013;65:534-551.

Why Care About axSpA?

Not every patient's back pain is mechanical	Much more common than we think	Frequently missed by health care providers	Associated with comorbidities such as HTN, CAD, and CVA

axSpA has great treatment options,
and if treated early, future progression may be prevented or delayed

Inflammatory Back Pain (IBP)[1-4]

- Cardinal symptom particularly in patients with axSpA
- Usually starts before aged 45 years and lasts >3 months
- Sensitivity of IBP for diagnosis of axSpA is 70%-80%, but specificity is 20%-40%
- In large cohorts of patients with back pain, up to 30% patients without axSpA may have IBP

1. Underwood MR et al. *Br J Rheumatol*. 1995;34:1074-1077. 2. Reveille JD et al. *Am J Med Sci*. 2013;345:431-436.
3. Poddubnyy D et al. *J Rheumatol*. 2011;38:2452-2460. 4. Poddubnyy D et al. *RMD Open*. 2018;4:e000825.

IBP vs MBP

	IBP	MBP
Onset	Insidious	Variable
Morning stiffness	Usually >30 min	Usually minor
Maximum pain/stiffness	Early morning and second half of night (OR = 20)	Late in day
Exercise/activity	Improves symptoms (OR = 23)	Worsens symptoms
Duration	Chronic	Acute or chronic
Age at onset	Before aged 45 y	Variable
Response to NSAIDs	Significant	Variable

Characteristics of IBP[1]

Age at onset <45 y
Duration >3 mo
Insidious onset
Morning stiffness >30 min
Improvement with exercise (OR = 23)
No improvement with rest
Awakening from pain, especially during second half of night, with improvement on arising (OR = 20)
Alternating buttock pain

IBP if 4 or more are positive

1. Taurog JD et al. *N Engl J Med*. 2016;374:2563-2574.

Radiographic Progression in nr-axSpA

Estimated that 10%-40% of patients with nr-axSpA progress to radiographic axSpA (AS) over a period of 2-10 years

Patients with nr-axSpA who do not progress to AS do not necessarily remit they just do not develop radiographic changes despite persistence of the IBP

Risk Factors Associated With Progression to AS

Consistent Risk Factors

- Baseline low grade x-ray sacroiliitis[1,2]
- Baseline MRI inflammation[3]
- Elevated CRP[2]

Inconsistent Risk Factors

- *HLA-B27* positivity inconsistent between studies[2,3]
- Buttock pain[4]
- Smoking[5,6]

1. Huerta-Sil G et al. *Ann Rheum Dis*. 2006;65:642-646. 2. Poddubnyy D et al. *Ann Rheum Dis*. 2011;70:1369-1374.
3. Bennett AN et al. *Arthritis Rheum*. 2008;58:3413-3418. 4. Sampaio-Barros PD et al. *J Rheumatol*. 2010;37:1195-1199.
5. Machado P et al. *Arthritis Rheum*. 2011;63(suppl 10):1650. 6. Poddubnyy D et al. *Arthritis Rheum*. 2012;64:1388-1398.

How Do We Make the Diagnosis of axSpA?

Gold standard for diagnosis of axSpA is clinician's judgement

No specific biomarker yet

CRP, *HLA-B27*, x ray SIJs, and MRI SIJs

Modified New York Criteria (1984) for AS[1]

Clinical Criteria

a. Low back pain and stiffness for more than 3 months that improves with exercise but is not relieved by rest
b. Limitation of motion of the lumbar spine in both the sagittal and frontal planes
c. Limitation of chest expansion relative to normal values correlated for age and sex

Radiologic Criterion

a. Sacroiliitis grade ≥2 bilaterally or grade 3-4 unilaterally

Definition of AS
If the radiologic criterion is associated with at least one clinical criterion

1. van der Linden S et al. *Arthritis Rheum*. 1984;27:361-368.

ASAS Classification Criteria for axSpA[1]

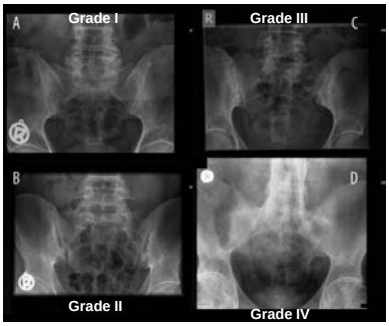
In patients with ≥3 mo back pain and age at onset <45 y

Sacroiliitis on imaging plus ≥1 SpA feature **or** *HLA-B27* plus ≥2 other SpA features

- SpA features
  - IBP; arthritis; enthesitis (heel); uveitis; dactylitis; PsO; Crohn's disease/colitis; good response to NSAIDs; family history for SpA; *HLA-B27*; elevated CRP
- Sacroiliitis on imaging
  - Active (acute) inflammation on MRI highly suggestive of sacroiliitis associated with SpA
  - Definite radiographic sacroiliitis according to modified New York criteria

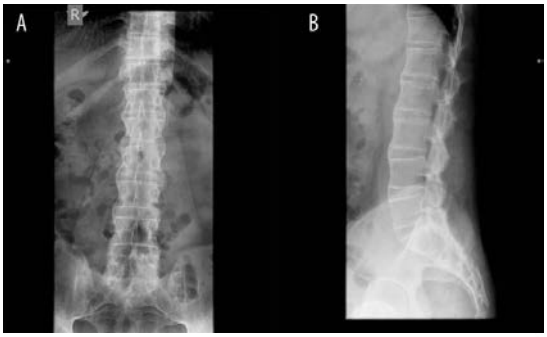
1. Rudwaleit M et al. *Ann Rheum Dis*. 2011;70:25-31.

Imaging in Spondyloarthritis[1]



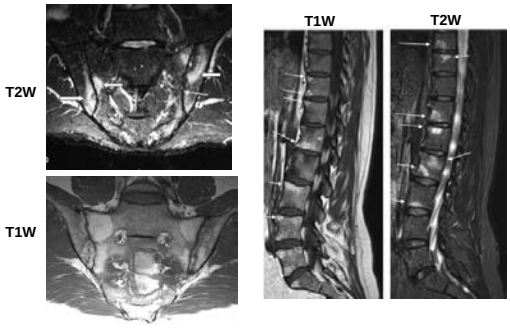
1. Sudoł-Szopinska I, Urbanik A. *Pol J Radiol*. 2013;78:43-49.

Bamboo Spine[1]



1. Sudoł-Szopinska I, Urbanik A. *Pol J Radiol*. 2013;78:43-49.

Imaging in Spondyloarthritis[1]



1. Baraliakos X et al. *Best Pract Res Clin Rheumatol*. 2016;30:608-623.

Probability of axSpA Diagnosis Based on Clinical Features and Imaging[1]

Insidious onset, chronicity (>3 mo), association with morning stiffness, improvement with exercise but not rest, and alternating buttock pain

Among patients with CBP in GP/PCP office — 5%

Presence of IBP — 15%

Presence of 1 or 2 additional SpA features: enthesitis, dactylitis, uveitis, positive family history, Crohn's disease, alternating buttock pain, PsO, asymmetrical arthritis, positive response to NSAIDs, acute phase reactants (raised ESR/CRP) — 30%-70%

HLA-B27+ (59%-90%) — >90%

Probability of axSpA

Imaging: X-ray SIJ+ = AS; X-ray SIJ- and MRI SIJ+ = nr-axSpA

1. Rudwaleit M et al. *Ann Rheum Dis*. 2004;63:535-543.

Cohort Studies: Key Differences Between AS and nr-axSpA[1-6]

Variable	AS	nr-axSpA
Age at onset	Similar	Similar
Sex	**Men:Women 3:1**	**Men:Women 1:1**
HLA-B27 positive	Similar	Similar
CRP	**Higher**	**Normal/High**
Pain scores	Similar	Similar
Disease activity	Similar	Similar
Functional capacity	**More impaired**	**Less impaired**
Spinal mobility	**More limited**	**Less limited**
TNFi response	Similar	Similar

1. Baraliakos X et al. *RMD Open*. 2015;1:e000053. 2. Kiltz U et al. *Arthritis Care Res (Hoboken)*. 2012;64:1415-1422. 3. Rudwaleit M et al. *Arthritis Rheum*. 2009;60:717-727. 4. Wallman JK et al. *Arthritis Res Ther*. 2015;17:378. 5. Callhoff J et al. *Ann Rheum Dis*. 2015;74:1241-1248. 6. Sieper J et al. *Ann Rheum Dis*. 2013;72:815-822.

Single Center, Multicenter, and International Studies Evaluating Strategies for the Screening and Referral of axSpA[1]

Author and Year	Referring Physicians	Strategy	N	Male, %	axSpA, %	AS %	nr-axSpA, %
Brandt 2007	Ortho primary care	• LBP >3 mo, onset at <45 y ≥1 of IBP, + *HLA-B27*, sacroiliitis on available imaging	350	48.6	45.4	50.3	49.7
Hermann 2009	GP	• Calin criteria (4/5)	92	41	33	53.3	46.7
Braun 2011	Ortho	• LBP >2 mo < 10 y, onset at <45, y ≥1 of AM stiffness, improvement with exercise, not rest, waking at night, improvement with NSAID	322	50	35	41.6	58.4
Poddubnyy 2011 (MASTER study)	GP and ortho	• LBP >3 mo, onset <45 y • Strategy 1: ≥1 of IBP, + *HLA-B27*, sacroiliitis on available imaging • Strategy 2: 2 of 5 IBP, + *HLA-B27*, sacroiliitis on available imaging, positive family history for AS, good response to NSAID	318 242	53 55	41.8 36.8	61.6 61.8	38.4 38.2
Sieper 2012 (RADAR study)	Primary care Other (GP, neuro, ortho)	• LBP >3 mo, onset <45 y • Strategy 1: ≥1 of IBP, + *HLA-B27*, sacroiliitis on available imaging • Strategy 2: 2 of 6 IBP, + *HLA-B27*, sacroiliitis on available imaging, positive family history for AS, good response to NSAID, EAM	504 568	47 55	35.6 39.8	77 78	23 22
van den Berg 2013	GP, eye, gastroenterology	• LBP >3 mo, but <2 y, onset <45 y	157	33	41.4	18	82
Deodhar 2014 (ProSpA study)	Self rheumatology Other physicians	• LBP >3 mo, onset <45 y ≥1 of IBP, + *HLA-B27*, sacroiliitis on available imaging	751	50	46	31	69

1. Danve A et al. *Clin Rheumatol*. 2015;34:987-993.

Possible Screening Approach for axSpA in Patients With Chronic Low Back Pain[1]

CBP (>3 mo) first symptoms in patients aged <45 years

IBP | **Sacroiliitis on any imaging** | ***HLA-B27+***

Refer to a rheumatologist

With this approach, a rheumatologist will need to see three patients with back pain to be able to diagnose one patient with axSpA

1. Sieper J et al. *Ann Rheum Dis*. 2005;64:659-663.

Why Care About axSpA?

Not every patient's back pain is mechanical	**Much more common than we think**	Frequently missed by health care providers	Associated with comorbidities such as HTN, CAD, and CVA

axSpA has great treatment options,
and if treated early, future progression may be prevented or delayed

How Common Is axSpA in Clinical Practice?

How Common Is axSpA in the United States?[1,2]

- NHANES 2009-2010 of 5,103 participants in the United States showed
  - Chronic low back pain in 19.4%
  - IBP in 7%
  - axSpA in 1% (0.9%-1.4%)
  - AS in 0.5%
  - *HLA-B27* prevalence 6.1%
    - Non-Hispanic whites (Caucasians): 7.5%
    - Mexican Americans: 4.6%

1. Weisman MH et al. *Ann Rheum Dis*. 2013;72:369-373. 2. Reveille JD et al. *Arthritis Rheum*. 2012;64:1407-1411.

Genetics of AS[1]

- Prevalence of *HLA-B27* in Caucasians is 6%-9%
- 90%-95% of patients with AS are *HLA-B27*+
- Less than 5% of patients who are *HLA-B27*+ develop AS; however, 20% of relatives of *HLA-B27*+ patients with AS will develop some kind of spondyloarthritis
- MZ twin concordance rate for AS is 63% compared with DZ concordance rate of 12.5%

1. Reveille J. *Ann Rheum Dis*. 2011;70(suppl 1):i44-i50.

Prevalence of AS and HLA-B27

Population	AS Prevalence, %	*HLA-B27* Prevalence, %
United States[1,2]	0.52	6
The Netherlands[3]	0.1	8
Germany[4]	0.55	9
Norway[5]	1.1-1.4	14
Haida Indians[6]	6.1	50

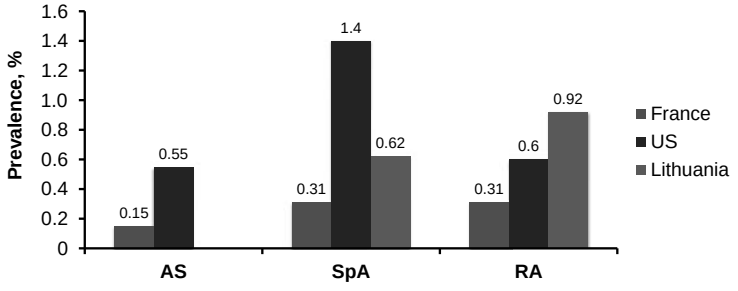
1. Helmick CG et al. *Arthritis Rheum*. 2008;58:15-25. 2. Reveille JD et al. *Arthritis Rheum*. 2012; 64:1407-1411.
3. van der Linden S et al. *Arthritis Rheum*. 1984; 27:241-249. 4. Braun J et al. *Arthritis Rheum*. 2005;52:4049-4050.
5. Gran T et al. *Ann Rheum Dis*. 1985;44:359-367. 6. Gofton JP et al. *Ann Rheum Dis*. 1966;25:525-527.

Frequency of *HLA-B27* in Patients With SpA[1,2]

Disorder	Frequency, %
AS	80-90
Reactive arthritis	30-50
Psoriatic arthritis	25
Psoriatic spondylitis	60
Enteropathic arthritis	7
Enteropathic spondylitis	70
General US population	6.1

1. Reveille JD, Arnett FC. *Am J Med*. 2005;118:592-603. 2. Reveille JD, Weisman MH. *Am J Med Sci*. 2013;345:431-436.

axSpA May Be More Common Than Rheumatoid Arthritis in the United States[1-4]



1. Saraux A et al. *Ann Rheum Dis*. 2005;64:1431-1435. 2. Guillemin F et al. *Ann Rheum Dis*. 2005;64:1427-1430.
3. Adomaviciute D et al. *Scand J Rheumatol*. 2008;37:113-119. 4. Helmick CG et al. *Arthritis Rheum*. 2008;58:15-25.

Why Care About axSpA?

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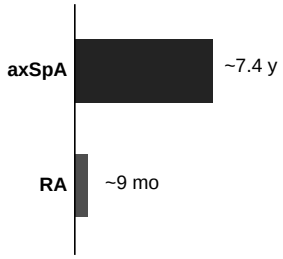
Diagnostic Delay

Average Time to Diagnosis[1,2]



1. Chan KW et al. *Arthritis Rheum.* 1994;37:814-820. 2. Redeker I et al. 2018 American College of Rheumatology/Association of Rheumatology Health Professionals Annual Meeting (2018 ACR/ARHP). Abstract 1658.

axSpA is Often Undiagnosed in Patients Seen by Rheumatologists for Evaluation of Chronic Back Pain—PROSpA Study[1]

Referral strategy: If patients with CBP starting before aged 45 years with either IBP, HLB-B27 positivity, or sacroiliitis

Prevalence of axSpA was 46%

There is a 14-year delay in the diagnosis of axSpA in the United States

42% of patients were existing patients in rheumatology practices

1. Deodhar A et al. *Arthritis Rheumatol.* 2016;68:1669-1676.

AS Is Often Misdiagnosed by Nonrheumatologists[1]

Truven Healthcare Database: 127 million patients

63% of AS diagnoses made by nonrheumatologists

When patients with a diagnosis of AS are referred to a rheumatologist, the diagnosis is confirmed in only 42% of cases

1. Deodhar A et al. *Clin Rheumatol.* 2016;35:1769-1776.

Kaiser Permanente Northern California: Under-Recognition and Lack of Referral of Patients With AS by PCPs[1]

Kaiser Permanente Northern California Database

Point prevalence of "any spondyloarthritis diagnosis was 0.2%, AS was 0.1%; that is one-tenth of national estimates of prevalence

Less than 50% of patients diagnosed were referred to rheumatology

1. Curtis JR et al. *Perm J.* 2016;20:15-151.

Reasons for Missed and Delayed Diagnosis

- Common nature of back pain in general population
- Patients with axSpA are seen by other clinicians before rheumatologists
  - Family practice, internal medicine, chiropractors, osteopaths, physical therapists, orthopedic surgeons, spine surgeons, neurosurgeons, physiatrists, dermatologists, ophthalmologists, gastroenterologists
- Radiographs of the SI joints may be normal (nr-axSpA).
- Most common MRI scan ordered for low back pain is L Spine, sacroiliitis may be missed.
- Lack of reliable biomarkers other than *HLA-B27*

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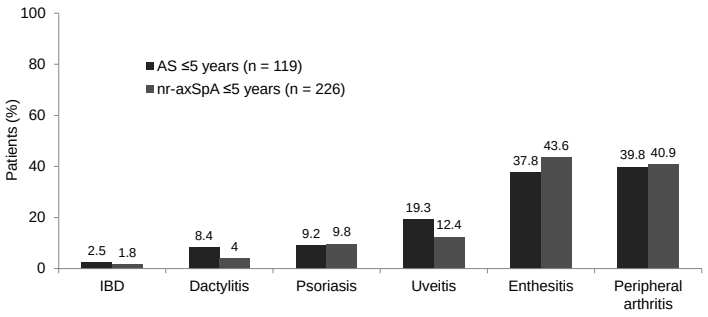
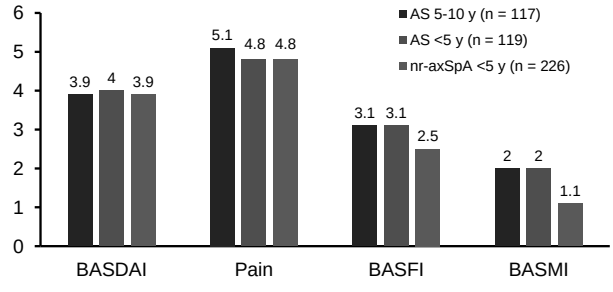
Extra-Spinal Manifestations[1-3]



1. Rudwaleit M et al. *Arthritis Rheum.* 2009;60:717-727. 2. Kopylov U et al. *J Rheumatol.* 2018;45:498-505. 3. Deodhar A. *Am J Manag Care.* 2019;25:-S0

Burden of Disease in AS and nr-axSpA Is Similar[1]



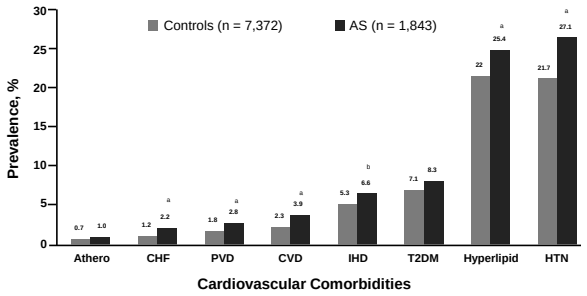
1. Rudwaleit M et al. *Arthritis Rheum.* 2009;60:717-727.

Late Complications of axSpA[1]

Extra-Articular Manifestations	Clinical Manifestations
Cardiac	Aortitis; aortic insufficiency; conduction disorders; bundle-branch and atrioventricular blocks
Renal	Secondary amyloidosis; IgA nephropathy
Pulmonary	Fibrosis of the upper lobe and pleural thickening
Neurologic	Cauda equina syndrome

1. Ewerton Maia Rodrigues C et al. *Rev Bras Rheumatol*. 2012;52:375-383.

Cardiovascular Comorbidities in Patients With AS: Integrated US Health Plan Data[1]



[a] $P < .01$. [b] $P < .005$.
1. Han C et al. *J Rheumatol*. 2006;33:2167-2172.

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and if treated early, future progression may be prevented or delayed

Goals of axSpA Management

- Reduce symptoms
- Maintain spinal flexibility and posture
- Reduce functional limitations
- Maintain ability to work
- Decrease complications

Initial Therapy: Nonpharmacologic Interventions

Patient education

- Nature of disease and advice about need for a lifelong exercise and posture-training program
  and about their working and leisure habits relevant to axSpA
- Medication compliance and monitoring of disease activity and for potential AEs of therapies

Counseling regarding smoking cessation

Depression screening and psychosocial support

Physical therapy

- Newly diagnosed patients should be referred for physical therapy
- Exercises include postural training, range of motion exercises, stretching, and recreational activities
- Spinal manipulation should be avoided in patients with spinal fusion or advanced spinal osteoporosis

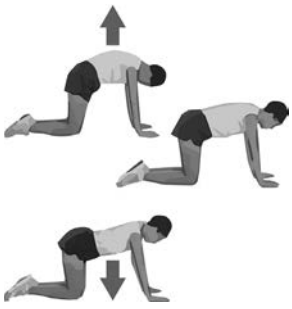
Exercise and Physical Therapy[1]

Cochrane review of 11 trials with 763 participants

- Individualized home-based or supervised exercise programs are better than no exercise
- Supervised group PT is better than home exercise
- Combined in-patient spondyloarthritis exercise followed by group PT is better than group PT alone

ACR/SAA/SPARTAN

- PT has been strongly recommended in both active and stable AS
- Recommendations
  - Supervised PT + unsupervised back exercises
  - Both land-based and aquatic PT are very effective



1. Dagfinrud H et al. *Cochrane Database Syst Rev*. 2008:CD002822.

Pharmacological Treatment Options

- NSAIDs
- csDMARDS
- TNF inhibitors
- IL-17A inhibitors

JAK inhibitors are in development

First-Line Treatment of Active AS: 2019 ACR/SAA/SPARTAN Recommendations[1]

Active AS and nr-axSpA (Axial Disease)

NSAIDs

- Continuous
- No preferred NSAID

Physical therapy

- Active over passive
- Land-based over aquatic

Against systemic GCs

Determine additional disease manifestations

Peripheral Arthritis Despite NSAIDs

Local GC if ≤2 joints
SSX

- SSZ over MTX

Against LEF, APR, THL, and PAM

Isolated Sacroiliitis or Enthesitis

NSAIDs

Isolated Sacroiliitis or Enthesitis Despite NSAIDs

Local GC

Avoid Achilles, patellar, and quadriceps entheses GC injections

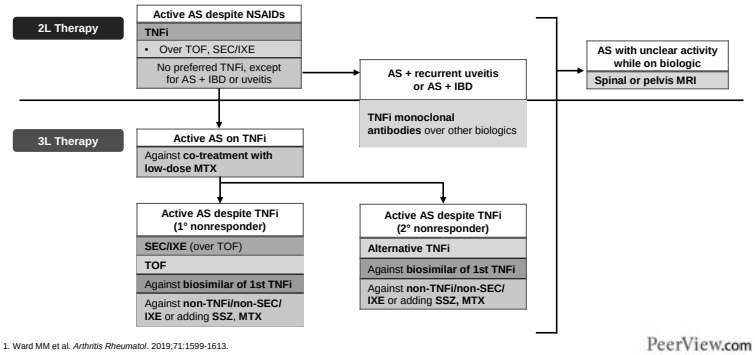
1. Ward MM et al. *Arthritis Rheumatol*. 2019;71:1599-1613.

Multiple RCTs Support Efficacy of NSAIDs in axSpA[1,2]

- NSAIDs as first-line therapy in axSpA/AS
  - Various NSAIDs are equally effective
  - Should use two full strength NSAIDs before advancing to a biologic
  - Unclear if NSAIDs prevent radiographic progression in AS

1. Ward MM et al. *Arthritis Rheumatol*. 2016;68:282-298. 2. Kroon FP et al. *Cochrane Database Syst Rev*. 2015;7:CD010952.

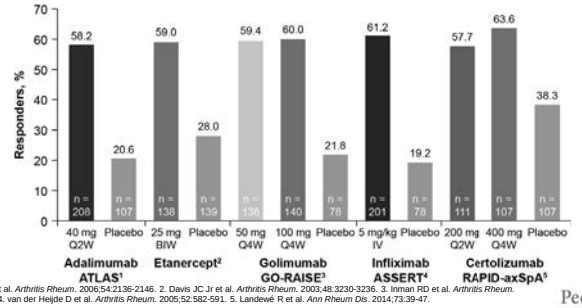
Active AS Despite NSAIDs: 2019 ACR/SAA/SPARTAN Recommendations[1]



1. Ward MM et al. *Arthritis Rheumatol*. 2019;71:1599-1613.

TNFi Therapy

ASAS20 response rates at week 24 for infliximab, week 14 for golimumab, and week 12 for adalimumab, certolizumab pegol, and etanercept



1. van der Heijde D et al. *Arthritis Rheum*. 2006;54:2136-2146. 2. Davis JC Jr et al. *Arthritis Rheum*. 2003;48:3230-3236. 3. Inman RD et al. *Arthritis Rheum*. 2008;58:3402-3412. 4. van der Heijde D et al. *Arthritis Rheum*. 2005;52:582-591. 5. Landewé R et al. *Ann Rheum Dis*. 2014;73:39-47.

TNFi: axSpA[1]

Variables associated with TNFi response

Elevated CRP/ESR	Short duration of symptoms	Inflammation noted by MRI

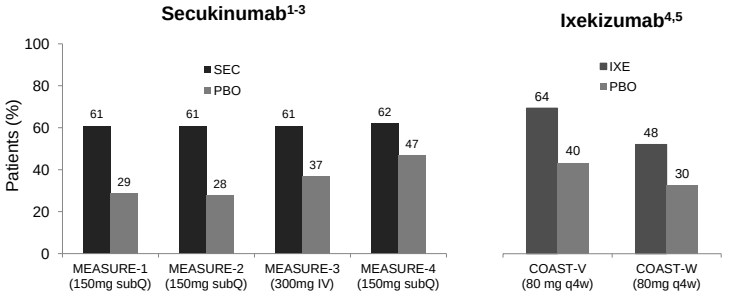
30%-40% of patients with AS continue with active disease despite NSAIDs/TNFi therapy

1. Sieper J et al. *Lancet*. 2017;390:73-84.

IL-17A Inhibitors for axSpA

Secukinumab (approved)	Ixekizumab (approved)	Bimekizumab (in development)	Netakimab (in development)

Efficacy of IL-17A Inhibitors in the Treatment of AS: ASAS20 at Week 16[1]

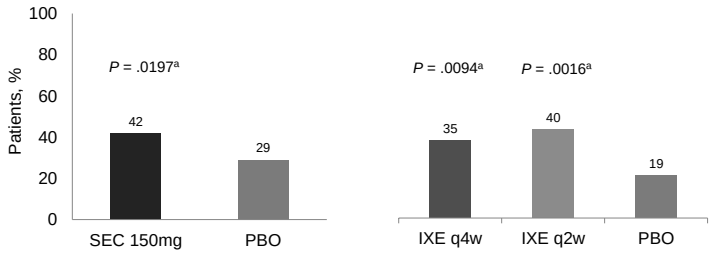


1. Baeten D et al. *N Engl J Med*. 2015;373:2534-2548. 2. Pavelka K et al. *Arthritis Res Ther*. 2017;19:285. 3. Kivitz AJ et al. Rheumatol Ther. 2018;5:447-462. 4. van der Heijde D et al. *Lancet*. 2018;392:2441–2451. 5. Deodhar A et al. *Arthritis Rheumatol*. 2019;71:599-611.

Efficacy of IL-17A Inhibitors in the Treatment of nr-axSpA: ASAS40 at Week 16

Secukinumab (TNFi-naïve patients): PREVENT[1]

Ixekizumab (inadequate response or intolerance to NSAID therapy): COAST-X[2]



[a] versus placebo.
1. Deodhar A et al. *Arthritis Rheumatol*. 2020;73:110-120. 2. Deodhar A et al. *Lancet*. 2020;395:53-64.

ACR/SAA/SPARTAN Treatment Recommendations 2019: IL-17A Inhibitors[1]

Recommendation	Level of Evidence
• In patients with AS despite treatment with NSAIDs, secukinumab or ixekizumab is strongly recommended over no treatment with secukinumab or ixekizumab	High
• In patients with active AS despite treatment with TNFi, secukinumab or ixekizumab is conditionally recommended over treatment with a different TNFi in patients with a primary nonresponse to TNFi	Very low
• In adults with active AS despite treatment with NSAIDs who have contraindications to TNFis, secukinumab or ixekizumab are conditionally recommended over treatment with methotrexate, sulfasalazine, or tofacitinib	Low

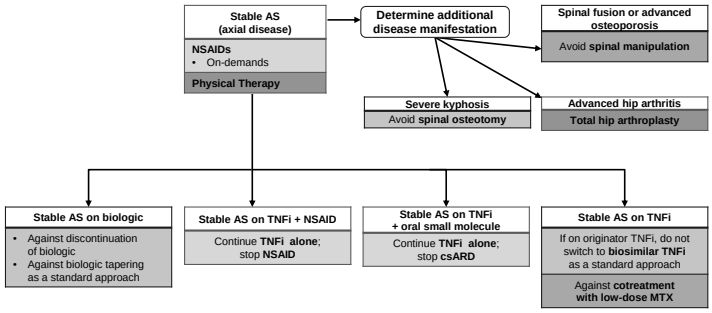
1. Ward MM et al. *Arthritis Rheumatol*. 2019;71:1599-1613.

JAK Inhibitors for axSpA[1-3]

Tofacitinib	Filgotinib	Upadacitinib

1. van der Heijde D et al. *Ann Rheum Dis*. 2017;76:1340-1347. 2. van der Heide D et al. *Lancet*. 2018;392:2378-2387.
3. van der Heijde D et al. 2019 ACR/ARP Annual Meeting (ACR 2019). Abstract 2728.

Treatment of Stable AS: 2019 ACR/SAA/SPARTAN Recommendations[1]



1. Ward MM et al. *Arthritis Rheumatol*. 2019;71:1599-1613.

Summary[1,2]

- axSpA (AS and nr-axSpA) is the most frequent type of spondyloarthritis
- Early referral is very important for timely diagnosis and management of axSpA
- Average delay in diagnosis is 8-11 years
- Disease activity (BASDAI) is similar in AS and nr-axSpA
- Patients with nr-axSpA respond as well to TNF inhibitors as well as patients with AS
- Differentiating features of nr-axSpA from AS
  - M:F 1:1
  - Less frequent CRP elevation and MRI lesions
  - Less impaired mobility

1. Taurog JD et al. *N Engl J Med*. 2016;375:1303. 2. Sieper J, Poddubnyy D. *Lancet*. 2017;390:73-84.

Audience Q&A

PeerView Live

Please remember to complete and submit your

Program Evaluation: https://PeerView.com/axSpA-Eval-WXC

Visit us at: PeerView.com/WXC

- Download slides and Practice Aids
- Watch the online version of this activity
- Request a live meeting at your institution
- Join the conversation on Twitter @PeerView

Thank you and good day.

PeerView Live

Abbreviations

AAU: acute anterior uveitis
ACR: American College of Rheumatology
APR: apremilast
AS: ankylosing spondylitis
ASAS: Assessment of Spondyloarthritis International Society
ASDASCRP: Ankylosing Spondylitis Disease Activity Score–C-Reactive Protein
ASDASESR: Ankylosing Spondylitis Disease Activity Score–Erythrocyte Sedimentation Rate
axSpA: axial spondyloarthritis
BASDAI: Bath Ankylosing Spondylitis Disease Activity Index
BASFI: Bath Ankylosing Spondylitis Functional Index
BASMI: Bath Ankylosing Spondylitis Metrology Index
BMP: bone morphogenic protein
CAD: coronary artery disease
CLBP: chronic lower back pain
CPB: chronic back pain
CRP: C-reactive protein
csARD: conventional synthetic antirheumatic drug
csDMARD: conventional synthetic disease-modifying antirheumatic drug
CVA: cerebrovascular accident
CZP: certolizumab pegol
DC: dendritic cell

Abbreviations (Cont'd)

DZL dizygotic
EAM: extra-articular manifestations
ER: endoplasmic reticulum
ESR: erythrocyte sedimentation rate
F/H: family history
GC: glucocorticoids
GP: general practitioner
HLA-B27: human leukocyte antigen B27
HLA-B27–B2m: human leukocyte antigen B 27 beta-2-microglobulin
HTN: hypertension
IBD: inflammatory bowel disease
IBP: inflammatory back pain
IgA: immunoglobulin A
IL-17: interleukin 17
IL-23: interleukin 23
IL-23R: interleukin 23R
ILC3: type 3 innate lymphoid cells
Interferon γ: interferon gamma
IXE: ixekizumab
JAK: Janus kinase
KIR3DL2: Killer cell immunoglobulin-like receptor, three Ig domains and long cytoplasmic tail 2
LBP: lower back pain
LEF: leflunomide
LR: likelihood ratio

Abbreviations (Cont'd)

M-H: Mantel–Haenszel
MBP: mechanical back pain
MTX: methotrexate
MZ: monozygotic
Mφ: macrophage
NBBM: nonbiologic background medication
Neuro: neurologist
NHANES: National Health and Nutrition Examination Survey
nr-axSpA: nonradiographic axial spondyloarthritis
Ortho: orthopedist
PAM: pamidronate
PCP: primary care provider
PsA: psoriatic arthritis
PsO: psoriasis
PT: physical therapy
Q2W: every 2 weeks
Q4W: every 4 weeks
RA: rheumatoid arthritis
ReA: reactive arthritis
RF: rheumatoid factor
ROM: range of motion
SAA: Spondylitis Association of America
SEC: secukinumab
SI: sacroiliac
SIJ: sacroiliac joint

Abbreviations (Cont'd)

SpA: spondyloarthritis

SRTN: Spondyloarthritis Research and Treatment Network

SSZ: sulfasalazine

STIR: short tau inversion recovery

Th17: helper T cell 17

THL: thalidomide

TNF: tumor necrosis factor

TNFi: tumor necrosis factor inhibitor

TNF-α: tumor necrosis factor alpha

TOF: tofacitinib

Wnt: wingless-related integration site

PeerView Live

Overcoming Challenges in the Diagnosis and Management of Axial Spondyloarthritis

New Insights and Implications for Clinical Practice

To assist us in evaluating the effectiveness of this activity and to make recommendations for future educational offerings, please take a few minutes to complete the evaluation form. PVI, PeerView Institute for Medical Education, respects and appreciates your opinions. You may return this evaluation form on-site to an AAFP or PeerView staff member or via mail, fax, or email.

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New York, NY 10014

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Activity Evaluation Form

1. To what extent have the information and practice strategies discussed in this activity improved your ability to competently manage patients/support patient care?

Not at all				Very much	
1	2	3	4	5	N/A

2. After participating in this activity, how often do you plan to do the following?

	Never	Infrequently	Sometimes	Frequently	Always	
Consider axSpA in patients with chronic low back pain who are younger than 45 years	1	2	3	4	5	N/A
Recommend appropriate initial treatment for newly diagnosed patients with axSpA	1	2	3	4	5	N/A
Collaborate with rheumatologists to provide optimal treatment and longitudinal support for patients with axSpA	1	2	3	4	5	N/A

3. Please indicate your level of agreement with the following statements:

	Strongly **disagree**				Strongly **agree**
The content was presented in a fair and unbiased manner.	1	2	3	4	5
The content was evidence-based.	1	2	3	4	5
The content was relevant to my practice.	1	2	3	4	5
The format of this activity was useful and conducive to learning.	1	2	3	4	5
The faculty demonstrated expertise in subject matter.	1	2	3	4	5
The interactive questions positively impacted my learning.	1	2	3	4	5

4. As a result of your participation in this activity, please indicate your ability to meet each of the stated educational objectives.

	Not able				**Very able**
Identify axial spondyloarthritis (axSpA) in patients with inflammatory back pain via assessment of medical history, musculoskeletal symptoms and findings, and extra-articular manifestations and comorbidities	1	2	3	4	5
Apply classification criteria and diagnostic tests into clinical practice to support early detection of axSpA	1	2	3	4	5
Assess efficacy and safety data related to novel biologic options for axSpA, recognizing the potential clinical impact on the management of patients who do not respond well to traditional pharmacologic therapies	1	2	3	4	5
Employ treatment plans for individual patients with axSpA in accordance with current evidence, expert recommendations, and patient needs and preferences	1	2	3	4	5
Recognize the importance of collaborating with rheumatologists to provide optimal treatment and longitudinal support for patients with axSpA	1	2	3	4	5

5. Please indicate the likelihood of the following statements:

	Not at all likely				**Very** likely
I will make changes to my practice after participating in this activity.	1	2	3	4	5
Practice changes I make based on this activity will improve my and the healthcare teams' ability to affect patients' outcomes.	1	2	3	4	5
I would participate in future activities on this topic presented in a similar format.	1	2	3	4	5
I would recommend this activity to my colleagues.	1	2	3	4	5

6. Which of the following barriers or challenges that you encounter in your care of patients or practice will this activity help you overcome? (Indicate all that apply.)

- ❑ Engaging patients and caregivers in shared decision making
- ❑ Coordinating care with interprofessional team
- ❑ Patient adherence
- ❑ Decision making in the presence of conflicting evidence
- ❑ Lack of training/experience with this specific topic
- ❑ Cost/Reimbursement/Therapy Approval Status
- ❑ Other (please specify): Detailed feedback is encouraged and appreciated _____
- ❑ None; I do not encounter barriers in the care of patients/my practice

7. Do you intend to change your practice as a result of participating in this activity? ❑ **Yes** ❑ **No** If so, how? If not, why not?

8. Please provide any other feedback or comments regarding this activity.

Contact Information

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Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

EKG Review

Presented by:
John Villasenor, MD

Approved for 1.0 Prescribed CME

Saturday, July 31, 2021
9:00—10:00pm

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
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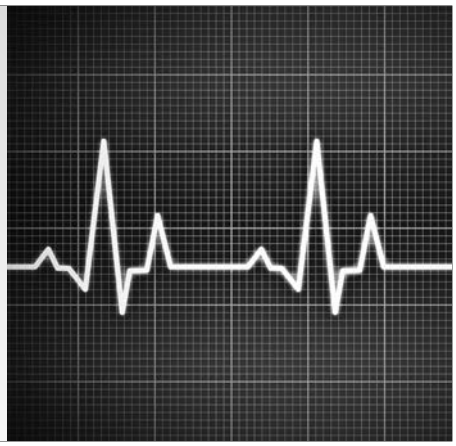
NOTES

EKG Refresher

JOHN E. VILLASENOR MD

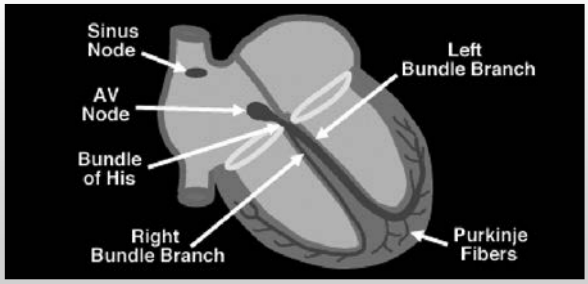


Understanding EKGs

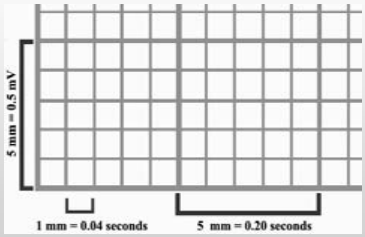
INTRODUCTION

- General Principles
- Enlargement and Hypertrophy
- Arrhythmias
- Conduction blocks
- Preexcitation Syndromes
- Myocardial Infarcts and ischemia
- Electrolyte Disturbances
- Examples

General Principles

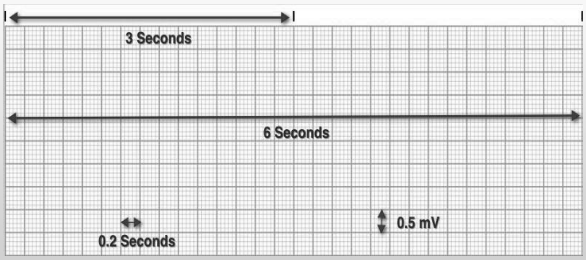


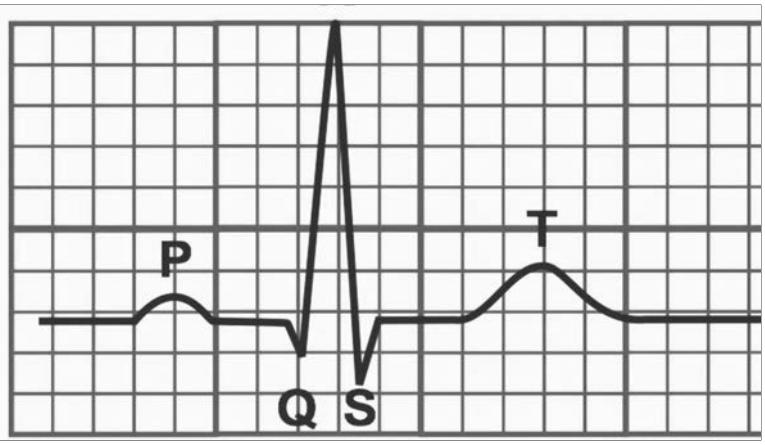
General Principles



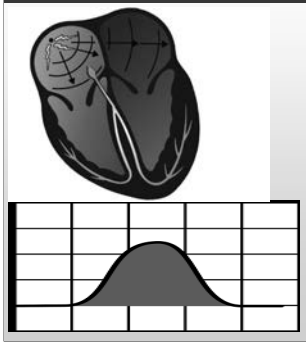
- Smallest box is 0.04 seconds and 1mm
- Bigger boxes 0.2 seconds and 5 mm
  - Composed of 5 small boxes of 0.04 seconds

General Principles



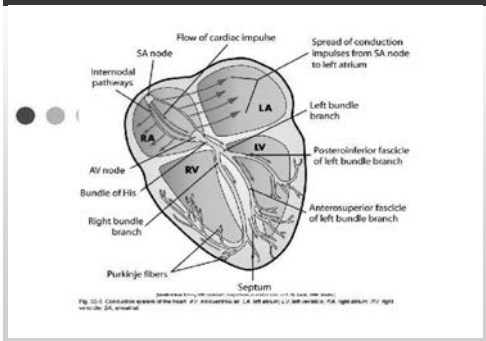


Atrial Depolarization



- SA node in the upper Right atrium fires and atria contract first right atrium then left atrium
- Forms a P Wave which consist of the first half right atrium second part left atrium
- Then a pause occurs when the electrical signal reaches the AV node near the intraventricular septum that delay conduction to nearly a pause to allow the atria to complete contraction before contraction of ventricles occurs

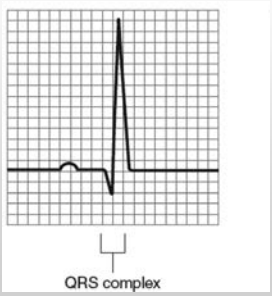
Ventricular Depolarization



- After the pause from the AV node the current continues down to the path of the ventricles reaching first the
  - bundle of His
  - then Bundle branches
  - and lastly Purkinje fibers

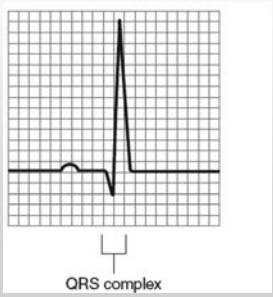
- Note: LBB splits into
  - Septal fascicle
  - Left anterior fascicle
  - Left posterior fascicle

QRS Complex



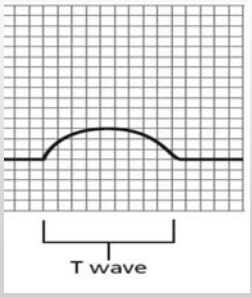
- First part of QRS is from the depolarization of the IV septum via septal fascicle of the LBB
  - Small Q wave
- Both ventricles then depolarize making up the remainder of the complex which structurally depicts of the left ventricle due to size
  - LV is 3 times the size of RV in normal cases

QRS Complex



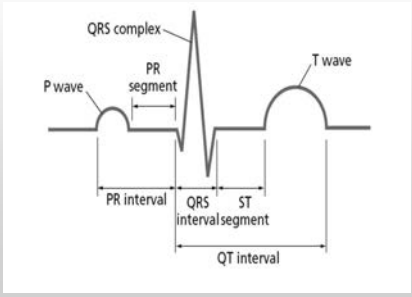
- Larger in size than P waves due to muscle size of ventricles
- Atrial repolarization not seen due to sheer size of QRS

Ventricle Repolarization



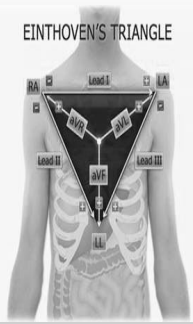
- A much slower electrical current than Ventricular Depolarization that gives us the T wave

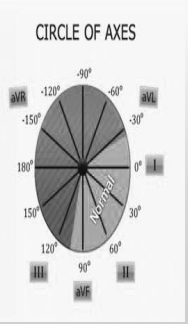
Nomenclature



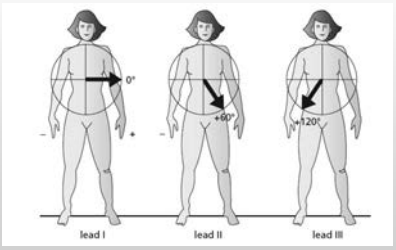
- PR interval
  - is the start of AD to start of VD
- PR segment
  - end of AD and start of VD
- QRS interval
  - time of VD
- ST segment
  - end of VD to start of VR
- QT interval
  - start of VD to end of VR
- R to R interval
  - 1 cycle

Einthoven's Triangle



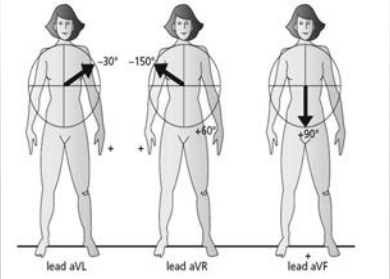


3 Standard limb leads



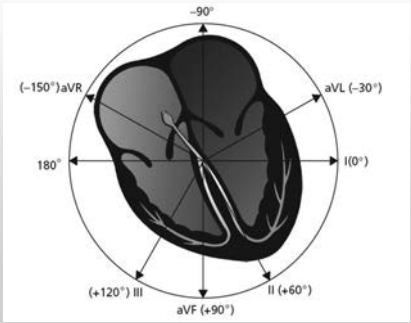
- 3 Standard limb leads
  - Lead I
    - Positive Left arm, negative right arm
    - Vector to 0 degrees
  - Lead II
    - Positive legs, and negative right arm
    - Vector to positive 60 degrees
  - Lead III
    - Positive legs, negative left arm
    - Vector to positive 120 degrees

3 Augmented limb leads



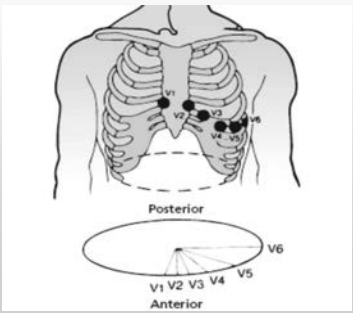
- 3 Augmented limb leads
  - aVL
    - Positive left arm, other limbs negative
    - Vector is negative 30 degrees
  - aVR
    - Positive right arm, other limbs negative
    - Vector is negative 150 degrees
  - aVF
    - Positive legs, other limbs negative
    - Vector is positive 90 degrees

6 Limb leads



Lead	Angle
Inferior Leads	
Lead II	+60°
Lead III	+120°
Lead aVF	+90°
Left Lateral Leads	
Lead I	+0°
Lead aVL	−30°
Right-sided Lead	
Lead aVR	−150°

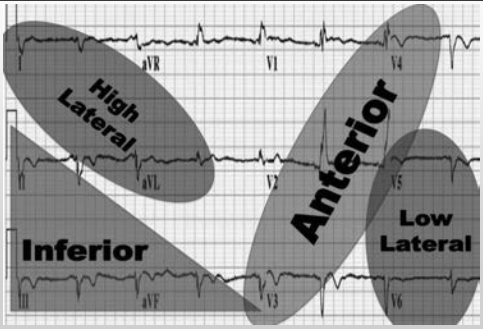
6 Precordial leads



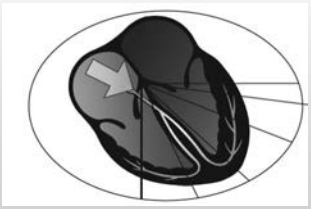
- V1
  - 4th intercostal space of right side of sternum
- V2
  - 4th intercostal space of left side of sternum
- V3
  - Between V2 and V4
- V4
  - 5th intercostal space in the midclavicular line
- V5
  - Between V4 and V6
- V6
  - 5th Intercostal space in the midaxillary line

Categorizing leads

- Leads
  - Anterior leads: V2, V3, V4
  - Left Lateral leads: I, aVL, V5, V6
  - Inferior leads: II, III, aVF
  - Right ventricular: V1, aVR



Recap on the P Wave

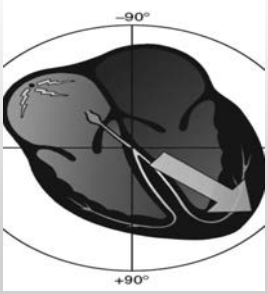


- Current starts in the SA node (upper right atrium) and goes in the direction to AV node on general direction of the left ventricle
- PR interval from start of Atrial depolarization to start of Ventricular depolarization is usually 0.12 to 0.2 seconds (3-5 small boxes)

Normal P Wave Morphology

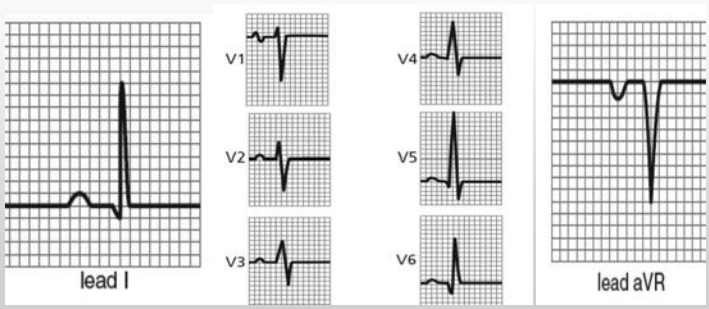
- aVR
- Lead I
- V6
- Lead III
- V1

QRS Complex

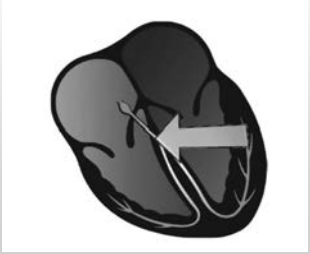


- Starts off after ther AV node current runs down bundle of HIS then down small LBB to the small septal fascicle creating a small septal Q wave, then followed by massive positive deflection creating the R wave followed by a deep negative deflection
- QRS Interval duration of QRS usually last 0.06-0.1 seconds

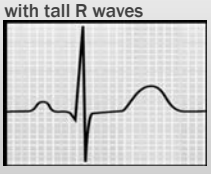
Normal QRS morphology in different leads



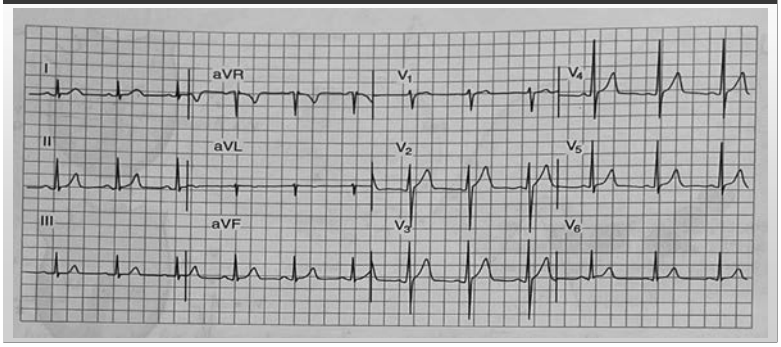
T Wave



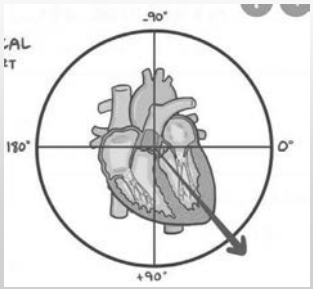
- Remember Ventricular repolarization so regains electrical positive charges
- Most T waves positive in leads with tall R waves



Normal EKG

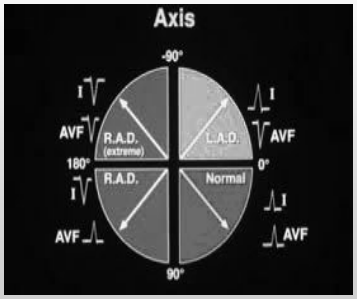


Electrical Axis



- Our normal electrical axis goes in the vector direction to the left lower quadrant from 0 degree to +90 degrees.
- To help determine axis on EKG use lead I (0 degrees) and aVF (+90)
  - Positive QRS in Lead I lets us know vector is between -90 and +90 degrees
  - Positive QRS in aVR lets u know vector is between 0 to +180.
  - To find specifically find the biphasic then plot perpendicular to it

4 different axis



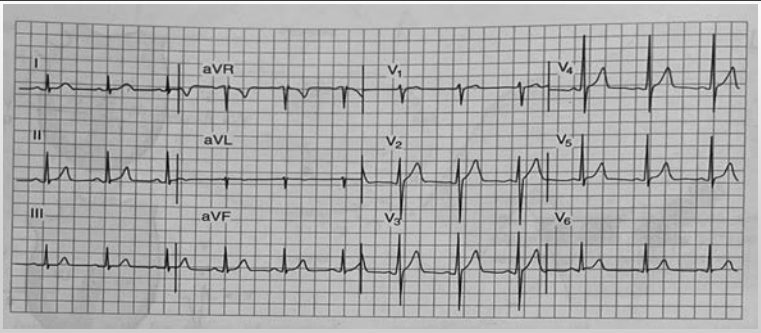
- Left axis deviation:
  - Lead I is positive and aVF is negative
  - From 0 to -90 degrees
- Right axis deviation
  - Lead I is negative and aVF is positive
  - From +90 to +180
- Extreme axis deviation
  - Lead I and aVF negative
  - From +180 to -90

Axis table

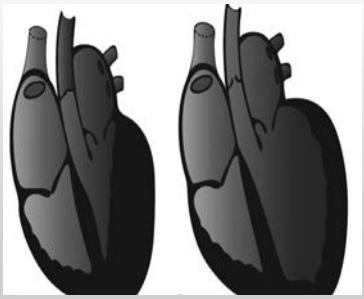
QRS Deflection		Axis
Lead I	Lead aVF	
+	+	Normal
+	-	LAD
-	+	RAD
-	-	Extreme Axis

- Remember the medical student thumb technique
- Let's try on the next slide

What axis is it?

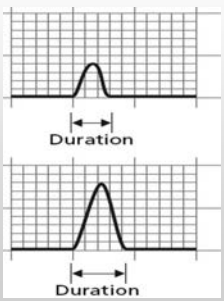


Enlargement and Hypertrophy



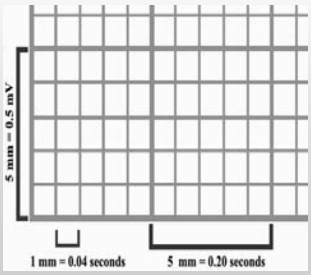
- Enlargement gives reference to dilatation of any of the 4 chambers of the heart
  - Usually referred more to Atrial enlargement but can also be ventricle
  - Valvular disease can be major causes such as mitral and aortic regurgitation
- Hypertrophy in EKGs gives reference to the increase in mass of a ventricle
  - Having to work out harder increasing in size due to causes such as HTN and Aortic stenosis

EKG changes hypertrophy or enlargement



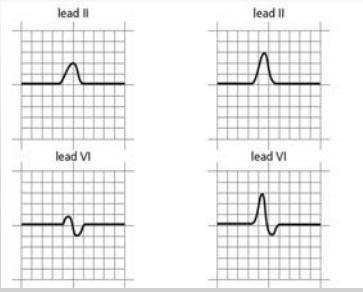
- Longer depolarization period leads to an increases in duration (width)
- Increase in voltage from increase mass leads to an increase in amplitude (height)
- Increase in size can cause a shift in the electrical axis vectors

Atrial enlargement



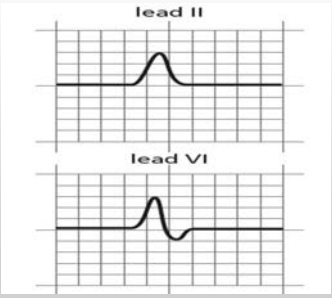
- P wave should be less than 0.12 seconds (3 small boxes) in duration and voltage should be less than 2.5 mm (2.5 small boxes up)

Atrial enlargement



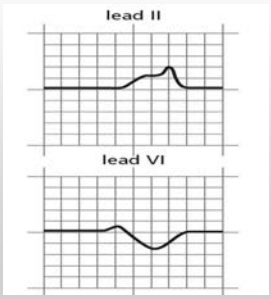
- Leads that assist with determining enlargement are lead II and V1
  - Lead II is parallel to the vector of atrium
  - V1 is perpendicular and shows separation of right and left biphasic p wave

Right atrial enlargement



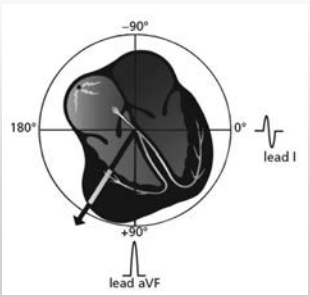
- With Right Atrial Enlargement duration does not increase but height does, the duration does not change because the second part of the p wave is left atrium in source
- Tallest P wave now in lead aVF or lead III no longer II
- Best view is in lead II and V1
- Criteria needs to be P waves with height >2.5 mm in a inferior lead (II, III, aVF)

Left Atrial Enlargement



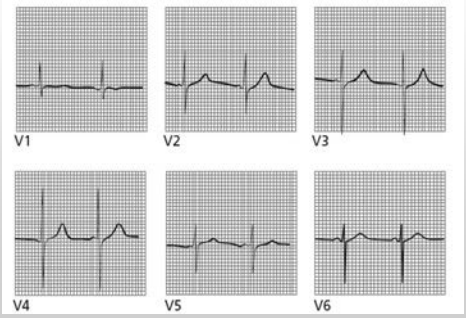
- Left Atrial Enlargement involves the 2nd part of the P wave causing an increase in height and width
- Criteria is
  - in V1 the second part of the P wave must have a drop >1 mm below the baseline
  - also the left atrial portion of the P wave must have a width >0.04 seconds

Right Ventricular Hypertrophy



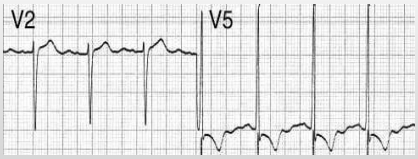
- Usually seen in Right axis deviation
  - Lead I negative aVF positive
- Common causes of RVH Pulmonary disease or Congenital heart disease

RVH



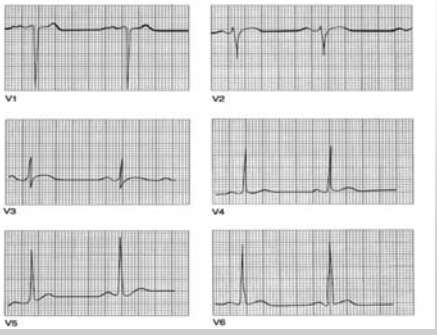
- Lead I is mildly negative
- Precordial leads V1-V5 mildly reversed
- Lead V1 now R waver is taller than the S wave
- Lead V6 now S wave is taller than the R wave

Left Ventricular Hypertrophy



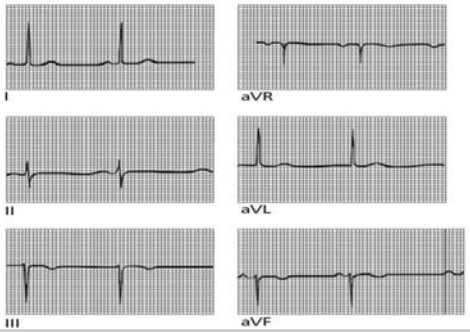
- Common causes HTN, aortic stenosis
- Usually Left axis deviation noted but not diagnostic needs to meet criteria for true diagnosis, remember Lead I positive and aVF negative; -90 to 0 degrees
- Key features are:
  - enlarged R wave height in leads over the left ventricle
  - S wave height enlarged in leads over the right ventricle

Left Ventricular Hypertrophy Criteria



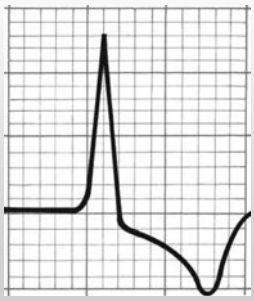
- Precordial Leads Criteria
  - R wave height in lead V5 or V6 plus the S wave height in V1 or V2 >35 mm
  - R wave height in lead V5 >26 mm
  - R wave height in lead V6 >20 mm
  - R wave height in V6 > R wave height in V5

LVH Criteria



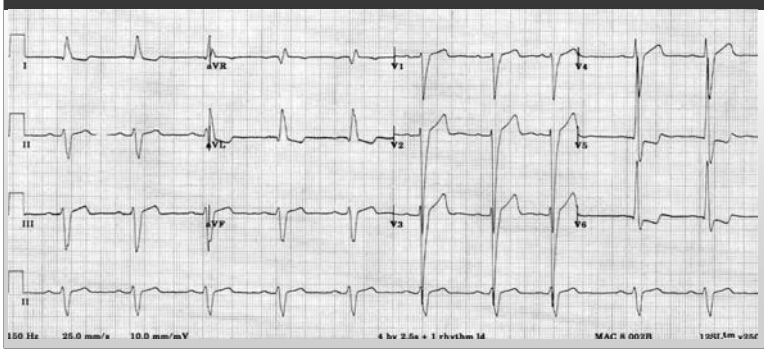
- Limb Leads Criteria
  - R wave height in lead aVL >11 mm
  - R wave height in lead aVF >20 mm
  - R wave height in lead I > 13 mm
  - R wave height in lead I plus the S wave height in lead III > 25 mm

Ventricular secondary effects



- Know as secondary repolarization abnormalities
  - 1) Downslopping ST segment depression
  - 2) T wave inversion
  - Mechanisms of action theories to be due to strain
- If seen most common in
  - RVH in V1 and V2
  - LVH in I, aVL, V5, and V6

LVH putting limb and precordial leads together



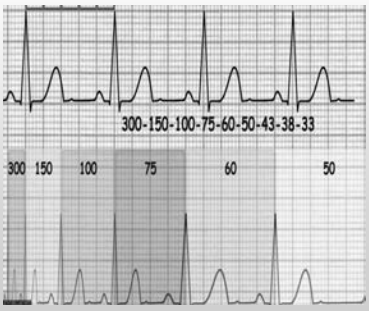
Arrhythmias

- A disturbance in rate, regularity, site of origin, or conduction
- Common causes
  - Hypoxia: lung disease, PE
  - Ischemia\irritability: myocarditis
  - Sympathetic stimulation: hyperthyroid, CHF, CNS, exercise
  - Drugs
  - Electrolyte disturbance: K, Ca, Mg
  - Bradycardia: bradytachycardia: SSS
  - Stretch: enlargement and hypertrophy, CHF, Valve disease

Before we look at different types lets first learn rate

- Remember
- 1 small square = 1 mm = 0.04 sec
- 1 large square = 5 mm = 0.2 sec
- 5 large square = 25 mm = 1 second

Calculate the Rate

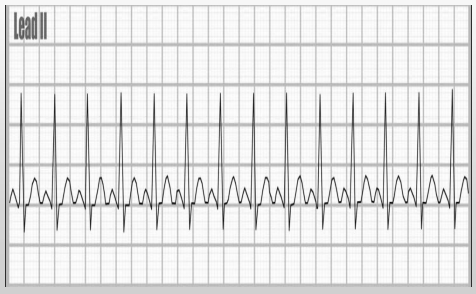


- Quick 3 step method to determine heart rate
  - Locate a R wave close to a darker line
  - Count the large squares until the next R wave
  - Quick ways is to divide 300 by the number of big boxes
    - So if 1 box is the R to R = 300 bpm
    - …. If 2 boxes the R to R = 150 bpm
    - …. If 3 boxes the R to R = 100 bpm
    - …. If 4 boxes the R to R = 75 bpm
    - …. If 5 boxes the R to R = 60 bpm
    - …. If 6 boxes the R to R = 50 bpm

Types of Arrhythmias

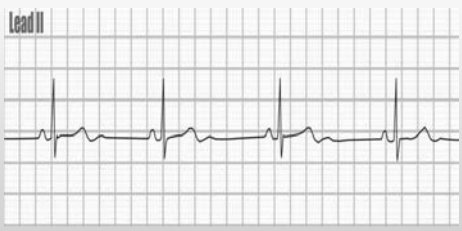
1. Sinus arrhytmias of sinus orgin: start with depolarization of the SA node but either to slow or fast or irregular
2. Ectopic rhythm: electrical current arising from other foci than the SA node
3. Reentrant arrhythmias: Trapped electrical current within a circuit, it's shape and track is made by an anatomic or electrical anomaly
4. Conduction blocks: starts at the SA node and has "road blocks" along the normal pathway
5. Prexcitation syndromes: Electrical current uses a accessory pathway that bypasses the normal electrical direction, a "short cut"

Arrythmias of Sinus Origin, Sinus Tachycardia



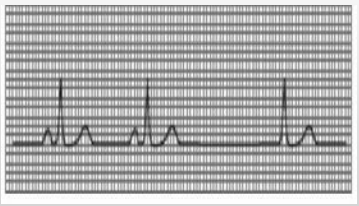
- Sinus tachycardia
  - Exercise, stimulants
  - CHF
  - Lung disease
  - Hyperthyroid
  - HR > 100 bpm for Cardiologist >110 bpm
- Inhalation = increases HR

Bradycardia



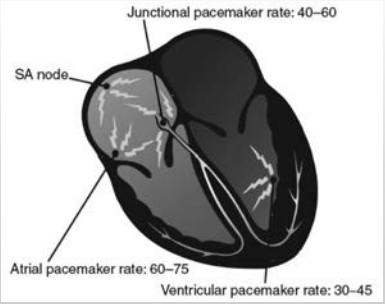
- Sinus Bradycardia
  - Medications: beta blockers, Calcium channel blockers, opioids
  - Athletes
  - HR <60 BPM
- Expiration = slows HR

Sinus arrest, Asystole then escape beats



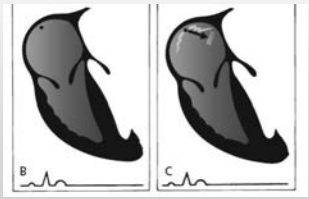
- When the SA the nodes stops sinus arrest occurs, prolonged sinus arrest with no other electrical activity occurs it is called Asystole
- During sinus arrest other myocardial cells can fire and act similar to pacemaker cells and create escape beats which rescue the pathway
- In example notice no P wave in the junctional escape beat

Nonsinus Pacemakers



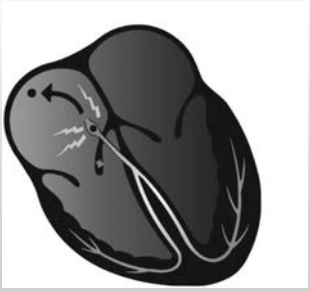
- Atrial pacemaker cells have a rate of 60-75 bpm
- Pacer cells around the AV node are called junctional pacemakers which fire at 40-60 bpm
- Ventricular pacer cells fire at 30-45 bpm
- Any of these can assist when the SA node is not firing adequately, the most common helper are the junctional pacers
  - These escape beats (will have no P wave) but retrograde P waves may follow

Sinus arrest vs. Sinus block



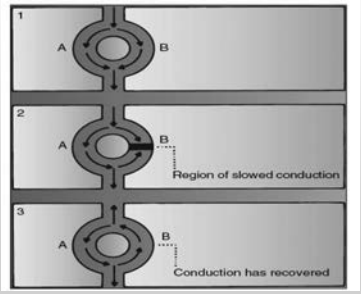
- Sinus arrest
  - there is a malfunction of the sinus mechanism to fire its current
  - No electrical activity
- Sinus exit block
  - Failure of current to leave the SA node and into the atria
  - Electrical activity

Ectopic Rhythms



- Rhythms that originate from other areas of the atria non SA node
- They can be single or sustained beats
- Formed by intrinsic pacemaker electroactivity at a single foci or roaming one
  - Can be enhanced by stimulants, digitalis toxicity, beta agonist, caffeine, alcohol, illicit drugs

Reentrant Rhythms

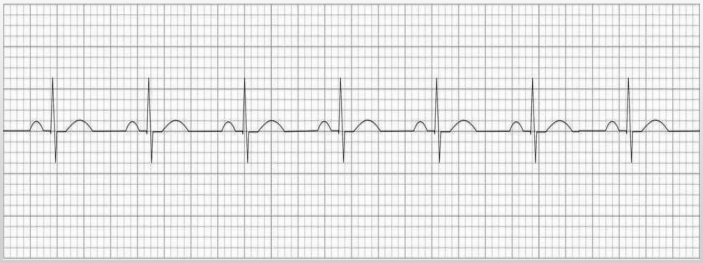


- Once again a current formation is originated from non SA node and takes over the conduction pathway; a problem of impulse transmission
- 1) is normal in our diagram
- 2) there is slowed conduction by ischemia or fibrosis that causes a delay which throws off the cycle causing to pathway A to now circle back around
- 3) a new reentrant conduction is now formed and overrides SA node flow

The big 4 questions when analyzing a EKG

1. Are P waves present?
   - Check Lead II and aVR for positive p waves
   - If yes then origin from the atria
   - If no P waves; then it arises below the atria in the AV node or ventricles
   - If abnormal p wave location
     - Think retrograde P waves
2. Are the QRS complexes narrow or Wide
   - <0.12 seconds or > 0.12 seconds
   - Narrow QRS means normal current flow a wide QRS means origin is in the ventricles moving slower and causing a longer duration
3. What is the relationship between the P waves and the QRS Complexes?
   - Does P wave always precede? Are they 1:1, sinus or atrial origin?
4. Is the Rhythm Regular or Irregular?
   - We will get to this in the next slides

Let try those 4 questions out

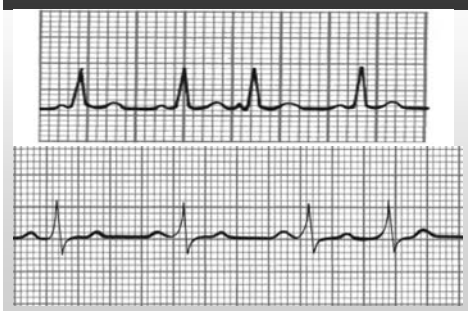


SVT Arrhythmias

- Premature Atrial Contractions (PACs)
- AV Nodal Reentrant Tachycardia (Paroxysmal SVT)
- Atrial Fibrillation
- Atrial Flutter
- Multifocal Atrial Tachycardia
- Paroxysmal Atrial Tachycardia (Ectopic atrial tachycardia)
- AV reciprocating tachycardia

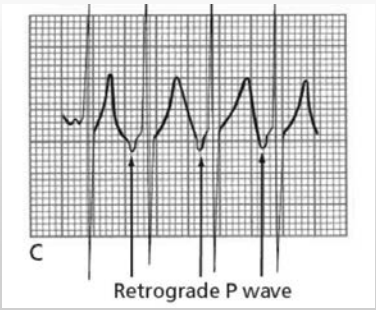
- Originate in the atria or AV node
- Can be 1 beat or sustained, can last second to a lifetime
- Look for P waves in Lead II or V1

PACs and Junctional premature beats



- PACs
  - Origin is the atria
  - Faster P wave
  - Different shaped from prior P waves
- Junctional premature beats
  - Origin near AV node
  - Skipped P wave
  - Appears similar to junctional escape beat but however these occur earlier and escape beats occur later
- They both conducted normally to the ventricles and have narrow QRS complexes

AV Nodal Reentrant Tachycardia (Paroxysmal SVT)

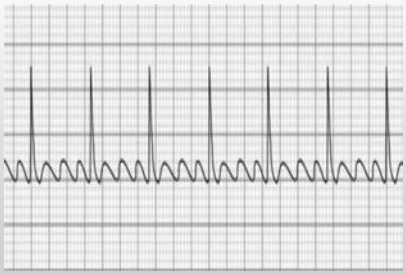


- Common
- Rapid onset, can be set off by a PAC or Junctional premature beat
- Seen in healthy hearts
- Regular rhythm, rate 150-250 bpm
- As the name implies has a reentrant pathway track within the AV node
- Lead II or III has retrograde P wave
  - Pseudo R' in lead V1 that reflects a superimposed retrograde P wave in QRS
- Narrow QRS

Carotid massage effects

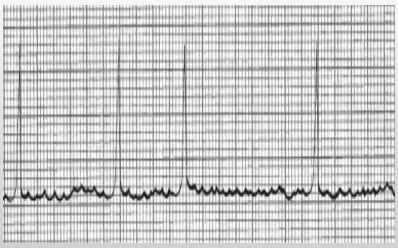
- Right carotid stimulates the SA node thru vagal input
- Left carotid stimulates the AV node
- Can stop or slow down reentrant currents
- However caution if you do choose to do it

Atrial Flutter



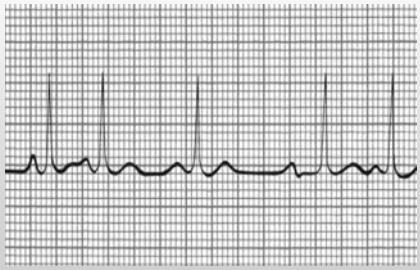
- Atrial origin, famous sawtooth appearance
- Regular rhythm with rate of 250-350 bpm
- Created by a reentrant pathway that cycles around the annulus of the tricuspid dvalce
- Depolarization in atrium is so fast that P waves demarcated by the baseline are not visualized, instead a continues up and down flutter wave is seen, usually best seen in Lead II and III
- The AV node cannot process the rapid flow from the atrium and unable to keep up with QRS leading to what is a AV block
- A. Flutter usually has a 2:1 AV block (2 flutter waves per 1 QRS)
- Carotid massage makes it worst increase to up to 5:1

Atrial Fibrillation



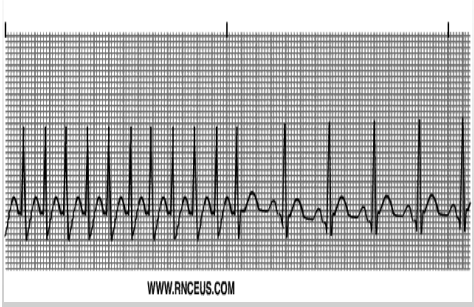
- Erratic atrial electrical activity, can fire up to 350-500 currents to the AV node which cannot process all and with an average rate of 120-180 bpm
- Caused by several small reentrant pathways swirling around
- No real P waves
- Baseline will appear nearly flat with mild fibrillation "undulating" waves
- Irregularly irregular appearance of QRS without P waves is key

Multifocal Atrial Tachycardia and Wandering Atrial Pacemaker



- Irregular, rate of 100-200 bpm, when rate if under 100 its called WAP
- Unsystematic discharges of multiple different ectopic atrial foci
- Not affect by Carotid massage
- Has clear P waves before QRS unlike A fib
- Criteria must have 3 different P wave morphologies

Paroxysmal Atrial Tachycardia

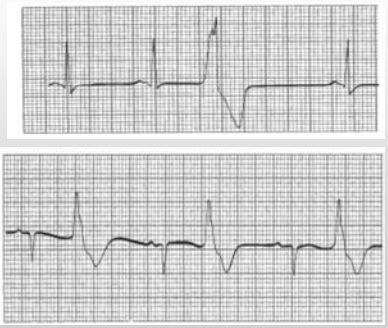


- Regular rhythm with rate of 100-200 bpm
- From a reentrant pathway within the atria or from increased automaticity of ectopic atrial focus
- Has a "warm up" and "cool down" state that helps distuguish between PSVT
- No affected by Carotid massage

Ventricular Arrythmias

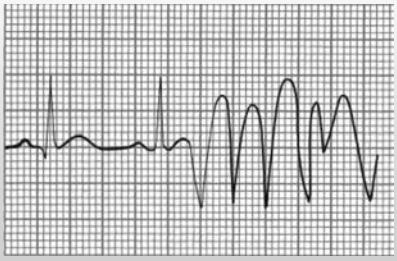
- Premature Ventricular Contractions
- V Tachycardia
- V Fibrillation
- Accelerated Idioventricular Rhythm
- Torsade de Pointes
- Arise the below the AV node
- Mild to life threatening

PVCs



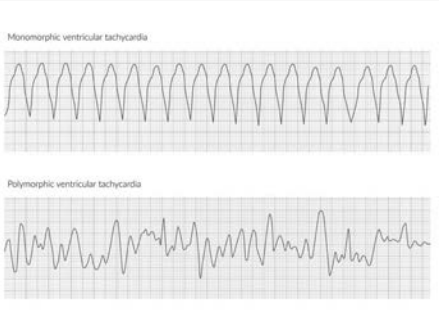
- Most common of the Ventricular arrythmias
- Wide and abnormal QRS due to VD does not follow the traditional Ventricular circuit
- QRS >0.12 seconds (3 smalls boxes) in most leads
- May have retrograde P waves or no P waves
- usually has a pause before the next beat, if no pause called a "interpolated PVC"
- Commonly seen on their own
- Caution if seen after a MI which can trigger V tach or V Fib
- Bigeminy when 1:1 one normal sinus run and one PVC
  - Trigeminy is when 2:1 two normal sinus beats and one PVC

PVCs rule of malignancy



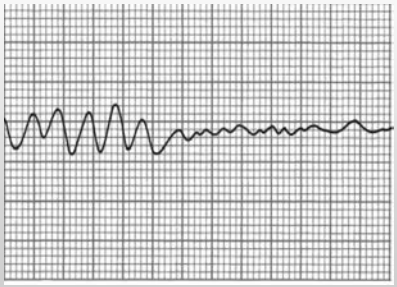
1. Recurrent PVCs
2. Consecutive run of PVCs of 3 or more
3. Different PVC morphologies
4. PVCs on T waves of prior cycle named ""R on T" phenomenon, very vulnerable time during VR and can cause V Tach to start
5. A PVC during a new MI

Ventricular Tachycardia



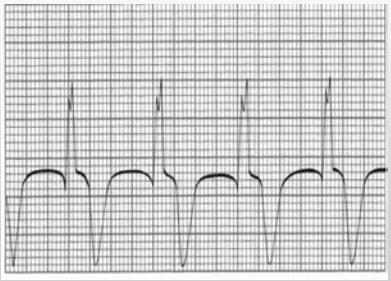
- A run of ≥3 consecutive PVCs
- Rate of 120-200 bpm
- Sustained if > 30 seconds
- Can be uniform or polymorphic
  - Uniform seen more in healed infarts or scarred myocardial tissue
  - Polymorphic seen in acute coronary ischemia, new infarct, electrolyte changes, prolonged QT interval

Ventricular Fibrillation



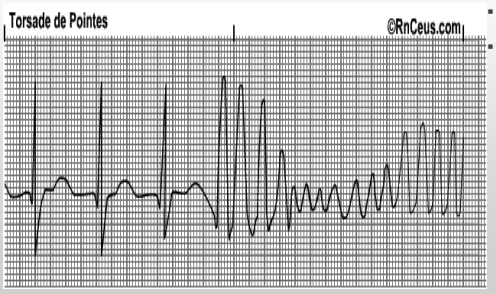
- Cardiac emergency can lead to sudden death
- Can be either (fine) smooth undulating or (coarse) spasmodic
- No clear cut QRS complexes
- No Cardiac output
- Causes
  - MI, Heart failure, hypoxemia, hypercapnia, shock, hypotension, electrolyte disturbance, stimulant drug overdose

Accelerated Idioventricular Rhythm



- Benign seen after new MI or after PCI
- Regular rhythm and rate of 50-100
- Likely ventricular escape focus
- Rarely sustained, when drops under 50 just idioventricular rhythm
- No P wave with wide QRS

Torsade de Pointe

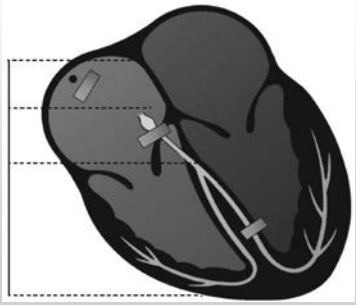


- "Twisting of the points"
- A derivative of V Tach with prolonged QT interval
  - Can be from congenital
  - Electrolyte disturbance Ca, Mg, K
  - Acute MI
  - Meds: SSRI, Tricyclics, antipsychotics, fluoroquinolones, Zofran, Azithromycin, …
  - PVC falling on T wave
  - Undulating around the baseline with change in height

Recap Supraventricular and Ventricular Arrhythmias

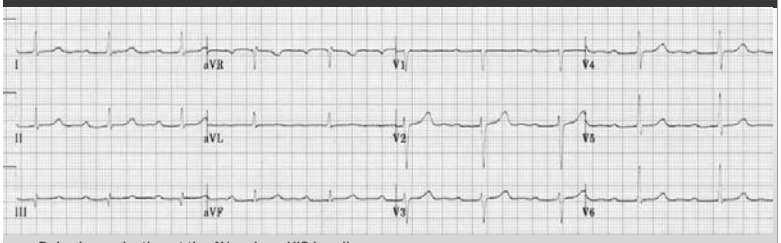
- Supraventricular
  - Narrow QRS <0.12 sec
  - Carotid massage may improve
- Ventricular
  - Wide QRS >0.12 sec
  - Not affected by Carotid massage

Conduction Blocks



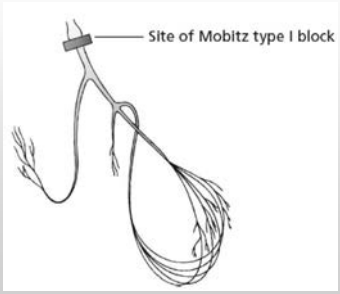
1. Sinus node block
   - Sinus exit block
     - SA node fires routinely but is blocked and not relayed by atrial tissue
     - Already discussed this earlier
2. AV block
   - A block between AV node and HIS bundle most common
3. Bundle branch block
   - Block at one ventricular branch or both or partial in left bundle

AV Blocks: First degree AV Block



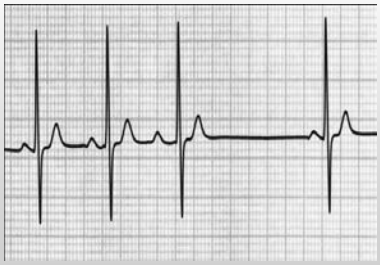
- Delay in conduction at the AV node or HIS bundle
  - More of a delay than a true block
- PR interval > 0.2 seconds (1 big box)
- Common and usually asymptomatic

AV Block: 2nd degree



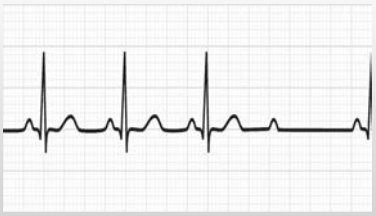
- AV node is not processing all atrial currents and will have a >1:1 P wave to QRS
- 2 type of 2nd degree block
  - Mobitz type 1 (Wenckebach)
  - Mobitz type 2

Mobitz Type I Wenckebach



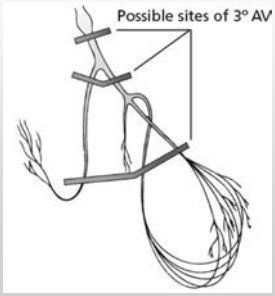
- Block within the AV node
- The block is variable and increasing with each proceeding impulse
- The new impulses encounter a longer delay in the AV node until a impulse fails to make it through the AV node creating 'dropped' QRS
- In other words PR interval that prolongs until a QRS is dropped
- Repeats this pattern

Mobitz Type 2



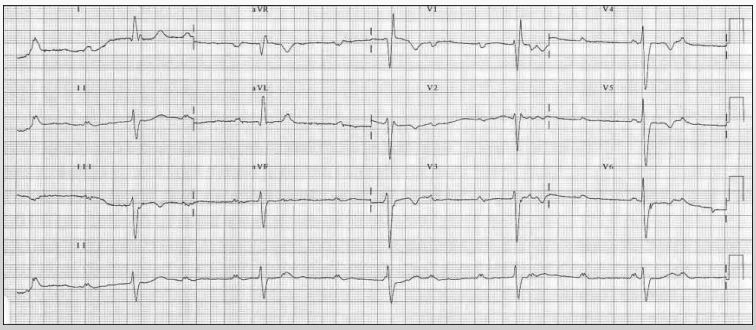
- Block is below the AV node in the HIS bundle or near it
- A "all or nothing" phenomenon
  - 2 or more normal cycles proceeded by a P wave with no QRS behind it
  - Inconsistent, has ratios of 2:1, 3:2 etc
- To differentiate from type 1 with Type 2 there is no PR interval lengthening
- More concerning than type 1

3 Degree AV Block

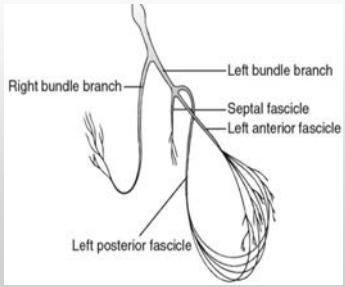


- No atrial current makes it to the ventricles
  - Ventricles react with escape beats with at rate of 30-45
- Complete heart block
  - Can be at the AV node or lower
- Now the atria is still beating at its 60-100 rate
- To diagnosis needs
  - AV dissociation = Atria and Ventricles beating independently with ventricles much slower rate than atria

3rd Degree AV Block

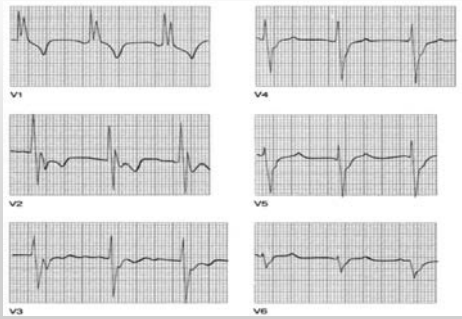


Bundle Branch Blocks



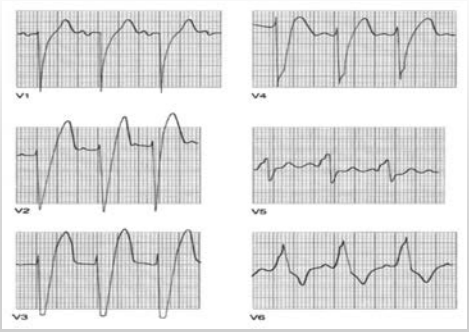
- Conduction delay or block thru the Right or Left bundle branches
- To help make diagnosis the QRS will have several changes
- Incomplete BBB
  - When LBBB or RBBB appear but QRS is 0.10 to 0.12 seconds

Right Bundle Branch Block



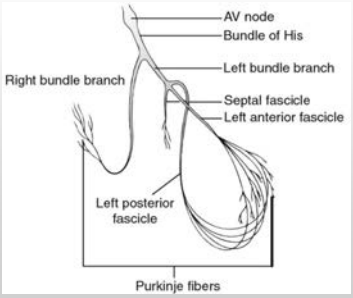
- Right ventricle depolarization is delayed and does not start until LV is nearly depolarized
- This delay in RV depolarization prolongs the total time for total VD creating >0.12 second QRS
- The wide QRS has a unique shape over the RV V1 and V2 unopposed after the LV has finished its cause a second R wave R prime making a rabbit ears look
- In the lateral leads it causes a reverse change of deep S waves

Left Bundle Branch Block



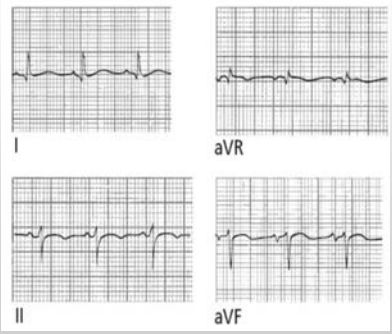
- LV depolarization is delayed
- QRS >0.12 seconds
- QRS over Lateral leads will have change in morphology with a lag in the rise of tall R waves that have notches on top and or broad
- QRS on RV leads will have broad S deep waves
- Ventricular hypertrophy cannot be diagnosed if BBB are present

Hemiblocks



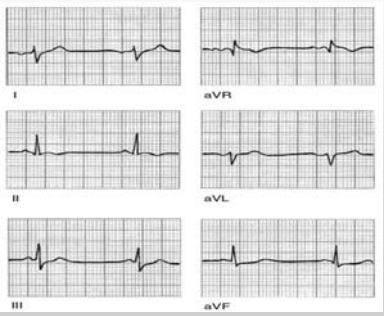
- Found on one of the Left bundle branch
- Left anterior or Left posterior
- Can affect the axis deviation
- Left ant. Fascicle lies superior and lateral to Left posterior fascicle
- QRS is not widen unlike BBB

Left anterior hemiblock



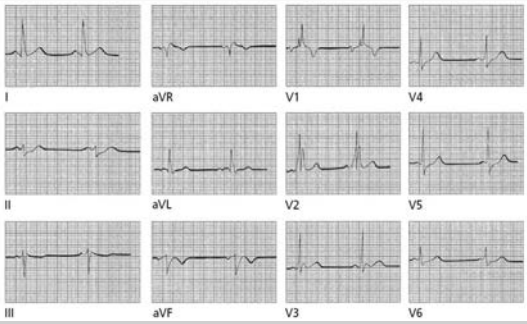
- The pathway down LAF is blocked and the current from LPF wraps around to assist
- Causing a Left axis deviation positive in lead I and negative in aVF
- Tall positive R waves are seen in left lateral leads from this hemiblock

Left Posterior Hemiblock



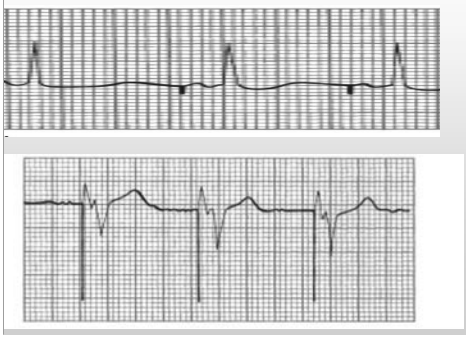
- Reverse from LAF
- The pathway down LPF is blocked and the current from LAF wraps around to assist
- Causing a Right axis deviation positive in aVF and negative in lead I
- Tall positive R waves are seen in inferior leads and deep S wave in Lateral leads

Bifascicular blocks



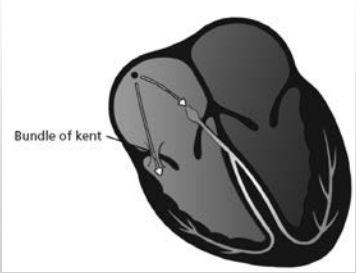
- RBBB and a hemiblock either LAF or LPF
- With a RBBB there will be a QRS >0.12 seconds, RSR' in V1, V2
  - If LAF will have Left axis deviation
  - If LPF will have Right axis deviation

Pacemakers on EKGs



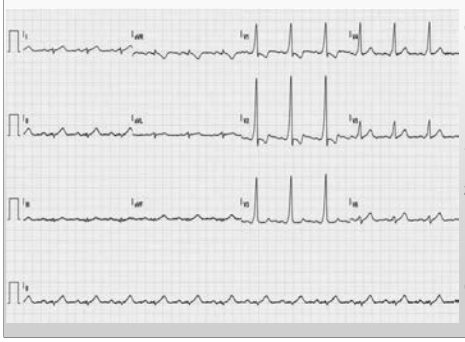
1. Atrial Pacemaker
   - Pacer spike followed by a P wave then normal PR interval and QRS
2. Ventricular Pacemaker
   - Bizarre and wide QRS similar to a PVC
   - May see P wave
3. Dual Chamber
   - 2 spikes one before a P wave and one before a wide bizarre QRS

Preexcitation Syndromes



- There is usually a 0.1 second pause at the AV node
- in Preexcitation there is a accessory pathway that bypasses the AV node to reach the ventricles with no delay
  - In other words a "short cut"

Wolff-Parkinson-White

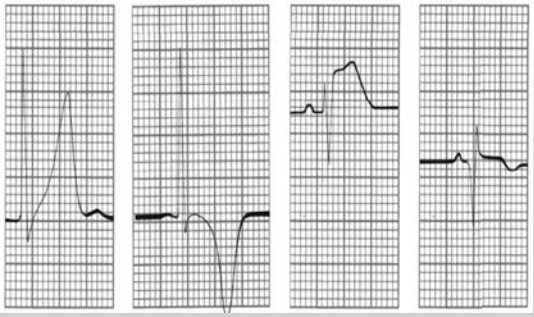


- This short cut pathway is a discreet conduction pathway that connect both atria and ventricles it can be Left atrium to left ventricle or right sided
  - Premature VD leads the criteria:

1. PR interval being shortened <0.12 seconds
2. QRS complex to widen >0.10 seconds including a "delta wave"
   - Wide because premature activation adds a upstroke wave to the normal QRS increasing its width overall

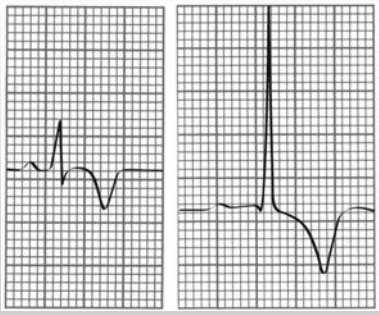
- Can occur with A. Fib and SVT

Myocardia Ischemia and Infarction



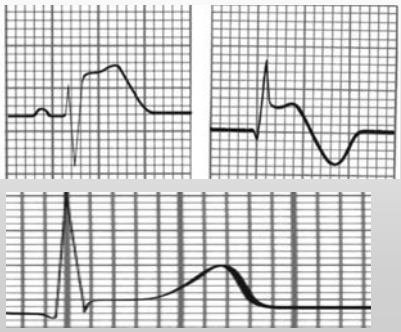
- 3 general EKG changes noted on a ST elevated Myocardial Infarction (STEMI)
  1. T waves peak then invert
  2. ST segment elevation
  3. Q waves appearing

Hyperacute T waves or Inverted T waves



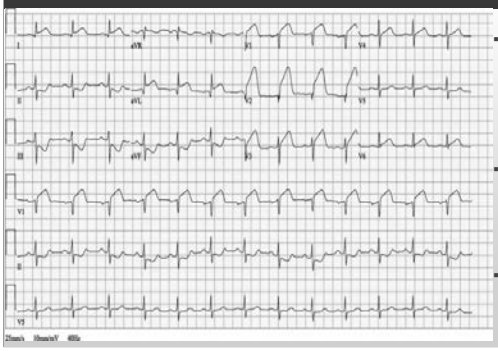
- Reflection of ischemia = lack of blood flow, initially peak then several hours later invert
- They can switch back to normal in cases of ischemia, in cases of infarct they stay inverted for month to years
- TWI can been seen with BBB and hyperventilation
- In cases of ischemia are usually symmetrical
- In cases with prior known TWI they can revert back to normal in ischemia or new infarct
- Normal in some athletes isolated to V1, V2, V3 or lead III

ST segment



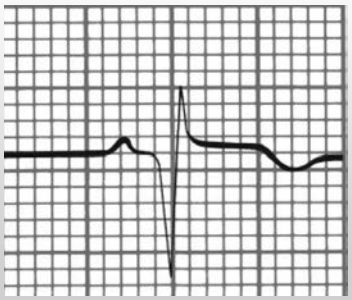
- 2nd change that happens acutely in a STEMI
- Significant amount of injury if this is present
- Us the TP segment to reference the ST segment
- Return to baseline in several hours, persistent can be from a ventricular aneurysm
- Junction point elevation common in healthy young folks in V1, V2, V3 has a small notch or slur downsloping of the R wave
- True ischemia ST is bowed upward

ST Elevation Criteria



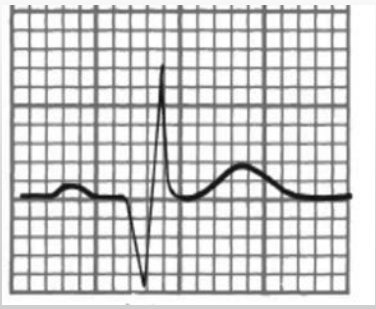
- ST elevation In V2 V3
  - Men <40 yo; >2.5 mm increase
  - Men >40 yo; >2.0 mm increase
  - Women >1.5 mm increase
- ST elevation in other leads
  - Men <40 yo; >1 mm increase
  - Men >40 yo; >1 mm increase
  - Women >1 mm increase
- ST elevation must be in at least 2 leads

Q Waves



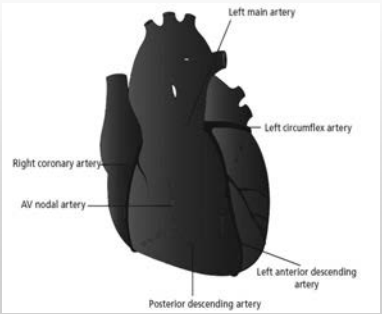
- New Q waves indicate irreversible damage
- Diagnostic for an MI
- Can be seen several hours after a STEMI, usually after ST elevation has gone down
- Can have them for life
- When a area of myocardium is permanently damaged that area will have a negative deflection creating the Q wave
- Leads distant from the infarcted tissue can have ST segment depression
- Ex. Lead III should be positive

Q wave criteria



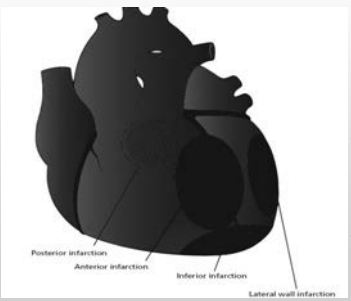
- Ischemia Q Waves are wide and deep
  1. Must have >0.04 seconds
  2. Depth must be 25% of the height of the R wave of the same QRS
- Normal Q waves
  1. Small in lateral I, aVL, V5, V6
  2. Q wave in only V3 no other leads

Locating the infarct



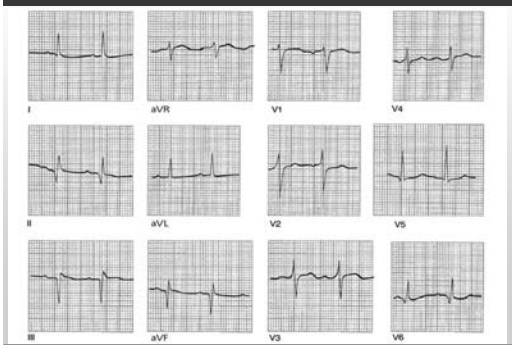
- Right coronary artery passes between the Right atrium and Right ventricle then moves to the posterior surface of the heart
  - Descending branch feeds the AV node
- Left main artery splits into the Left anterior descending artery and left circumflex artery
  - LAD goes between the 2 ventricles and feeds the anterior wall of the heart and most of the Interventricular septum
- Circumflex artery goes between the Left atrium and Left ventricle and lateral wall of the left ventricle

Locations of Infarcts



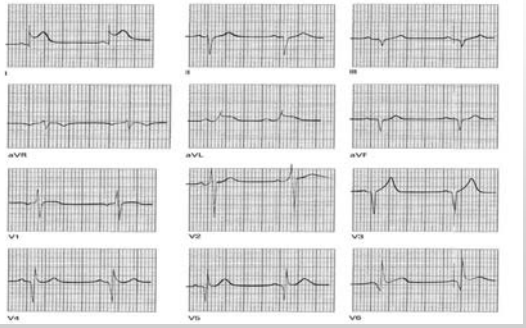
1. Inferior infarct: diaphragm surface of the heart caused by occlusion of RCA or descending branch
   - Inferior leads: III, III, aVF
   - Reciprocal in Anterior lateral leads
2. Lateral infarct: left lateral wall of heart, occlusion of LCA
   - Left lateral leads: I, aVL, V5, V6
   - Reciprocal in inferior leads
3. Anterior infarct: anterior surface of the Left Ventricle, occlusion of the LAD
   - Any precordial lead can change V1-V6
   - If occlusion is of Left main artery can cause a anterolateral infarct with precordial and lead I and aVL
   - Reciprocal is inferior leads
4. Posterior infarct: posterior surface of the heart, occlusion of RCA
   - usually occurs with inferior or lateral infarcts
   - to diagnosis needs to see in reciprocal changes in anterior leads with tall R wave and ST depression in leads V1-V3
   - Mirrors image of a anterior infarct

Inferior MI



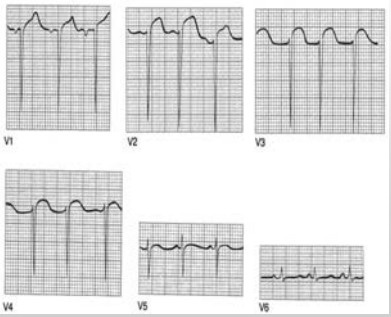
- Inferior infarct: diaphragm surface of the heart caused by occlusion of RCA or descending branch
  - Inferior leads: III, III, aVF
  - Reciprocal in Anterior lateral leads

Lateral MI



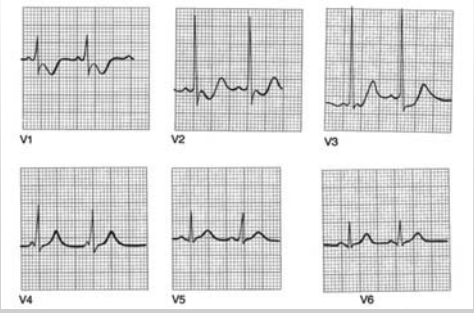
- Lateral infarct: left lateral wall of heart, occlusion of LCA
  - Left lateral leads: I, aVL, V5, V6
  - Reciprocal in inferior leads

Anterior MI



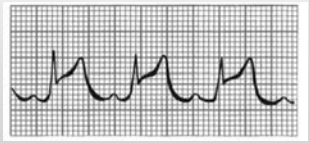
- Anterior infarct: anterior surface of the Left Ventricle, occlusion of the LAD
  - Any precordial lead can change V1-V6
  - If occlusion is of Left main artery can cause a anterolateral infarct with precordial and lead I and aVL
  - Poor R wave progression
  - Reciprocal is inferior leads

Posterior MI



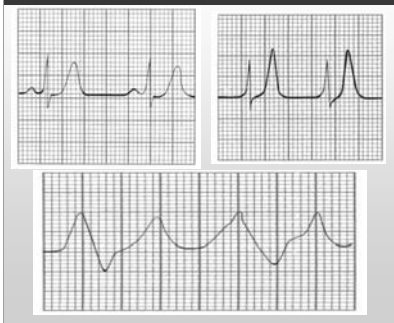
- Posterior infarct: posterior surface of the heart, occlusion of RCA
  - usually occurs with inferior or lateral infarcts
  - to diagnosis needs to see in reciprocal changes in anterior leads with tall R wave and ST depression in leads V1-V3
  - Mirrors image of a anterior infarct

Misc. MI related info



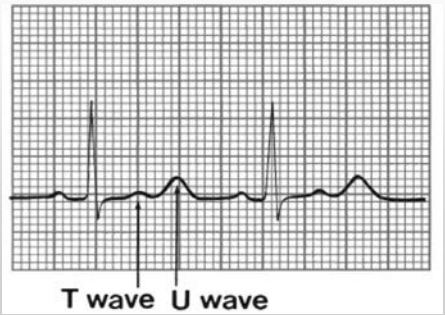
- Right Ventricle MI
  - Inferior MI with V1 changes
  - Preload sensitive caution with nitrate can cause severe hypotension
- New LBBB
  - Treat as a new MI, remember ischemia thought to play a role in LBBB
- Prinzmetal Angina
  - Coronary vasospasms
  - ST elevation quickly reversible with nitroglycerin

Electrolyte Disturbances



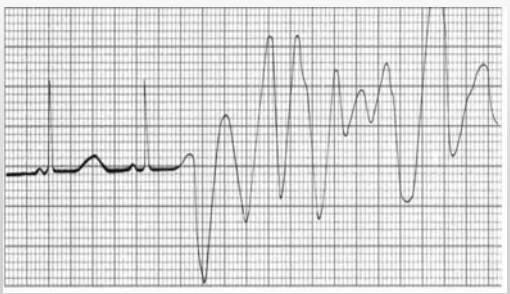
- Hyperkalemia
  - Increase in Potassium initially causes a increase in T wave height nearly all leads distinguishing from Mis
  - If potassium continues to increase it will prolong the PR interval and flatten the P waves
  - The QRS will widen and merge with the T waves if potassium worsens creating a sine wave pattern
    - Hyperkalemia distinguished from other wide QRS with right axis deviation
  - Risk of Ventricular fibrillation

Hypokalemia



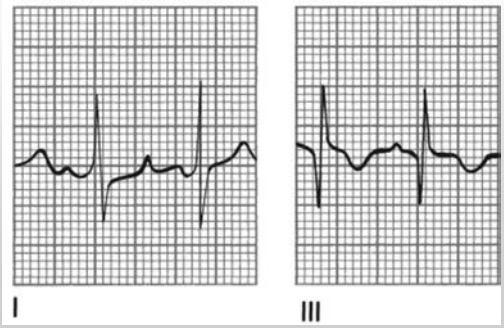
- Can cause
  - ST depression
  - Flatten the T waves and prolonged QT interval
  - Appearance of U waves, seen after the T wave
  - Severe hypokalemia can lead to ST elevation, SVT, and V Tach

Calcium changes the QT interval



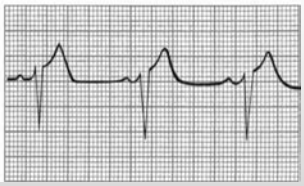
- Hypocalcemia
  - Prolongs the QT interval
- Hypercalcemia
  - Shortens the QT interval
- Torsade de Pointes
  - Prolonged QT interval

Acute PE



- Massive PE can cause
  - RVH due to a dilated ventricle
  - RBBB
  - Large S waves in lead I and deep Q waves in lead III called the S1Q3 pattern
  - TWI in V1, V2
  - Seen with A. Fib and Sinus tachycardia

Acute Pericarditis



- ST elevation and flat T waves or TWI seen throughout all leads
- STs are upward concave (saddle shaped)
- No Q waves seen
- If effusion will cause
  - Decreased voltage on EKG
  - Electrical alternans
    - Change in electrical axis

Citations

- Thaler, Malcolm. The Only EKG Book You'll Ever Need. Wolters Kluwer Health, 2017. [Wolters Kluwer].
- Loscalzo, Joseph. Harrison's Cardiovascular Medicine. McGrawHill Medical, 2010.

Thank you

- Contact info: John.Villasenor@optum.com

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

Anti-Obesity Medications

Presented by:
Justine Suba-Cohen, DO, MHS

Approved for 1.0 Prescribed CME

Saturday, July 31, 2021
10:15—11:15am

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
775-826-5100 • www.NVAFP.com

NOTES

Anti-Obesity Medications

Justine Suba-Cohen, DO, MHS
NAFP Summer CME Meeting 2021

INTRODUCTION

DISCLOSURES

- None

OBJECTIVES

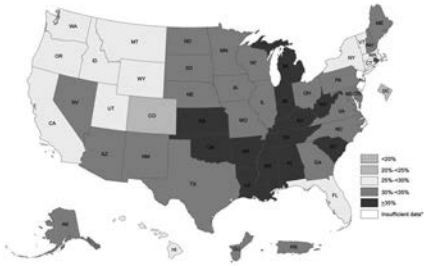
- Define obesity and its prevalence
- Discuss management of obesity and identify when to consider AOMs
- Assess different FDA-approved short term and long term AOMs
- Evaluate off-label medications
- Explore the role of Supplements
- Review common medications that affect weight

WHAT IS OBESITY?

"A **chronic, relapsing, multifactorial, neurobehavioral disease,** wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences."

-Obesity Medicine Association

HOW PREVALENT IS IT?



- The prevalence of obesity in adults was 42.4% in 2018.
- The prevalence of severe obesity in adults was 9.2%.
  - It was highest in women, non-Hispanic black adults, low income, and among adults aged 40–59.

https://www.cdc.gov/obesity/data/prevalence-maps.htm
https://www.cdc.gov/obesity/data/adult.html

HOW IS IT MEASURED?

WHO CLASSIFICATION OF WEIGHT STATUS	
WEIGHT STATUS	**BODY MASS INDEX (BMI), kg/m²**
Underweight	<18.5
Normal range	18.5 – 24.9
Overweight	25.0 – 29.9
Obese	≥ 30
Obese class I	30.0 – 34.9
Obese class II	35.0 – 39.9
Obese class III	≥ 40

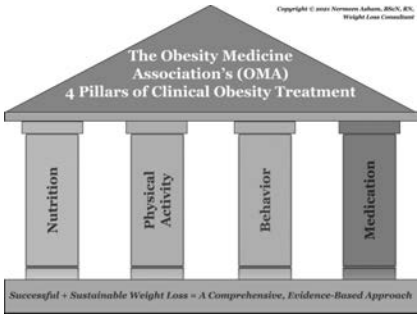
Health risk	WOMEN	MEN
Low Risk	below **31.5** inches	below **37** inches
Moderate Risk	**31.5 to 35*** inches	**37 to 40** inches
High Risk	**35*** inches or more	**40.2** inches or more

Table 6.14
General Body-fat Percentage Categories

Classification	Women (% fat)	Men (% fat)
Essential fat	10–13%	2–5%
Athletes	14–20%	6–13%
Fitness	21–24%	14–17%
Average	25–31%	18–24%
Obese	32% and higher	25% and higher

https://www.ncbi.nlm.nih.gov/books/NBK535456/figure/article-18425.image.f1/
https://www.whylexercise.com/waist-circumference.html
https://www.acefitness.org/education-and-resources/lifestyle/blog/112/what-are-the-guidelines-for-percentage-of-body-fat-loss/

HOW DO WE TREAT IT?



https://obesitymedicine.org/omas-four-pillars-the-bedrock-of-obesity-management-and-treatment/

FOUR PILLARS

- **Nutrition**: A deficit of at least 500 kcal per day. This is about 1200-1500 kcal for women and 1500-1800 kcal for men.
- **Behavioral Interventions:** 12-26 sessions a year, motivational interviewing, worksite interventions and incentive programs
- **Exercise:** 150-300 min of moderate-intensity activity or 75-160 min of vigorous activity per week
- **Medications**

MOTIVATIONAL INTERVIEWING

Table 2. Motivational Interviewing Techniques

Technique	Example	Rationale
Ask permission to discuss behavior-change topic	"Would it be okay if we talked about your weight today?"	When patient gives permission, he or she is more open to the conversation
Show empathy	"Losing weight is very challenging."	Aids in building rapport, particularly in difficult discussions
Scale motivation (0 = low to 10 = high)	"On a scale of 0 to 10, with 10 being the highest, how motivated are you to try to lose weight?"	Assesses motivation to change; if very low, the patient may not be ready for change; if high, additional intervention strategies may be successful
Scale confidence (0 = low to 10 = high)	"On a scale of 0 to 10, with 10 being the highest, how confident are you that you can lose weight?"	Identifies need for interventions to overcome obstacles
Inquire about the scores on above scales	"Why did you choose 3 instead of 2? What would help you move from 3 to 4?"	Furthers the conversation on thinking about behavior change
Use decisional balance technique (explore pros and cons of change vs. no change)	"What are the pros of losing weight?" "What are the pros of not losing weight?" "What are the cons of losing weight?" "What are the cons of not losing weight?"	Helps patient and physician understand barriers to and motivators for change
Listen for change talk and reinforce it; let the patient take ownership by generating ideas for change	Patient: "I think I could try to walk more." Physician: "That's a fantastic idea that will help you move toward your goal."	Provides encouragement and helps promote confidence in patients

Information from references 11 and 12.

Am Fam Physician. 2016 Sep 1;94(5):361-368.

WHEN SHOULD WE CONSIDER AOM MEDICATIONS?

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	References
BMI should be calculated for all patients 18 years and older, and those with obesity should be referred for intensive, multicomponent behavioral interventions.	B	2
Increased physical activity should be recommended for weight loss in combination with diet and behavioral modifications.	B	20
Physicians should consider medications for weight loss in patients with a BMI of 30 kg per m^2 or greater, or 27 kg per m^2 or greater who also have comorbidities and have unsuccessfully tried diet and lifestyle modification first.	C	26
Patients with a BMI of 40 kg per m^2 or greater and those with a BMI greater than 35 kg per m^2 who also have obesity-related comorbidities should be referred for consideration of bariatric surgery. Patients with a BMI greater than 30 kg per m^2 who also have obesity-related comorbidities may be candidates for adjustable gastric banding.	B	36

BMI = body mass index.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

Am Fam Physician. 2016 Sep 1;94(5):361-368.

WHAT ARE OUR TREATMENT GOALS?

- >=5% Weight loss = RESPONDERS
- Improvement in comorbid conditions/reduction of meds
- Improvement in eating behaviors
- Improvement in quality of life

SYMPATHOMIMETIC

- Approved for short term use = 12 weeks
- Schedule IV: Phentermine and Diethylpropion
- Schedule III: Benzphetamine and Phendimetrazine

PHENTERMINE

- SAFETY
  - CI in CVD, hyperthyroidism, glaucoma, pregnancy/nursing, h/o drug abuse
  - EKG prior to initiation?
- TOLERABILITY
  - Increases HR, BP, insomnia, dry mouth, constipation, and nervousness
- EFFECTIVENESS
  - 8% Weight loss
- PRICE
  - $10 GoodRx, usually covered by insurance
- SIMPLICITY
  - Start 37.5mg half a tablet in AM, add 2nd half to dosing regimen if needed
  - Also comes in 8mg (1/2 -1 tab qd-tid), 15mg, 30mg, 37.5mg capsules

CAN WE USE PHENTERMINE LONGER THAN 3 MONTHS?

Safety and Effectiveness of Longer-Term Phentermine Use: Clinical Outcomes from an Electronic Health Record Cohort.

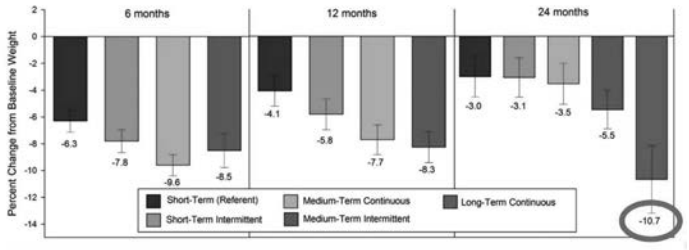
Lewis KH[1], Fischer H[2], Ard J[1], Barton L[2], Bessesen DH[3], Daley MF[4], Desai J[5], Fitzpatrick SL[6], Horberg M[7], Koebnick C[2], Oshiro C[8], Yamamoto A[2], Young DR[2], Arterburn DE[9]

WHO WAS INCLUDED IN THE STUDY?

- Mean BMI 38
- Responders = 3% weight loss at 12 weeks
- Classified into five groups
  - Short term (Referent)
  - Short term intermittent
  - Medium term continuous
  - Medium term intermittent
  - Long term continuous

Lewis et al. Obesity (2019) 27,591-602

WHAT DID LEWIS ET AL. FIND?



Lewis et al. Obesity (2019) 27,591-602

WHAT DID LEWIS ET AL. FIND?

- HEART RATE
  - No difference between groups at 24 months
- BLOOD PRESSURE
  - Systolic: Comparison groups had lower BP than Short Term group at 24 months
  - Diastolic: No differences in comparison groups at any time period
- INCIDENT OF CVD OR DEATH
  - 41 OF 13, 972 (0.3%) experienced an event
  - No qualifying CVD or deaths in long-term continuous group
  - No significant differences between groups

Lewis et al. Obesity (2019) 27,591-602

LONG TERM FDA APPROVED MEDICATIONS

Table 3. Medications Approved for the Long-Term Treatment of Obesity

Medication	Maintenance dosing	Mechanism of action	Weight loss relative to placebo	Study duration	Adverse effects	Contraindications*	Approved by U.S. Food and Drug Administration
Liraglutide (Saxenda)	0.6, 1.2, 1.8, 2.4, or 3 mg per day subcutaneously	Glucagon-like peptide-1 inhibitor	1.2 mg: 4.8 lb (2.1 kg) 1.8 mg: 6.2 lb (2.8 kg) 2.4 mg: 7.7 lb (3.5 kg) 3 mg: 9.7 to 13 lb (4.4 to 5.9 kg)	20 to 56 weeks	Abdominal pain, constipation, decreased appetite, diarrhea, dizziness, fatigue, headache, hypoglycemia, increased lipase levels, nausea, vomiting	Personal or family history of medullary thyroid cancer or multiple endocrine neoplasia type 2	2014
Lorcaserin (Belviq)	10 mg two times per day	5-HT2C receptor agonist	7 lb (3.2 kg)	One to two years	Back pain, constipation, cough, dizziness, dry mouth, fatigue, headache, hypoglycemia, nausea	—	2012
Naltrexone/bupropion (Contrave)	Two 8/90-mg tablets two times per day	Opioid antagonist/ aminoketone antidepressant	10 mg: 7.7 lb 52 mg: 10.4 to 10.8 lb (4.7 to 4.9 kg)	56 weeks	Constipation, diarrhea, dizziness, dry mouth, headache, insomnia, nausea, vomiting	Abrupt discontinuation of alcohol, benzodiazepines, barbiturates, or antiepileptics, anorexia nervosa, bulimia, long-term opioid use, seizure disorder, uncontrolled hypertension, use of a monoamine oxidase inhibitor within 14 days	2014
Orlistat (Xenical)	60 or 120 mg three times per day	Lipase inhibitor	60 mg: 5.5 lb (2.5 kg) 120 mg: 7.5 lb (3.4 kg)	One to four years	Fecal incontinence, fecal urgency, flatus, increased defecation, oily stool	Cholestasis, chronic malabsorption syndrome	1999
Phentermine/ topiramate (Qsymia)	7.5/46 mg per day 15/92 mg per day	Sympathomimetic/ antiepileptic	7.5/46 mg: 14.8 lb (6.7 kg) 15/92 mg: 19.6 lb (8.9 kg)	56 to 108 weeks	Constipation, dizziness, dry mouth, dysgeusia, insomnia, paresthesia	Glaucoma, hyperthyroidism, use of a monoamine oxidase inhibitor within 14 days	2012

*—All are contraindicated during pregnancy.
Information from references 21 through 32.

Am Fam Physician. 2016 Sep 1;94(5):361-368.

XX LORCASERIN (BELVIQ) XX

- SAFETY
  - **Withdrawn from market on February 2020,** safety clinical trial showed an increase in occurrence of cancer (pancreatic, colorectal, and lung cancers)
    - 462 (7.7%) patients were diagnosed with 520 primary cancers compared to the placebo group, in which 423 (7.1%) were diagnosed with 470 cancers.
    - FDA determined the potential cancer risk increases the longer people take Belviq
- TOLERABILITY
  - Was well tolerated, less than 1% discontinued treatment
- EFFECTIVENESS
  - 12.9lbs vs 5.6lbs from placebo, 47% lost 5% body weight vs 23% in placebo
- PRICE
  - $213 for 30 days
- SIMPLICITY
  - 10mg BID

ORLISTAT

- SAFETY
  - Severe liver injury has been reported (12 out of 13 reports occurred outside of US)
  - Fat soluble vitamins lowered, recommend fat-soluble vitamins supplementation
    - Due to decrease in vitamin K, warfarin dose may need to be lowered
  - Oxalate induced AKI has been reported
  - CI in pregnancy, chronic malabsorption, cholestasis, history of calcium oxalate stones
- TOLERABILITY
  - Common side effects include oily stool, flatus, fecal urgency and fecal incontinence
- EFFECTIVENESS
  - Average weight loss of 7.5lb and 5.5lb greater than placebo for 120mg and 60mg doses
- PRICE
  - $52 60mg for 120 Caps, $674 120mg 90 caps GoodRx
- SIMPLICITY
  - 60-120mg capsule tid during or up to one hour after a fat containing meal

PHENTERMINE/TOPIRAMATE (QSYMIA)

- SAFETY
  - In 2 56-week RCT with >3500 participants, patients taking phentermine/topiramate did not have more serious adverse events than those taking placebo except for 1% who developed nephrolithiasis
  - Does NOT increase symptoms of depression
  - Does NOT increase risk of arrhythmias, valve disease, or MI
  - Pregnancy category X - negative pregnancy test, dispensed by certified pharmacies
  - Topiramate can cause cleft lip/palate
  - CI in HTN, Glaucoma, MAOI in 14 days
- TOLERABILITY
  - Dry mouth (13-21% vs 2%), constipation (15-17% vs 6%), paresthesia (14-21% vs 2%), insomnia, irritability, and altered taste sensation (carbonated beverages taste flat/metallic)
  - Word recall goes away when medication is stopped

Am Fam Physician. 2014 Oct 15;90(8):576-578.

PHENTERMINE/TOPIRAMATE (QSYMIA)

- EFFECTIVENESS
  - 7.5/46mg 7-8% body weight, 15/92mg: 11% body weight
  - 5% weight loss: NNT 3-4 lower dose, NNT 2-3 higher dose
  - 10% weight loss, NNT 4-9 lower dose, NNT 3 higher dose
- PRICE
  - $186-$205 For 30 days on GoodRx, covered by a lot of commercial plans, savings card $70 for retail pharmacies or directly from MedVantx for $98
- SIMPLICITY
  - Patients are instructed to start at the lowest daily dosage (3.75/23 mg) and increase after 14 days to the target dosage (7.5/46 mg per day).
  - If patients do not lose 3% of their body weight after 12 weeks, the dosage should be increased to 11.25/69 mg once daily for another 14 days before increasing to the maximum dosage of 15/92 mg per day.
  - Patients should stop taking phentermine/topiramate after 12 weeks at this dose if they have not lost 5% of their body weight. Should be d/c gradually as abrupt withdrawal of topiramate can cause seizures.

Am Fam Physician. 2014 Oct 15;90(8):576-578.

NALTREXONE/BUPROPION (CONTRAVE)

- SAFETY
  - Labeled as increasing the risk of depression and suicidal behavior, based on Bupropion alone
  - May raise BP and HR, should not be used in uncontrolled HTN
  - Should not be prescribed to patients with a known seizure disorder or those already taking opioids
  - Pregnancy category X and should not be taken during breastfeeding
- TOLERABILITY
  - Nausea (30% vs 5%), constipation (15 vs 6%), headache (14 vs 9%), dizziness, insomnia and dry mouth (7-10%)
  - In the Contrave Obesity Research I, there was only a 50% completion rate over 56 weeks
- EFFECTIVENESS
  - Contrave Obesity Research I and II trials showed a weight reduction of 10.4-10.8lbs greater than placebo
  - Although mean weight loss was greater with Contrave, mean reductions in bP and HR were significantly greater in the placebo group

Am Fam Physician. 2015 Apr 15;91(8):554-556.

NALTREXONE/BUPROPION (CONTRAVE)

- PRICE
  - $297 for 120 tabs on GoodRx, $115 savings card, $99 mail order
- SIMPLICITY
  - Week 1: 1 tab in am
  - Week 2: 1 tab BID
  - Week 3: 2 tabs in AM, 1 tab in PM
  - Week 4: 2 tabs BID
  - If >=5% weight loss after 12 weeks, therapy should be continued for up to one year. Contrave has not been studied beyond 56 weeks.

Am Fam Physician. 2015 Apr 15;91(8):554-556.

LIRAGLUTIDE 3.0mg (SAXENDA)

- SAFETY
  - Acute gallbladder disease and cholecystitis can occur in patients rapidly losing weight, the rates are higher in patients using Liraglutide (NNH=100 vs 250)
  - Acute pancreatitis (0.3%)
  - Hypoglycemia: both severe (NNH=143) and symptomatic (NNH=10) hypoglycemia in type 2DM who are taking sulfonylureas and liraglutide
  - Contraindicated in patients with a family or personal history of medullary thyroid cancers and MEN type 2
  - Pregnancy Category X, should not be taken during breastfeeding
- TOLERABILITY
  - GI symptoms are common, 10% stop treatment because of adverse effects such as nausea (39%), diarrhea or constipation (20%), and vomiting (15%)
  - Can use antacid or H2 blockers

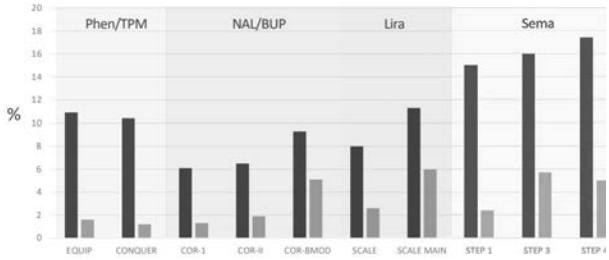
Am Fam Physician. 2016 Jul 15;94(2):161-166.

LIRAGLUTIDE 3.0mg (SAXENDA)

- EFFECTIVENESS
  - Liraglutide + lifestyle counseling for one year resulted in average of 8.9-13.3lbs greater weight loss
  - 5% body weight loss at 1 year NNT=2-3, 10% weight loss NNT 4-5
- PRICE
  - $1319 for 1 carton with 5 pens on GoodRx
  - As little as $25 per 30-day supply (1 box) with savings card, save up to $200 per 30-day supply (1 box) with cash
- SIMPLICITY
  - Starting dose of 0.6mg daily sc
  - Dosage increased weekly by 0.6mg (10 clicks) until target dose of 3mg

Am Fam Physician. 2016 Jul 15;94(2):161-166.

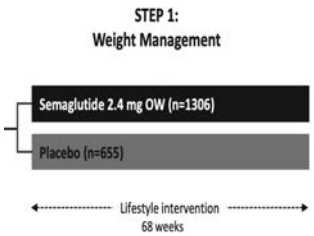
PERCENT WEIGHT LOSS COMPARISON



STEP TRIALS

SEMAGLUTIDE TREATMENT EFFECT IN PEOPLE WITH OBESITY

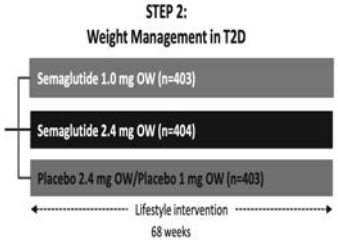
SEMAGLUTIDE 2.4mg



- 1961 Participants, published in NEJM
- 14.9% reduction in BW vs 2.4% reduction in placebo
- 86.4% of the Semaglutide group lost at least 5% BW
- 32.3% lost >=20% BW

Wilding JPH, et al. N Engl J Med 2021;384:989-1002

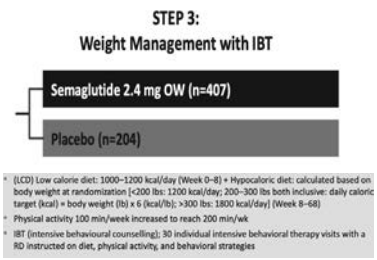
SEMAGLUTIDE 2.4mg



- 1210 Participants with T2DM and overweight or obesity, published in The Lancet
- Semaglutide 1.0mg vs 2.4mg
- 2.4mg = 9.64% reduction in BW
  - Better glycemic control
  - Reductions in cardiometabolic risk
  - Improved physical function
- 1.0mg = 6.99% reduction in BW
- Placebo = 3.42%

Davies M, et al. *Lancet.* 2021;397(10278):971-984.

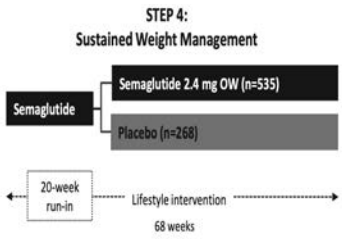
SEMAGLUTIDE 2.4mg



- 611 Participants, published in JAMA
- Semaglutide 2.4mg + Intensive Behavioral Therapy
- 16.0% reduction in BW vs 5.7% reduction in placebo
- 86.6% of the Semaglutide + IBT lost at least 5% in BW

Wadden TA, et al. JAMA. 2021;325(14):1403-1413

SEMAGLUTIDE 2.4mg



- 902 Participants, published in JAMA
- 2.4mg for 20 weeks, then assigned to receive either Semaglutide or placebo for the remaining 48 weeks
- Semaglutide for additional 48 weeks lost an additional 7.9% of BW for total weight loss of 17.4%
- Placebo for additional 48 weeks regained an average of 6.9% for total weight loss 5%

Rubino D, et al. JAMA. 2021. doi:10.1001/jama.2021.3224 (Epub)

GENESIS 100 (PLENITY)

- SAFETY
  - Approved as a device for BMI 25-40
  - No difference in overall incidence of adverse events (AEs) vs placebo
  - Contraindicated in pregnant or allergic, may alter absorption of medications
  - Avoid in esophageal anatomic anomalies, suspected stricture, complications from prior GI surgery
- TOLERABILITY
  - Most common side effects are diarrhea, distended abdomen, infrequent bowel movement, and flatulence
- EFFECTIVENESS
  - GLOW Study: 6 month, double blind RCT, 436 overweight and obese adults with or without T2DM
  - Average of 6% loss in BW vs 4% in placebo
  - 59% participants lost >=5% in BW

https://www.myplenity.com

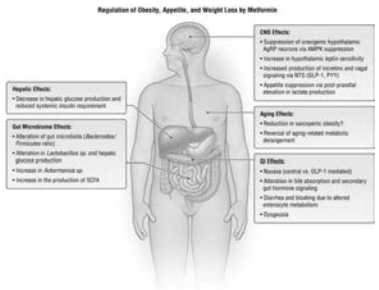
GENESIS 100 (PLENITY)

- PRICE
  - Not in pharmacies yet
- SIMPLICITY
  - 3 caps BID 20 minutes before lunch and dinner with 16 oz water

https://www.myplenity.com

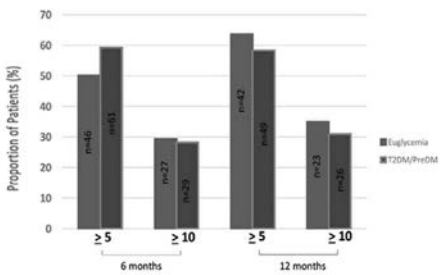
OFF-LABEL MEDICATIONS

METFORMIN



Yerevanian et al. Curr Obes Rep June 2019; 8(2):156-164. Igel et al. Curr Atheroscler Rep 2016;18:16.

METFORMIN



- ≥ 5% weight loss
  - 64% of euglycemic
  - 58% of PreDM/DM
- ≥ 10% weight loss
  - 34% vs. 31% in placebo

Chukir et al Obesity Res Clin Pract 2021;15:64-68

TOPIRAMATE

A randomized double-blind placebo-controlled study of the long-term efficacy and safety of topiramate in the treatment of obese subjects

J Wilding [1], L Van Gaal, A Rissanen, F Vercruysse, M Fitchet, OBES-002 Study Group

Affiliations + expand

PMID: 15486569 DOI: 10.1038/sj.ijo.0802783

- Three doses: 96, 192, 256mg/day + behavioral intervention
- At 60 weeks, subjects in the placebo group lost 1.7% of their baseline body weight, while subjects in the topiramate 96, 192, and 256 mg/day treatment groups lost 7.0, 9.1, and 9.7%, respectively
- 18% of subjects in the placebo arm lost >=5% in BW vs 54, 61, and 67% of subjects receiving topiramate 96, 192, and 256 mg/day, respectively
- 6% of subjects in placebo lost >/=10% in BW vs 29, 40, and 44% of subjects receiving topiramate 96, 192, and 256 mg/day, respectively
- Typical dose: 25-100mg daily

Evidence Summary and Clinical Stance for Individual Weight-Loss Supplements

SUPPLEMENT	EVIDENCE SUMMARY: PRODUCT QUALITY	PRODUCT SAFETY	PRODUCT EFFICACY	CLINICAL STANCE*
Psyllium[26]	Present‡	Present	Uncertain†	Caution and monitor
Pyruvate[35]	Uncertain	Uncertain	Uncertain¶	Caution and monitor
Spirulina (also known as blue-green algae)	Uncertain	Uncertain	Absent§§	Discourage
St. John's wort	Uncertain	Uncertain	Uncertain†	Caution and monitor
Vitamin B_6	Present‡	Present	Uncertain†	Caution and monitor

Am Fam Physician. 2004 Nov 1;70(9):1731-1738

WHAT ROLE DO SUPPLEMENTS PLAY?

- CHITOSAN: derived from chitin found in crustacean shells, prevent fat absorption by binding negatively charged fat molecules within the intestinal lumen.
  - A meta-analysis of five RCTs showed a 7.4lb weight reduction for chitosan over placebo. All of the studies were conducted by the same team of investigators and several methodological concerns were noted. Subsequently, three other researchers reported well-designed RCTs that **failed to show any differences in weight loss.**
- EPHEDRA alkaloid-caffeine combination: E*phedra sinica* (or *Ma huang* in Chinese) is a shrub native to China and Mongolia that contains sympathomimetic compounds referred to as ephedra alkaloids.
  - It showed a weight loss of 0.9 kg (2 lb) more per month for ephedra-containing supplements compared with placebo. There were 87 reports to the FDA MedWatch program about episodes of hypertension, arrhythmias, myocardial infarction, stroke, and seizures. Ten events led to death and 13 yielded permanent disability. Although ephedra-caffeine combinations may be effective for modest weight loss, **safety issues motivated the FDA to ban their sale in April 2004**

Am Fam Physician. 2004 Nov 1;70(9):1731-1738

WHAT ROLE DO SUPPLEMENTS PLAY?

- GUAR GUM: derived from the Indian cluster bean, *Cyamopsis tetragonolobus*; a soluble fiber, which theoretically could absorb water within the gut, causing increased satiety and lower caloric intake. A meta-analysis of **11 RCTs of guar gum versus placebo for weight loss showed no benefit.**
- SPIRULINA: Spirulina (also known as blue-green algae) contains phenylalanine, which is purported to inhibit appetite. In 1981, **the FDA declared spirulina ineffective for weight loss,** and no subsequent studies to the contrary have been published.

Am Fam Physician. 2004 Nov 1;70(9):1731-1738

MEDICATIONS THAT AFFECT WEIGHT

Table 4. Common Medications that Contribute to Weight Gain or Loss

Medication type	Promote weight gain	Weight neutral/variable	Promote weight loss
Antidepressants	Amitriptyline, doxepin, imipramine, mirtazapine (Remeron), nortriptyline (Pamelor), paroxetine (Paxil), phenelzine (Nardil)	Citalopram (Celexa), desvenlafaxine (Pristiq), duloxetine (Cymbalta), escitalopram (Lexapro), fluoxetine (Prozac), sertraline (Zoloft), venlafaxine	Bupropion (Wellbutrin)
Antipsychotics	Chlorpromazine, clozapine (Clozaril), olanzapine (Zyprexa), paliperidone (Invega), quetiapine (Seroquel), risperidone (Risperdal)	Aripiprazole (Abilify), haloperidol, ziprasidone (Geodon)	—
Cardiovascular agents	Amlodipine (Norvasc), atenolol, felodipine, metoprolol, nifedipine, propranolol	Angiotensin-converting enzyme inhibitors	—
Diabetic agents	Insulin, meglitinides, sulfonylureas, thiazolidinediones	Dipeptidyl peptidase-4 inhibitors	Alpha-glucosidase inhibitors, glucagon-like peptide-1 agonists, metformin, pramlintide (Symlin), sodium-glucose cotransporter-2 inhibitors
Hormones	Estrogens, steroids	—	Progestins, testosterone
Hypnotics	Diphenhydramine (Benadryl)	Benzodiazepines, trazodone	—
Mood stabilizers	Lithium	Oxcarbazepine (Trileptal)	—
Seizure medications	Carbamazepine (Tegretol), gabapentin (Neurontin), pregabalin (Lyrica), valproate (Depacon)	Lamotrigine (Lamictal), levetiracetam (Keppra), phenytoin (Dilantin)	Felbamate (Felbatol), topiramate (Topamax), zonisamide (Zonegran)

Adapted with permission from Obesity Medicine Association. Obesity algorithm. http://obesitymedicine.org/obesity-algorithm [registration required]. Accessed July 24, 2015, with additional information from reference 33.

Am Fam Physician. 2016 Sep 1;94(5):361-368.

SUMMARY

- Obesity is a serious, chronic, and TREATABLE disease affecting 42% of adults in the US
- Medications should be considered for patients who have not achieved weight loss goals with diet and lifestyle changes
- Between FDA approved short-term and long-term medications, Semaglutide has shown the most weight loss in the STEP trials
- No weight loss supplement meet the criteria for recommended use
- Be aware of medications that can affect weight, and the best way forward is to be vigilant from the very beginning

REFERENCES

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- Am Fam Physician. 2016 Jul 15;94(2):161-166.
- "Advancing the Field of Pharmacotherapy for Obesity: What Have the STEP Trials Taught Us?" 03:15–05:21
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- Davies M, et al. Lancet. 2021;397(10278):971-984.
- Wadden TA, et al. JAMA. 2021;325(14):1403-1413
- Rubino D, et al. JAMA 2021. doi:10.1001/jama.2021.3224 [Epub].
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- Chukir et al Obesity Res Clin Pract 2021;15:64-68
- Am Fam Physician. 2004 Nov 1;70(9):1731-1738

THANK YOU FOR YOUR TIME!

justine.suba@gmail.com

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

Demystifying MAT: Starting with Alcohol Use Disorder

Presented by:
Maureen Strohm, MD, FAAFP, FASAM

Approved for 1.0 Prescribed CME

Visit nvafp.com for any available handouts

Saturday, July 31, 2021
11:15am—12:15pm

NOTES

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

4th Annual NAFP Student/Resident Research Poster Displays

Approved for 1.0 Prescribed CME

Visit nvafp.com for any available handouts

Saturday, July 31, 2021
12:30—1:30pm

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
775-826-5100 • www.NVAFP.com

NOTES

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

An Introduction to Bedside Ultrasound

Presented by:
Ertha Nanton, MD, CPE

Approved for 2.0 Prescribed CME

Saturday, July 31, 2021
1:45—3:45pm

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
775-826-5100 • www.NVAFP.com

NOTES

An Introduction to Bedside Ultrasound (in office)

Ertha Nanton, MD, CCFP, ABFM, CPE
Chief Medical Officer (clinical Services),Nevada Health Centers
Core Faculty at Southern Hills Family Medicine Program (Sunrise Consortium)

Objectives

- 1. Define terms used in ultrasound
- 2. Describe the reference point on the probe
- 3. Introduce the two common planes of view
- 4. Describe different forms of artifact
- 5. Types of probes
- 6. Describe movements of the probe using the three Ss, H & R
- 7. Describe how to perform the scan using the mneumonic "DOGG"
- 8. Perform an abdominal aortic scan, check for free fluid in the abdomen, transabdominal obstetric scan, gallbladder scan

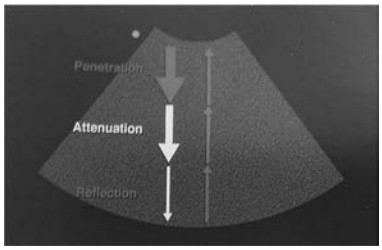
What is Ultrasound?

- Sound waves with higher frequencies that are not detectable by humans but can penetrate tissue.
- These sound waves echo off the tissue to the probe with varying reflection and this is how images can be detected on a screen.

What is Ultrasound?

- Ultrasound waves pass through a medium (PENETRATION)
- As it moves deeper into the medium the energy dissipates in the the process of ATTENUATION
- Some of the energy bounces back to the source known as REFLECTION

Penetration, Attenuation and Reflection



Socransky and Wiss, 2012

Glossary of Terms

- Echogenic: much of the sound waves bounces off (reflected) an object back to the probe (appears white on the screen)
- Echolucent: very little sound wave is reflected back from the object to the probe (appears dark on the screen)

Glossary of terms cont'd

- Hyperechoic: objects appear whiter than surrounding tissue (typical of solids) e.g bone is 100% hyperechoic
- Hypoechoic: objects appear darker than surrounding tissue (typical of liquids)e.g urine in the bladder is hypoechoic

Glossary of terms cont'd

- Isoechoic: objects appear similar in color to the surrounding tissue
- Anechoic: absolutely no sound waves are reflected back to the probe (appears black) : in the deep areas of the scan

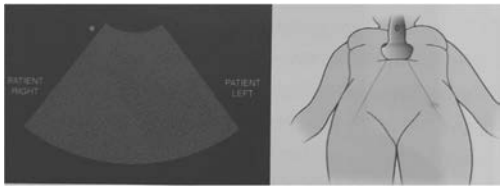
The reference point/Indicator/ mark

- The point of reference to orient the probe.
- It is always placed to patient's right side (transverse view) and toward the head (longitudinal view)

Socransky and Wiss, 2012

Two Planes of View

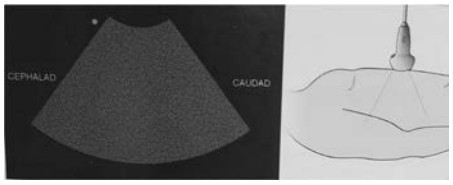
- 1. Transverse Plane (Right to Left)



Socransky and Wiss, 2012

Two Planes of View

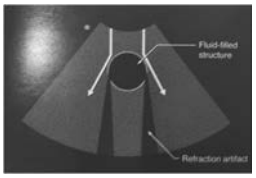
- 2. Longitudinal View: Head (cranial) to toe (caudal)



Socransky and Wiss, 2012

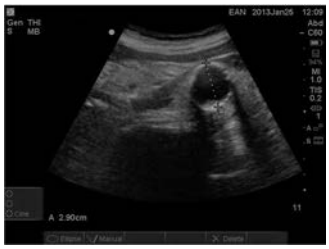
Edge Artifact

- Ultrasound waves hit a smooth, curved, fluid filled structure (e.g gallbladder, aorta) and is deflected (refracted). This causes a shadow on the edges



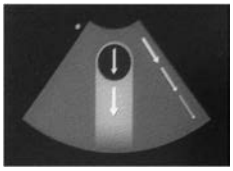
Socransky and Wiss, 2012

Edge Artifact



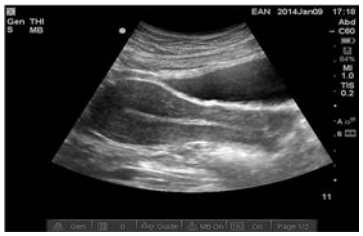
Enhancement

- When ultrasound waves pass thorough a liquid medium (e.g. bladder) and hit a more solid/denser structure behind it (e.g. uterus), it makes the structure light up more.



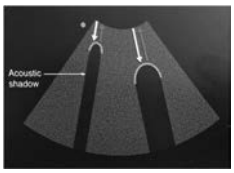
Socransky and Wiss, 2012

Enhancement



Acoustic shadow (window)

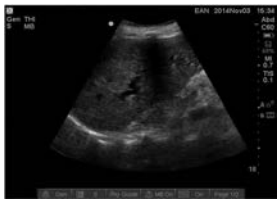
- Ultrasound waves hit a solid structure (e.g. bone) and the waves are reflected back to the probe. Because no waves get through, it casts a shadow behind the structure: appears black



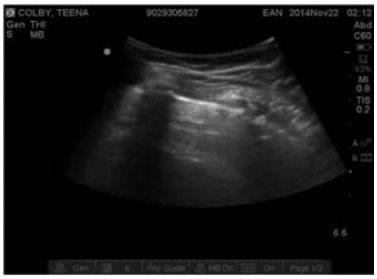
Socransky and Wiss, 2012

Acoustic shadow(window)

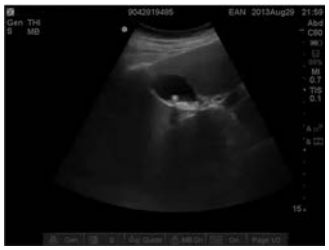
- Lines cut right across tissue planes and is straight (rib shadow)



Lung Sliding and rib shadows

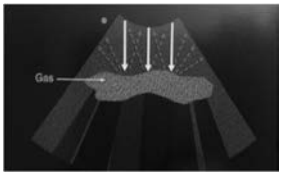


What's a good use for the acoustic shadow?



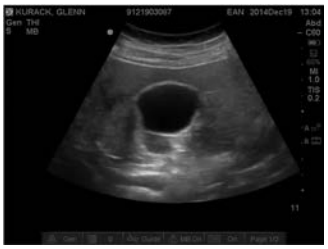
Scatter

- When gas is present, the US waves gets deflected forming a gray snow pattern called scatter



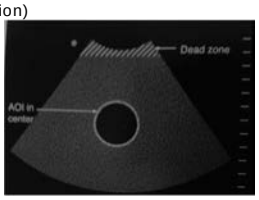
Socransky and Wiss, 2012

Scatter



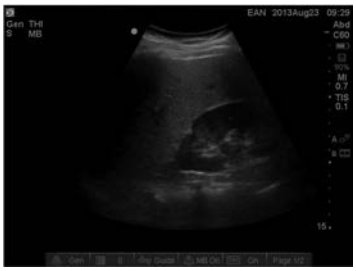
Dead Zone

- Represents the area that is a few centimetres below the top of the screen. Seen with the low frequency probe: gives no useful information)



Socransky and Wiss, 2012

Dead Zone



Types of probes

- 1. Curved array probe
- 2. Phased array probe
- 3. Microconvex probe
- 4. Linear array probe
- 5. Endocavitary probe

Curved Array Probe

Socransky and Wiss, 2012

Curved Array Probe

- Low frequency : 2-5 MHz (usually 3.5MHz)
- Good penetration (down to 25-30 cm)
- Gives a pie shaped half circle
- Used for abdominal ultrasound
- Disadvantages: poorer contact with skin; cannot get between ribs.

Phased Array Probe

Socransky and Wiss, 2012

Phased Array Probe

- Low frequency probe (usually 2.5MHz)
- Penetrates deep
- Smaller footprint and flat surface.
- Gets between intercostal spaces and better contact with skin
- Gives triangular image
- Disadvantages: Near field is small and the far field has poor resolution

Microconvex probe

Socransky and Wiss, 2012

Microconvex probe

- Cross between the curved and phased array probe
- Low frequency probe (3.5MHz)
- Penetrates deep down
- Fits better into tight spaces (between the ribs)
- The image is a cross between the curved and phased array probes

Linear Array Probe

Socransky and Wiss, 2012

Linear Array Probe

- High frequency probe (5-18MHz)
- Head is flat
- Gives a rectangular field of view
- Not great for penetration to deeper structures but higher resolution
- Negligible dead zone
- Ideal for small superficial structures like blood vessels and nerves

Endocavitary Probe

- Mid frequency probe with a long handle (6.5MHz)
- Greater amount of curvature of the probe.
- Field spans out more than the curved array probe
- Used predominantly for transvaginal exams, but can be used for identifying structures around a peritonsillar abscess

Movements of the probe

- Find the start **SITE**
- **SLIDE** the probe
- **SWEEP** the probe
- **HEEL** the probe (not always used)
- **ROTATE** the probe

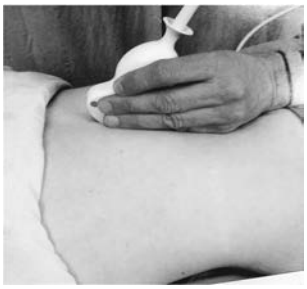
Sliding the probe (anterior/posterior)

Socransky and Wiss, 2012

Sweeping the probe (side to side)

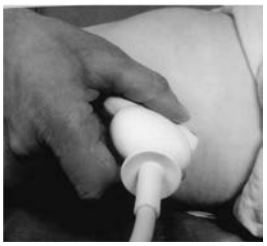
Socransky and Wiss, 2012

Heeling the probe (tilting upward)



Socransky and Wiss, 2012

Rotating the probe



Socransky and Wiss, 2012

Performing the scan

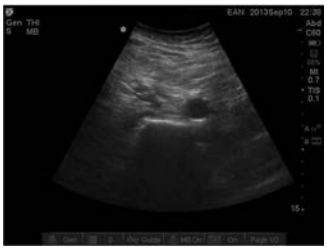
- **D**: **DIVE** to the lowest depth (about 25-30cm with the low frequency probe and 10-15cm with the high frequency)
- **O**: **Orient** the probe (where is the reference point)
- **G**: **Goo/Gel**: Apply copious amounts to probe (only apply to patient when doing a cardiac scan)
- **G**: **Gain** (may be adjusted accordingly):

It modulates the strength of the signal returning to the probe. Increase the gain, increase the whiteness and decrease the gain, increase the darkness

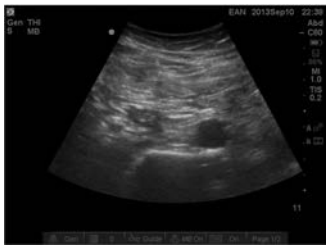
Abdominal aortic scan (transverse plane only)

- 1. Patient supine
- 2. D (dive to lowest depth)
- 3. Orient probe in transverse plane (ref point to patient's right)
- 4. Apply gel
- 5. Place probe in transverse plane just below xiphoid process
- 6. Find aorta and center it (thick walls, not compressible completely, no respiratory variability, closest to the spine)
- Slide caudally to bifurcation (two lumens seen)
- Measure aorta at different points along its length

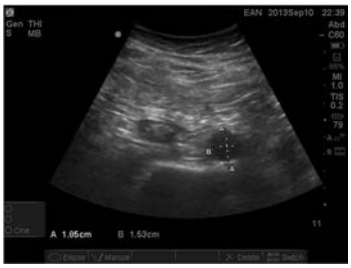
Abdominal aortic scan



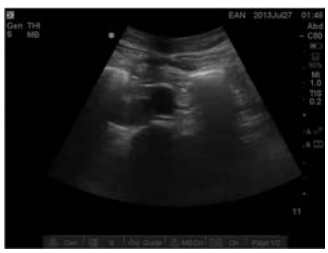
Abdominal aortic scan



Abdominal aortic scan

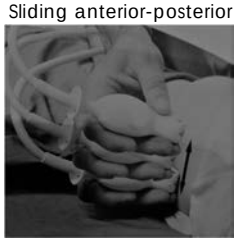


Abdominal aortic scan



The Abdominal Scan for Free Fluid (RUQ,LUQ, pelvis)

Sliding anterior-posterior



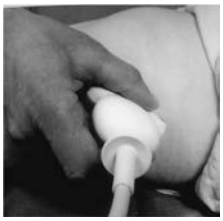
Sliding cranial-caudad

Socransky and Wiss, 2012

The Abdominal Scan for Free Fluid (RUQ,LUQ, pelvis)

Sweeping the probe

Rotating the probe

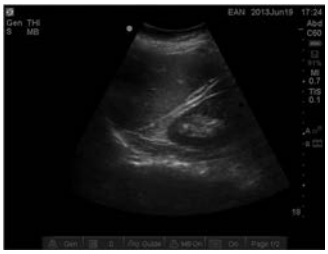


Socransky and Wiss

The Abdominal Scan for Free Fluid Liver and Kidney(RUQ)

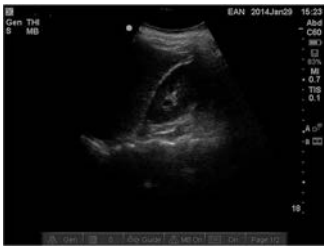
- Site: posterior axillary line at xiphoid process in longitudinal plane
- Slide: Slide anterior to posterior to find the kidney and interface
- Slide: Slide cranially and caudally (longitudinally) to center the interface
- Decrease depth to magnify
- You may need to rotate for a better view(get ribs out of way)
- Sweep through the hepatorenal interface (Morrison's pouch) looking for free fluid. This is your Area of Interest!

Organs orientation in longitudinal plane (RUQ)



Socransky and Wiss, 2012

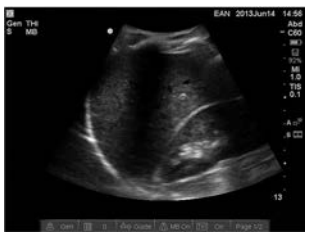
Hepatorenal interface



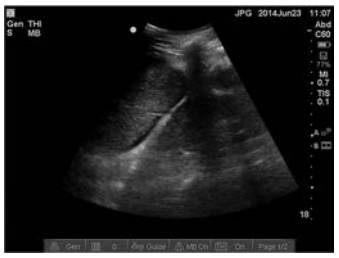
The Spleen and kidney scan (LUQ)

- Leave the depth at the magnification previously set
- Site: posterior axillary line slightly ABOVE the xiphoid process in longitudinal plane. (kidney is more cranial on that side)
- Slide: Slide anterior to posterior to find the kidney and interface
- Slide: Slide cranially and caudally (longitudinally) to center the interface
- Should see diaphragm from 6 to 9 o 'clock
- Sweep through the splenorenal interface (look for free fluid). This is your Area of Interest!

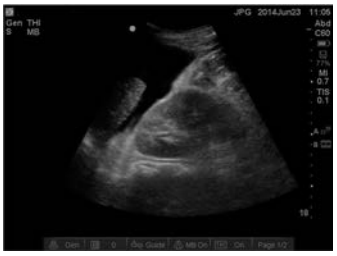
Splenorenal Interface



Free fluid in Morrison's Pouch (hepatorenal interface)



More free fluid anterior to the liver (ascites)

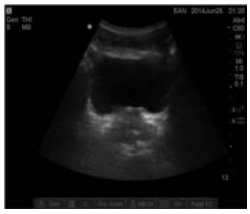


Pelvic scan (transverse plane)

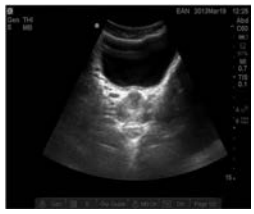
- Site: Immediately cephalad to the symphysis pubis in the transverse plane
- Slide: Slide probe laterally and medially to find the bladder (patient should have full bladder, if possible)
- Find the uterus (female) or prostate (male)
- Sweep cranially and caudally to look for free fluid
- Rotate probe in longitudinally to corroborate findings , if needed

Transabdominal pelvic scan (male and female)

- Male (Look for prostate)



- Female (Look for uterus)



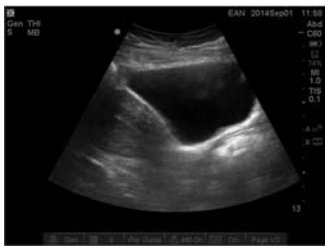
Transabdominal obstetric scan

- Start in longitudinal position in midline just above pubis symphysis
- Slide: Slide probe laterally and medially to find the bladder (patient should have full bladder)
- Uterus is juxtaposed posterior to bladder
- Center uterus, adjust depth and heel probe cephalad or caudad
- Sweep through uterus and endometrial stripe
- Rotate probe in transverse plane and sweep again

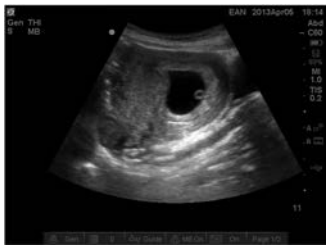
Identifying an intrauterine pregnancy (IUP)

- The **top three** out of FOUR are imperative
  - 1. **The decidual reaction** (hyperechoic decidual lining of the uterus): present around 14 days
  - 2**. The gestational sac**: present around (anechoic region within the decidua)
  - 3**. The Yolk Sac:** The "Double Ring Sign" (seen 5 weeks transvaginal and 6-7 weeks transabdominal)
  - 4. **Fetal Pole and cardiac activity** (6 weeks for transvaginal and 7-8 weeks for transabdominal : this trumps the yolk sac

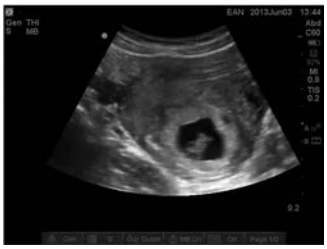
Normal transabdominal scan



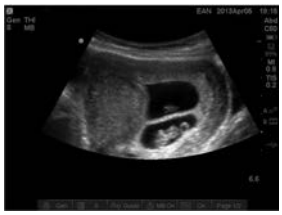
Identifying an intrauterine pregnancy (IUP)

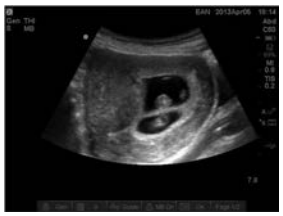


Identifying an intrauterine pregnancy (IUP)



Identifying an intrauterine pregnancy (IUP)

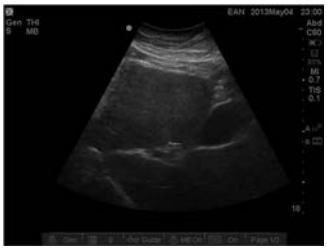




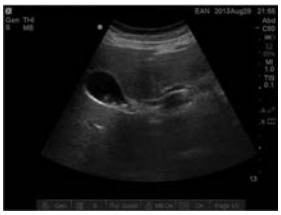
Gallbladder scan (left lateral decubitus)

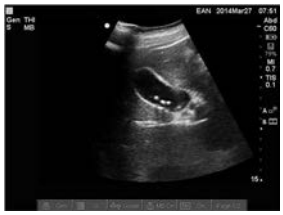
- Roll patient to left side (brings gallbladder closer to probe)
- Place probe (in heel cranial position)in epigastrium and slide laterally along the costal margin to the right anterior axillary line
- If not effective, turn probe in slightly oblique position and slide along the costal margin, keeping the liver in view
- Keep eyes in the near field for first cystic structure
- Ask patient to take deep breath in and hold (pushes bladder down)
- Find the "Exclamation Point sign" with the gallbladder, main lobar fissure and portal vein
- Look for stones, sonographic Murphy's sign, wall thickening (N: ≤3mm), distension (N:5cm(W)x10cm(L)), pericholecystic fluid, biliary sludge

Gallbladder scan (left lateral decubitus)



What is wrong here?





To become proficient in ultrasound

- Be consistent with your approach to ultrasound
- Understand your probes and their uses
- Ultrasound as many appropriate patients as you can
- Practice getting the image…the diagnosis will come
- Do not look at your hands, look at the screen
- Do not hold the probe in a "death grip"…your hands will tire easily
- Most of all: Get additional training!

- NOW LET'S GET SCANNING!

NOTES

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

High Risk Medication Use in the Elderly and Deprescribing

Presented by:
Alvin B. Lin, MD, FAAFP

Approved for 1.0 Prescribed CME

Saturday, July 31, 2021
4:00—5:00pm

Nevada Academy of Family Physicians • 705 Tahoe St., Ste. A, Reno, NV 89509
775-826-5100 • www.NVAFP.com

NOTES

High Risk Medication Use in the Elderly And Deprescribing

Alvin B. Lin, MD, FAAFP
CAQ Geriatric Medicine
Solo Private Practice, Las Vegas, NV
Saturday, July 31st, 2021

Objectives

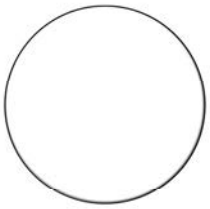
- Evaluate common prescribing errors in the elderly
- Develop an approach for safe drug prescribing and monitoring in the elderly
- Consider the application of the Beers and START/STOPP criteria for prescription of benzodiazepines and similar high-risk medications in the elderly patient population
- Adopt a step-wise approach to the evaluation of polypharmacy and describing process that includes follow-up monitoring for adverse effect and symptom rebound

Geriatric Prescribing Mantra

- Start low
- Go slow
- But get there!

Evaluate common prescribing errors in the elderly

AES Question



AES #1

- 70yo M, new to your practice, reports high blood pressure in afternoons & evenings, but fine in mornings. He's been taking Amlodipine 10mg, Metoprolol 100mg, HCTZ 12.5mg, & Losartan 50mg daily for years. This is a new phenomenon since moving here. Should you
- 1) increase Losartan to 100mg daily
- 2) split Amlodipine into 5mg twice daily
- 3) review/calibrate personal BP cuff
- 4) examine pill bottles

Common Prescribing Errors

- Indication errors
  - Underuse
  - Overuse
  - Misuse
- Dosing errors
- Drug-drug interactions

Common Prescribing Errors

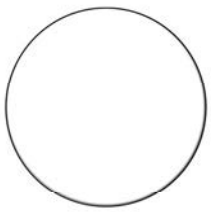
- Lack of patient information
  - Inadequate records
  - Undocumented allergies
- Communication failure
  - Clinician-patient communication
- Errors in special populations

Common Prescribing Errors

- Formulation issues
  - Bupropion
    - Extended release (QD) vs sustained release (BID)
  - Metoprolol
    - Succinate (QD) vs Tartrate (BID)
- Red flag overload aka alert fatigue
  - Too many red flags & alerts w/low credibility in real life

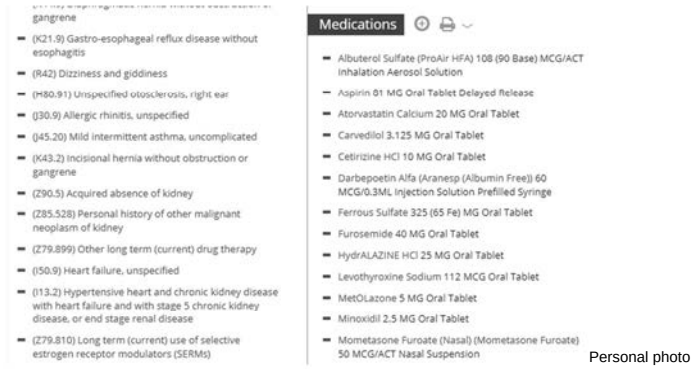
Develop an approach for safe drug prescribing and monitoring in the elderly

AES Question



AES #2

- Your 70yo M asks for enough refills to last a year b/c “that’s what my previous doctor always did for me.” Your response should be
- 1) Thank you. Next!
- 2) It’s my way or the highway, buddy!
- 3) Since we’re adjusting your regimen, you should come back sooner for a re-evaluation.
- 4) How about we split the difference at 6 months?

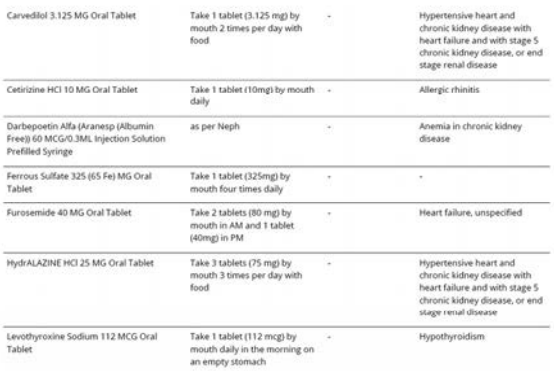


- gangrene
- (K21.9) Gastro-esophageal reflux disease without esophagitis
- (R42) Dizziness and giddiness
- (H80.91) Unspecified otosclerosis, right ear
- (J30.9) Allergic rhinitis, unspecified
- (J45.20) Mild intermittent asthma, uncomplicated
- (K43.2) Incisional hernia without obstruction or gangrene
- (Z90.5) Acquired absence of kidney
- (Z85.528) Personal history of other malignant neoplasm of kidney
- (Z79.899) Other long term (current) drug therapy
- (I50.9) Heart failure, unspecified
- (I13.2) Hypertensive heart and chronic kidney disease with heart failure and with stage 5 chronic kidney disease, or end stage renal disease
- (Z79.810) Long term (current) use of selective estrogen receptor modulators (SERMs)

Medications

- Albuterol Sulfate (ProAir HFA) 108 (90 Base) MCG/ACT Inhalation Aerosol Solution
- Aspirin 81 MG Oral Tablet Delayed Release
- Atorvastatin Calcium 20 MG Oral Tablet
- Carvedilol 3.125 MG Oral Tablet
- Cetirizine HCl 10 MG Oral Tablet
- Darbepoetin Alfa (Aranesp (Albumin Free)) 60 MCG/0.3ML Injection Solution Prefilled Syringe
- Ferrous Sulfate 325 (65 Fe) MG Oral Tablet
- Furosemide 40 MG Oral Tablet
- HydrALAZINE HCl 25 MG Oral Tablet
- Levothyroxine Sodium 112 MCG Oral Tablet
- MetOLazone 5 MG Oral Tablet
- Minoxidil 2.5 MG Oral Tablet
- Mometasone Furoate (Nasal) (Mometasone Furoate) 50 MCG/ACT Nasal Suspension

Personal photo



Carvedilol 3.125 MG Oral Tablet	Take 1 tablet (3.125 mg) by mouth 2 times per day with food	-	Hypertensive heart and chronic kidney disease with heart failure and with stage 5 chronic kidney disease, or end stage renal disease
Cetirizine HCl 10 MG Oral Tablet	Take 1 tablet (10mg) by mouth daily	-	Allergic rhinitis
Darbepoetin Alfa (Aranesp (Albumin Free)) 60 MCG/0.3ML Injection Solution Prefilled Syringe	as per Neph	-	Anemia in chronic kidney disease
Ferrous Sulfate 325 (65 Fe) MG Oral Tablet	Take 1 tablet (325mg) by mouth four times daily	-	-
Furosemide 40 MG Oral Tablet	Take 2 tablets (80 mg) by mouth in AM and 1 tablet (40mg) in PM	-	Heart failure, unspecified
HydrALAZINE HCl 25 MG Oral Tablet	Take 3 tablets (75 mg) by mouth 3 times per day with food	-	Hypertensive heart and chronic kidney disease with heart failure and with stage 5 chronic kidney disease, or end stage renal disease
Levothyroxine Sodium 112 MCG Oral Tablet	Take 1 tablet (112 mcg) by mouth daily in the morning on an empty stomach	-	Hypothyroidism

Personal photo

Brown Bag

- Ask questions
  - −Why are you taking this medication?
  - −Is it working?
  - −Any side effects?
  - −Can you afford it?

- Ask questions
  - −Why are you taking this medication?
    - Don't accept "because my doctor said to take it"
    - Don't need to go into nitty gritty detail
    - But make sure patient understands basic indication

- Ask questions
  - −Why are you taking this medication?
  - −Is it working?
    - Does it do what you want?
    - Does it do what your doctor wants?

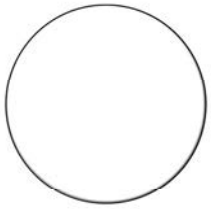
  - −Can you afford it?
    - What's your copay?
    - What happens when you run out of these samples?
    - What about generic alternative?

Brown Bag

- Set aside quarterly to semi-annual visits to do nothing but review medications
  - Set up practice policy of renewing/refilling all medications during in-person visits
  - Avoid refilling one medication at a time based upon pharmacy request w/o reviewing necessity/effectiveness/alternatives
  - Avoid refilling medications for a whole year
    - Makes patients & pharmacies happy
    - But near impossible to review appropriateness

Consider the application of the Beers and START/STOPP criteria for prescription of benzodiazepines and similar high-risk medications in the elderly patient population

AES Question



AES #3

- Your 70yo M returns c/o frequent voiding esp nocturia. Which treatment option(s) is(are) most fraught w/risk?
- 1) Stop his HCTZ 12.5mg
- 2) Add Oxybutynin 10mg
- 3) Advise him to stop consuming liquids after dinner
- 4) Minimize fluid consumption throughout the day
- 5) both 2) and 4)

Beers List

- Nothing to do w/Budweiser, Coors, Michelob & Samuel Adams
- Nothing to do w/ales, lagers, malts, porters & stouts
- Developed by Mark Beers, MD in 1991
- Revised 1997, 2003, 2012, 2015 & 2019

J Am Geriatr Soc. 2019 Apr;67(4):674-694

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Beers List

- More formally known as Beers Criteria for *Potentially* Inappropriate Medication (PIMs) Use in Older Adults
- Use of PIMs linked to poor health outcomes incl confusion, falls & mortality
- Use guideline as starting point for discussion
- Understand rationale as well as caveats
- Always search for safer (non)pharmacologic options
- Don't apply to end-of-life & hospice patients

J Am Geriatr Soc. 2019 Apr;67(4):674-694

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Beers List

- 30 medications or classes to be avoided in general
- 40 medications or classes to be used w/caution
- Avoid (chronic) NSAID use
  - Esp in setting of CHF and/or CKD
- Avoid NDHP CCB in HFrEF
- Avoid Ciprofloxacin (and quinolones in general)
  - Incr risk of tendon rupture & adverse neurologic effects

J Am Geriatr Soc. 2019 Apr;67(4):674-694

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Beers List

- Avoid Dabigatran (Pradaxa) & Rivaroxaban (Xarelto)
  - Incr risk of GI bleed if $\geq$75yo c/w other DOAC
- Avoid SSRIs & SNRIs in those w/h/o falls or fx
- Avoid Glimepiride & other sulfonylureas b/c incr risk severe prolonged hypoglycemia

J Am Geriatr Soc. 2019 Apr;67(4):674-694

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Beers List

- Avoid most antipsychotics in those w/Parkinson disease
  - May use Clozapine, Pimavanserin or Quetiapine if cannot avoid antipsychotic use

10 Medications to Avoid in Elderly

- NSAIDs
  - If can't be avoided, add PPI to reduce bleeding risk
  - Reconsider if >75yo, concomitant oral steroids and/or blood thinners ie VKA or DOACs
  - Really reconsider if CKD or CHF
- Digoxin
  - Avoid >0.125mg/d

10 Medications to Avoid in Elderly

- Sulfonylureas
  - Glyburide & chlorpropamide incr hypoglycemic risk
- Muscle relaxants
  - Carisoprodol, cyclobenzaprine & methocarbamol incr sedation & confusion, falls, constipation, dry mouth & urinary retention
- Sedative-hypnotics
  - Benzodiazepines & Z-drugs (for insomnia) incr sedation, confusion & fall risk

10 Medications to Avoid in Elderly

- Anticholinergics
  - Amitriptyline & Imipramine
  - Trihexyphenidyl
  - Dicyclomine
  - OTC diphenhydramine & chlorpheniramine
  - Incr confusion, constipation, dry mouth, urinary retention etc
- Meperidine
  - Incr seizure risk

10 Medications to Avoid in Elderly

- Antipsychotics
  - Unless being used to manage BPD/psychosis/schizophrenia
  - Do NOT use for insomnia
- Estrogen pills & patches
  - Incr DVT/PE risk

- American Geriatric Society's Health in Aging Foundation
  - https://www.healthinaging.org/tools-and-tips/ten-medications-older-adults-should-avoid-or-use-caution

STOPP/START

- 1st version in 2008
- Revised 2015
- Focus on avoiding adverse drug events (ADEs) & potential prescribing omissions (PPOs)

Age and Ageing. 2015;44:213-8

STOPP/START

- Screening Tool of Older People's Prescriptions (STOPP)
- Screening Tool to Alert to Right Treatment (START)

Age and Ageing. 2015;44:213-8

STOPP

- Look for meds prescribed
  - w/o evidence-based clinical indication
  - beyond recommended duration
  - in duplicate or more!

Age and Ageing. 2015;44:213-8

STOPP

- Cardiovascular
  - Digoxin for heart failure w/PEF (preserved ejection fraction)
  - Diltiazem or verapamil in NYHA III or IV heart failure
  - Diltiazem or verapamil in combination w/beta blocker

Age and Ageing. 2015;44:213-8

STOPP

- Cardiovascular
  - Loop diuretic for edema w/o clinical, biochemical or radiologic evidence of heart failure, liver failure, nephrotic syndrome or renal failure
  - Loop diuretic for HTN w/concurrent urinary incontinence

Age and Ageing. 2015;44:213-8

STOPP

- Cardiovascular
  - Thiazide diuretic if
    - Hypokalemia
    - Hyponatremia
    - Hypercalcemia
    - Gout
  - Phosphodiesterase 5 inhibitor if concurrent nitrate

STOPP

- Antiplatelets/anticoagulants
  - ASA >160mg/d
  - ASA w/h/o peptic ulcer disease w/o PPI
  - ASA + antiplt for 2o stroke prevention unless s/p stent <12mo or concurrent acute coronary syndrome or high grade symptomatic carotid stenosis

Age and Ageing. 2015;44:213-8

STOPP

- Antiplatelets/anticoagulants
  - Vitamin K antagonist or novel oral anticoagulant plus
    - Antiplatelet
    - ASA
    - NSAIDs

Age and Ageing. 2015;44:213-8

STOPP

- CNS & psychotropics
  - TCAs in dementia, benign prostate hypertrophy or urinary retention
  - SSRIs in current or recent hyponatremia
  - Antipsychotics in Parkinsonism or Lewy body disease

Age and Ageing. 2015;44:213-8

STOPP

- CNS & psychotropics
  - Antipsychotic in pts w/behavioral & psychological symptoms of dementia unless severe & other non-pharm tx have failed
  - Antipsychotics as hypnotics unless sleep disorder due to psychosis

Age and Ageing. 2015;44:213-8

STOPP

- Renal
  - NSAIDs if eGFR <50
  - Dabigatran if eGFR <30
  - Digoxin >0.125mg/d if eGFR <30
  - Metformin if eGFR <30
  - Rivaroxaban & apixaban if eGFR <15
  - Colchicine if eGFR <10

Age and Ageing. 2015;44:213-8

STOPP

- Gastrointestinal
  - PPIs >8wks for uncomplicated PUD
  - If chronic constipation, avoid those likely to exacerbate
    - Aluminum antacids
    - Anticholinergics
    - Antimuscarinics
    - Opioids
    - Oral iron
    - Verapamil

Age and Ageing. 2015;44:213-8

STOPP

- Gastrointestinal
  - Don't use chronic opioids w/o concomitant laxative

Age and Ageing. 2015;44:213-8

STOPP

- Respiratory
  - Theophylline as monotherapy for COPD
  - Systemic instead of inhaled corticosteroids in moderate-to-severe COPD
  - Benzodiazepines in acute or chronic respiratory failure

Age and Ageing. 2015;44:213-8

STOPP

- Musculoskeletal
  - NSAIDs other than Cox-2 inhibitor if h/o PUD or GI bleed, unless concurrent PPI or H2 blocker
  - NSAIDs if severe HTN or CHF
  - NSAIDs for gout if no contraindication to XOI
  - NSAIDs + corticosteroids w/o PPI prophylaxis

Age and Ageing. 2015;44:213-8

STOPP

- Endocrine
  - Thiazolidinediones in heart failure
  - Oral estrogen w/o progestogen if intact uterus
  - Androgens w/o hypogonadism

Age and Ageing. 2015;44:213-8

STOPP

- Fall risk
  - Benzodiazepines & nonBZD receptor agonists
  - Neuroleptics
  - Vasodilators
    - ACE inhibitors, alpha-1 blockers, ARBs & CCBs

Age and Ageing. 2015;44:213-8

START

- ACE inhibitor if stable HF and/or CAD
- Anticoagulation if chronic atrial fibrillation
- Antihypertensive if SBP >160mmHg or DBP >90mmHg
- Antiplatelet if h/o CAD, CVA or PVD

Age and Ageing. 2015;44:213-8

START

- Beta blocker if CAD or stable HF
- Bisphosphonate + vitamin D + calcium if long-term systemic corticosteroids
- Continuous oxygen if O2 sat <89%
- Dopamine or dopamine agonist if Parkinson's disease w/fxn impairment

Age and Ageing. 2015;44:213-8

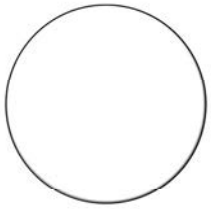
START

- Statin if h/o CAD, CVA or PVD unless >85yo
- Vitamin D if housebound, experiencing falls or osteopenic
- Xanthine oxidase inhibitors (XOIs) if recurrent gout

Age and Ageing. 2015;44:213-8

Adopt a step-wise approach to the evaluation of polypharmacy and describing process that includes follow-up monitoring for adverse effect and symptom rebound

AES Question



AES #4

- What is best way to deprescribe in setting of polypharmacy?
- 1) Ask questions, make change(s), reassess in 3-6mo
- 2) Ask questions, make change(s), make more changes
- 3) Make change(s), ask questions, reassess in 1 year
- 4) Ask questions, make change(s), reassess in 1 year

Ask Questions!

- Why are you taking this medication?
- Is it working?
- Any side effects?
- Can you afford it?

- Review of systems = review of medication list

Beware of Domino Effect

- Why are you taking Drug C? to manage side effects from Drug B
- Why are you taking Drug B? to manage side effects from Drug A

- Perhaps better to decr or d/c CCB rather than add diuretic
  - *JAMA Netw Open*. 2019;2(12):e1918425
  - doi:10.1001/jamanetworkopen.2019.18425
- Perhaps better to d/c diuretic rather than add antimuscarinic

Look for Duplicate Therapy

- Do you need both ASA & antiplt?
- Do you need both H2 blocker & PPI?
- Do you need both BB & NDHP CCB?

- Maybe you do, but often you don't!

Look for Contradictory Therapy

- Drug-drug interactions
  - Look for concomitant diuretic & antimuscarinic
  - Look for concomitant anticholinergic & AChEI
  - Look for concomitant alpha blocker & alpha agonist

- Drug-disease interactions
  - Look for (unnecessary) diuretic use in pts w/mobility issues
  - Look for alpha blocker use in pts w/orthostatic hypotension

Accept Higher/Lower Standards

- Does your patient really need SBP <130
- Does your patient really need A1c <7
- Does your patient really need LDL <70

Advocate for Your Patients' Pocketbook

- Too often our patients don't know better & are afraid to question authority, thus they can't advocate for themselves
- Do you need expensive Ranexa (ranolazine) if you've never tried inexpensive Isosorbide mononitrate?
- Do you need expensive Effient (prasugrel) if you've never tried less expensive Clopidogrel?

Agree to Disagree

- Agree to disagree, then reconvene
- Change a medication, change a dose, stop drug altogether, whatever, do something!
  - Then agree on time (3-6mo, perhaps even sooner) to reconvene & re-evaluate
- Repeat until medication list is cleaned up & makes sense

Barriers to Practice

- Lack of time
- Too many bottles!
- Faded labels
- Clinical inertia

Best Practice Recommendations

- Review diagnosis list
  - Make sure every diagnosis is prescribed appropriately
- Review medication list
  - Make sure every medication is linked to correct diagnosis
- Ask your elderly patients to bring in all their medications in their pill bottles at least once a year for brown bag review
  - Look for dispense date on bottle, in add'n to drug name/dose etc

Best Practice Recommendations

- For those patients who take 5 of more drugs, consider setting aside an appointment every 3-6mo to review all medications at once then prescribe enough until next review/refill appointment
  - Avoid filling medications one here & one there, on the run w/o asking
    - Why are you taking this?
    - Does it work?
    - Any side effects?

AES Answers

- AES #1
  - 4
- AES #2
  - 3
- AES #3
  - 5
- AES #4
  - 1

Nevada Academy of Family Physicians
32nd Annual Summer CME Meeting
July 30–August 1, 2021

Journal Jeopardy!

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Jeffrey Ng, MD, FAAFP

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NOTES