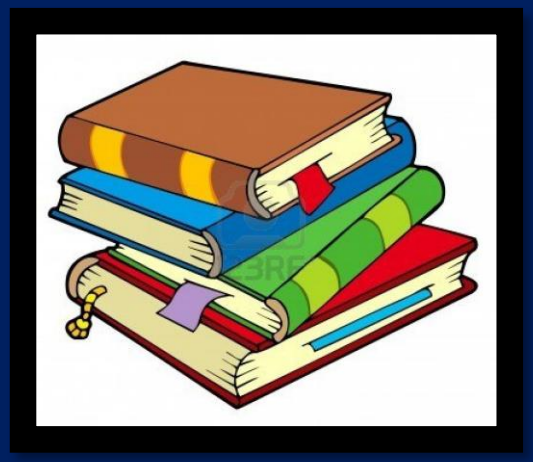

Academic Half Day: Making Your Medical Knowledge Stick

Education Scholars Team

Brenda Shinar, Director

Dana Archbold, Greg Dodaro, Lise Harper

Banner University Medical Center Phoenix

Arizona Medicine Phoenix

Objectives

1. Describe the characteristics of a great physician.
2. Understand your AHD obligation.
3. Understand the new process for active learning in AHD.
4. Understand the eight habits to effective studying.
5. Use one of the eight habits to learn and teach a piece of medical knowledge.
6. Use the advice you hear from past residents to help formulate your own study plan.

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Becoming a physician…

1. Medical school
2. Internship
3. Residency
4. Passing Boards

Becoming a *GREAT* physician…

1. Medical knowledge
2. Communication skills
3. Hard work ethic/ team player
4. Humility
5. Kind/Compassionate
6. Resilient

Medical Knowledge

- 10,000 hours of deliberate practice

- Time
- Effort
- Organization/Planning
- Little steps/bites with frequent review

Question 1.

Becoming a great physician is an arduous process and requires discipline, commitment, and endurance.

Which of the six attributes of a great physician is the most important, according to Dr. Shinar, and why?

A. Resilience
B. Compassion
C. Communication Skills
D. Medical Knowledge
E. Humility

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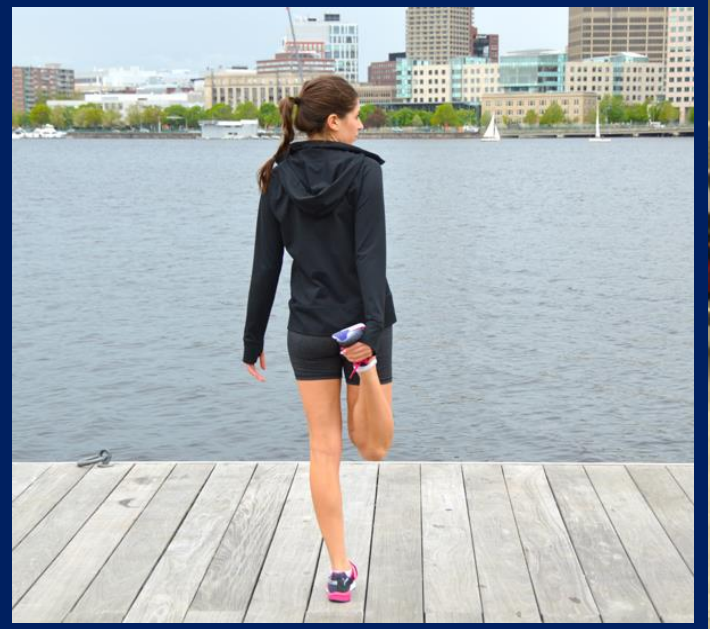
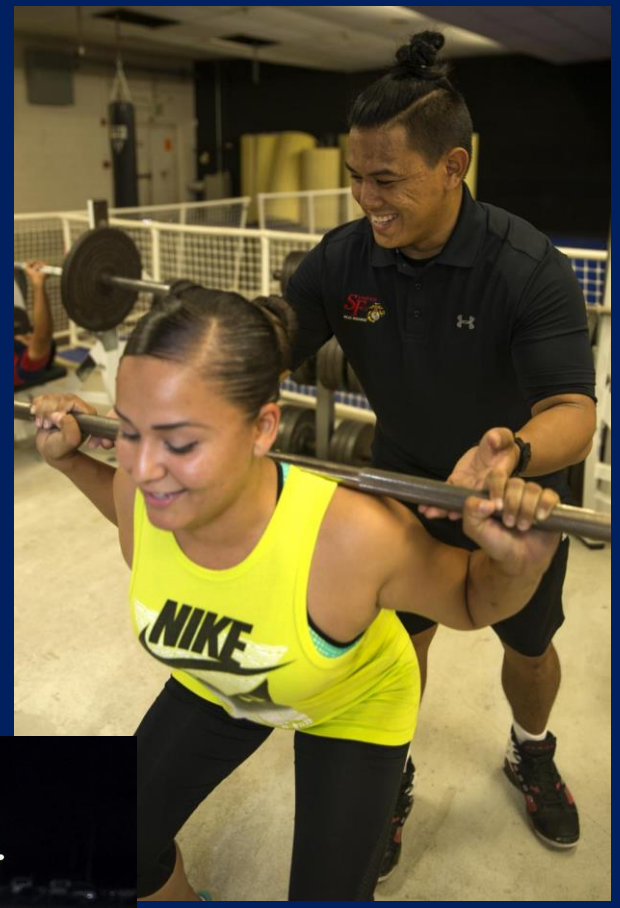
Academic Half Day (AHD)

- Articles/Objectives/ MKSAP questions
- Board Preparation/Patient Care
- ***This is now your job! You are being paid to learn!***

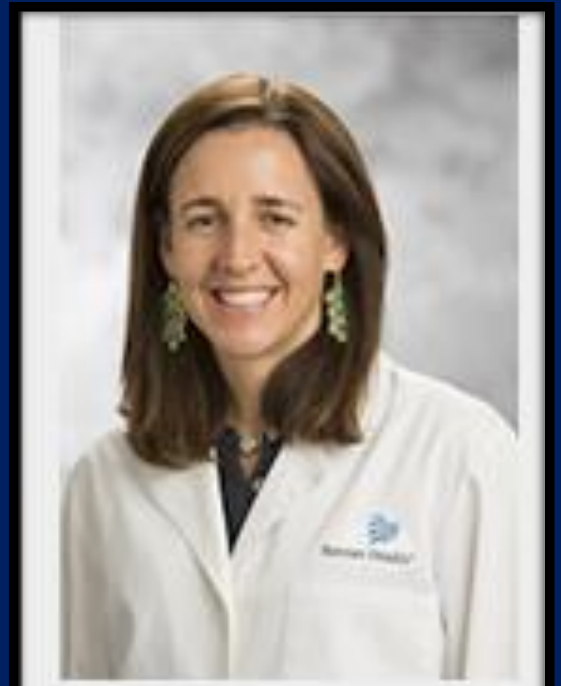
Academic Half Day

- We start at 9:15
- Prepare in advance*
- Bring your study materials
- Pay attention! Learn actively
- Write down something you learned from each lecture
- Engage your attendings when you go back to your clinical service…





Brenda Shinar, MD



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Education Scholars

2019-2020

Journal of the
American Association of Nurse Practitioners

REVIEW - SPONSORED

Medical management of acute coronary syndromes

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Keywords
Coronary artery disease; acute coronary syndrome; disease management; secondary prevention; nurse practitioner; advanced practice nurse.

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Abstract

Background: Recent updates to clinical guidelines and pharmacological indications have added to the complexity of acute coronary syndrome (ACS) management. Advanced practice nurses working with ACS patients need clear and up-to-date information to optimize patient care.
Purpose: To provide a practical overview of the management of ACS from patient presentation through to long-term secondary prevention based on recent guidelines and randomized controlled trials, with particular emphasis on medical management.
Methods: Systematically reviewed recent studies and guidelines published 2011–2015 using PubMed search terms including "ACS management," "ACS hospital care," and "ACS secondary prevention."
Conclusions: The last decade has seen an increase in the number of antithrombotic (anticoagulant and antiplatelet) agents and an expansion of their licensed indications for treatment of ACS patients. Future trials will help identify which subgroups of patients will gain the greatest benefit from more intense antithrombotic therapy.
Implications for practice: Management of ACS is dependent on individual patient characteristics and risk stratification. Greater choice among therapies available for acute and long-term management will help to achieve optimal, patient-tailored care.

Introduction

Coronary artery disease (CAD) is a leading cause of mor-

Etiology of ACS: Role of endothelial dysfunction, oxidative stress, and inflammation

ACS refers to a spectrum of coronary conditions in

Oncologic Emergencies: Recognition and Initial Management

Mark L. Higdon, DO; Charles J. Atkinson, MD; and Kelley V. Lawrence, MD
Novant Health Family Medicine Residency, Charlotte, North Carolina

Most oncologic emergencies can be classified as metabolic, hematologic, structural, or treatment related. Tumor lysis syndrome is a metabolic emergency that presents as severe electrolyte abnormalities. Stabilization is focused on vigorous rehydration, maintaining urine output, and lowering uric acid levels. Hypercalcemia of malignancy, which is associated with poor outcomes, is treated with aggressive rehydration, intravenous bisphosphonates, and subspecialty consultation. Syndrome of inappropriate antidiuretic hormone should be suspected if a patient with cancer has hyponatremia. This metabolic condition is treated with fluid restriction or hypertonic saline, depending on the speed of development. Febrile neutropenia is one of the most common complications related to cancer treatment, particularly chemotherapy. It usually requires inpatient therapy with rapid administration of empiric antibiotics. Hyperviscosity syndrome may present as spontaneous bleeding and neurologic deficits, and is usually associated with Waldenström macroglobulinemia. Treatment includes plasmapheresis followed by targeted chemotherapy. Structural oncologic emergencies are caused by direct compression of nontumor structures by metastatic disease. Superior vena cava syndrome presents as facial edema with development of collateral venous circulation. Intravascular stenting leads to superior patient outcomes and is used in addition to oncology-directed chemotherapy and radiation therapy. Malignant epidural spinal cord compression is managed in conjunction with neurosurgery, but it is classically treated using steroids and/or surgery and radiation therapy. Malignant pericardial effusion may be treated with pericardiocentesis or a more permanent surgical intervention. Complications of cancer treatment are becoming more varied because of the use of standard and newer immunologic therapies. Palliative care is increasingly appropriate as a part of the team approach for treating patients with cancer. (*Am Fam Physician*. 2018;97(11):741-748. Copyright © 2018 American Academy of Family Physicians.)

The National Cancer Institute estimates that 14.5 million persons in the United States have cancer, and that number could reach 19 million by 2024.[1] Family physicians should be familiar with the most prevalent oncologic emergencies because stabilization is often necessary, in addition to referrals for managing the underlying malignancy and initiating palliative measures.[2] Some oncologic emergencies can be categorized as metabolic, hematologic, structural, or treatment related *(Table 1[5])*.

Metabolic

TUMOR LYSIS SYNDROME

Tumor lysis syndrome is triggered by rapid, acute cell lysis caused by cancer treatment. It is often associated with che-

July 9, 2019 AHD Hospital Medicine

AHD Orientation

Acute Coronary Syndrome:

1. Describe the pathophysiology of STEMI and NSTEMI. Specifically describe the difference between type 1 and type 2 NSTEMI.
2. Understand the importance of risk stratification scores to determine likelihood of adverse events and optimal management strategy. Describe the GRACE and TIMI scores. Know the scores that indicate need for early invasive strategy.
3. Know the appropriate management of STEMI and NSTEMI based on ACC/AHA guidelines.
4. Describe the abnormal values for high-sensitivity troponin assays, and how to use the new assay in the evaluation of a patient with chest pain who rules out, rules in, and is in the indeterminant range for this biomarker.

Heme/Onc Emergencies:

Fill in the table as follows:

	SVC syndrome	Spinal cord compression	Tumor lysis syndrome	Hyperviscosity syndrome	TTP
Clinical syndrome					

GLOMERULAR DISEASES

	NEPHROTIC SYND.	vs	NEPHRITIC SYND.
CLINICAL	peripheral edema periorbital edema hypercoagulable state ↑infxn risk ONSET = insidious		HTN edema ↑JVP oliguria ONSET = abrupt
LABS	hypoalbuminemia HLD		normal / slightly ↓Alb ↓GFR
URINE	proteinuria > 3.5g/24h ⊕fatty casts/lipiduria		+/- mild proteinuria ⊕RBC casts/hematuria ARF = azotemia, oliguria

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS (=ANTIBODY-MEDIATED GN)

acute vs insidious
- hematuria, ↓UOP, HTN, edema
- fatigue, edema

SEROLOGY / MICRO CATEGORY	ANTI-GBM DISEASE	PAUCI-IMMUNE GN	IMMUNE COMPLEX GN		MIMICS
	linear deposit of Ig along GBM	absence of Ig	granular deposits of Ig		
SEROLOGY	anti-GBM Ab ⊖ANCA nL C3	⊕ANCA neg anti-GBM nL C3	low C3 neg anti-GBM ⊖ANCA	nL C3 neg anti-GBM ⊖ANCA	nL C3 neg anti-GBM ⊖ANCA
MICRO	linear Ig & C3	sparse or absent Ig/C3	granular Ig & C3	granular Ig & C3	sparse or absent Ig/C3
	Anti-GBM disease Goodpasture's	Wegner's (granulomatosis w/ polyangiitis) Microscopic polyarteritis nodosum Renal-limited crescentic GN		IgA nephropathy HS Purpura Fibrillary GN	Malignant HTN HUS / TTP Interstitial nephritis Scleroderma crisis Toxemia Atheroemboli

CATEGORIES

OVERFLOW PROTEINURIA
= increased excretion of LMW proteins can occur w/ marked overproduction of a particular protein → increased glomerular filtration & excretion

*CAUSES:
- MOST COMMON → Ig light chains in MM
- lysozyme (in AML)
- myoglobin (rhabdo)
- free Hgb (intravasc hemolysis)

TUBULAR PROTEINURIA
= tubulointerstitial diseases can lead to ↓ reabsorptive capacity of the proximal tubule & up to 2g proteinuria per day due to impaired tubular absorption of filtered albumin and loss of tubular proteins (β_2 microglobulin)

GLOMERULAR PROTEINURIA
= sensitive marker for glomerular disease
= develops 2/2 increased filtration of macromolecules (eg albumin) across the glomerular capillary wall

DIPSTICK TESTING: can quantify proteinuria, but ONLY measures albuminuria!

∴ good screening test for proteinuria but cannot quantify proteinuria or detect immunoglobulin light chains

⇩

if positive for protein, check a protein/creatinine or albumin/creatinine ratio

24 HR vs UPCR | UACR

PRO
- gold standard to measure protein excret.

CONS
- collection is difficult w/ errors

PROS
- easy collection
- good correlation

CONS
- heavily influenced by urine creatinine concentration & by total daily
- urine protein excretion can vary throughout the day

Differentiate onset and duration of vertigo in Menniere disease and BPPV. What are signs of central and peripheral vertigo?

	CENTRAL VERTIGO	PERIPHERAL VERTIGO
Nystagmus	can be any direction *can reverse direction when pt looks in direction of slow component	unidirectional horizontal w/ torsional component fast component toward normal ear
Response to provocative manuvers	short or no latency	latent 2-5s
N/V	variable +/- with HA	variable, can be worst at onset
Other neuro signs	CN issues present NO deafness/tinnitus	none +/- deafness/tinnitus
Posture dependency	severe instability ataxic gait +/- fall with walking	unidirectional instability gait preserved

<u>BPPV</u>

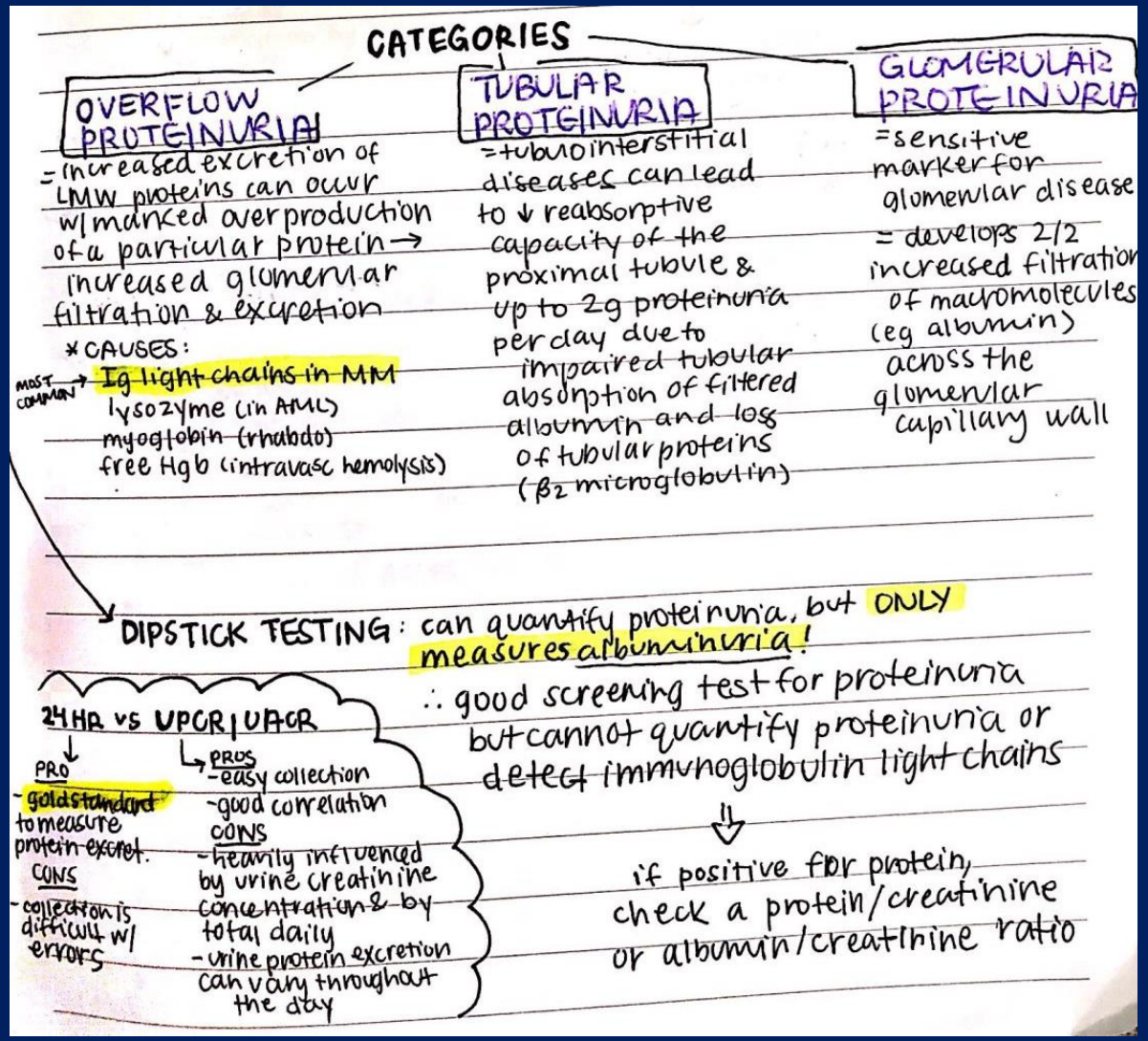
- recurrent
- brief episodes (secs)
- w/ predictable head movements or positional changes
- nystagmus: U/L, horizontal w/ torsional component, fast component to normal ear
- no neuro symptoms
- no auditory symptoms
- testing: reproducible w/ Dix Hallpike

<u>Meniere disease</u>

- recurrent
- episodes mins-hours
- spontaneous, no predictable factors
- nystagmus: U/L, horizontal w/ torsional component, fast component to normal ear
- no neuro symptoms
- +/- auditory symptoms: ear fullness/pain, U/L hearing loss, tinnitus
- testing: audiometry shows U/L low frreq sensorineural hearing loss

Expectations

- Prepare!
- Read articles ***and*** write out objectives before 9 am Tuesday morning each week
- Send via email to your education scholar before 9 am
- Organize to re-review when you take care of patients
- Review before practice test of the month



Question 2.

All residents are expected to prepare for AHD each week by reading articles, MKSAP and writing out the learning objectives for each lecture.

Categorical interns are required to email their objectives to their Education Scholar by what time on Tuesday morning?

A. 6 am
B. 7 am
C. 8 am
D. 9 am
E. 12:30 pm

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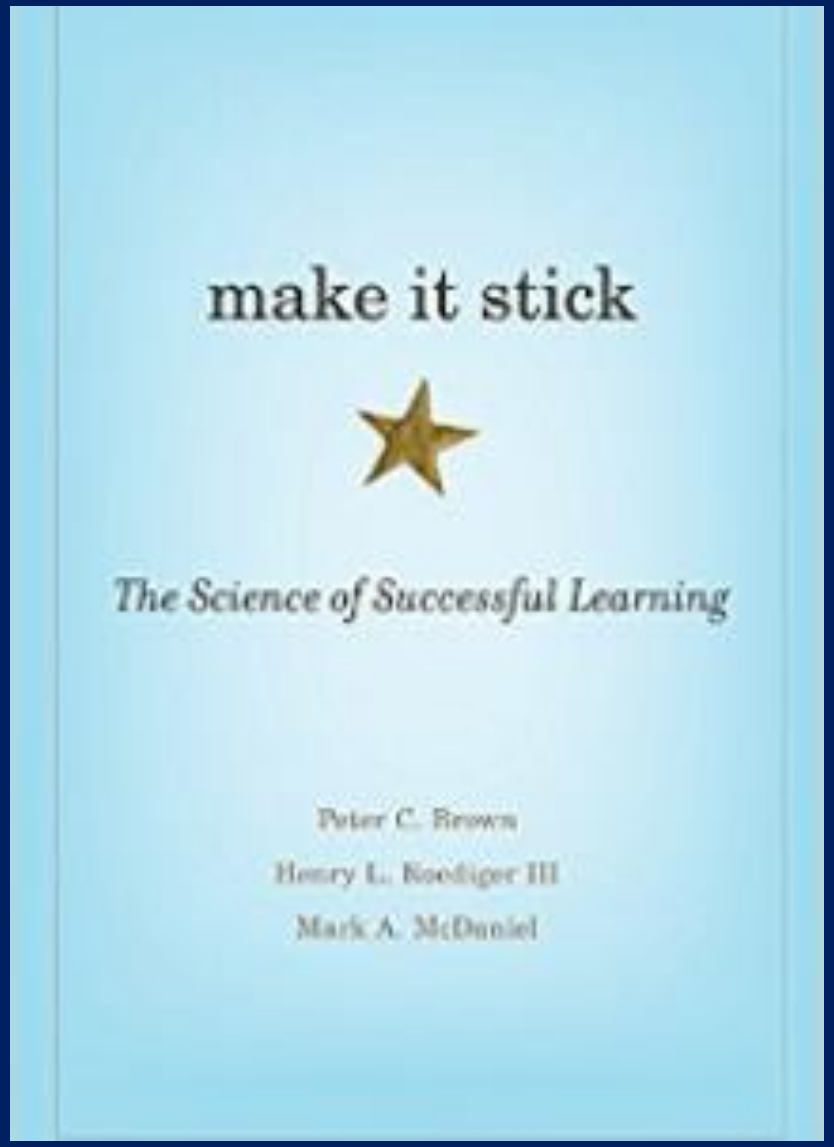
The Science of Learning

- There is no such thing as a personal learning style (visual, auditory, etc.)
- Learning is an acquired skill!
- Learning is deeper and more durable when it *requires effort.*
- ONLY reading and re-reading text gives a false security of mastery.
- Retrieval practice (recalling from memory) is more effective than re-reading and better when spaced out over time.



AHD “Lectures”

- Case-based instruction
- Audience response
- Numbered Pair Partners!
- Think about your answer/discuss with your partner, and may get a chance to share with the group if your number is called!
- Quizzing helps knowledge retention.

Question 3.

Active learning is important for knowledge retention. One of the new techniques for active learning in AHD is Numbered Pair Partners.

What is the process for Partner Pairs in AHD if I arrive too late to get assigned to my partner at 9:30?

A. I will join a previously paired team to make a threesome.
B. I will not participate in the numbered paired partners for that AHD.
C. I will take a number by myself and be responsible for answering the question without a partner.

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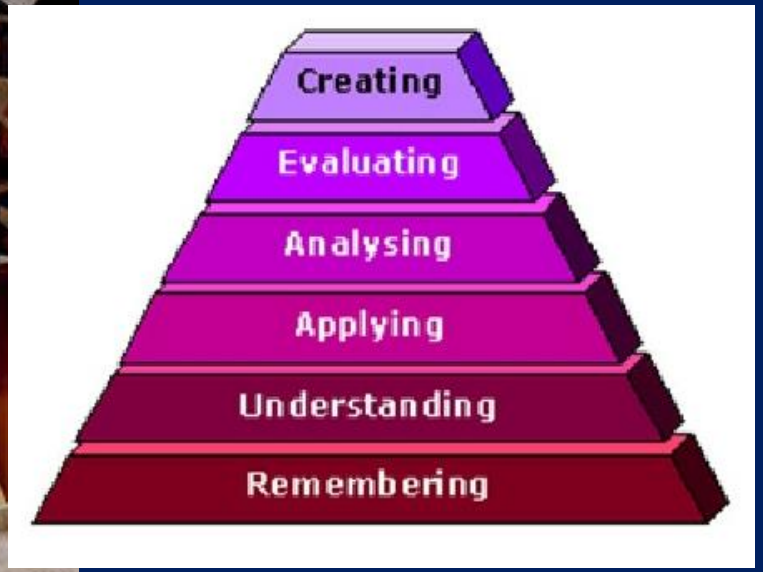
8 Habits for Effective Study

- 108 minutes a day
- Massive amount of information
- Retrieve from long-term memory
- Use for problem solving



1. Place for Study

- Alone vs. Partner
- Minimize distractions
- Try someplace different if you are having trouble

2. Set a Goal for Each Study Session

- Set a Specific, Achievable Goal
- 10/2 Rule
- How will you know you have mastered your goal?
- Test yourself
- Persevere until you pass!

3. Hand Write Your Notes

- Highlighting and underlining is useless
- Don’t cut and paste or copy verbatim
- Physically writing preferred over typing
- Summarize in your own words
- Sort/Categorize
- Color-code

4. Draw Pictures

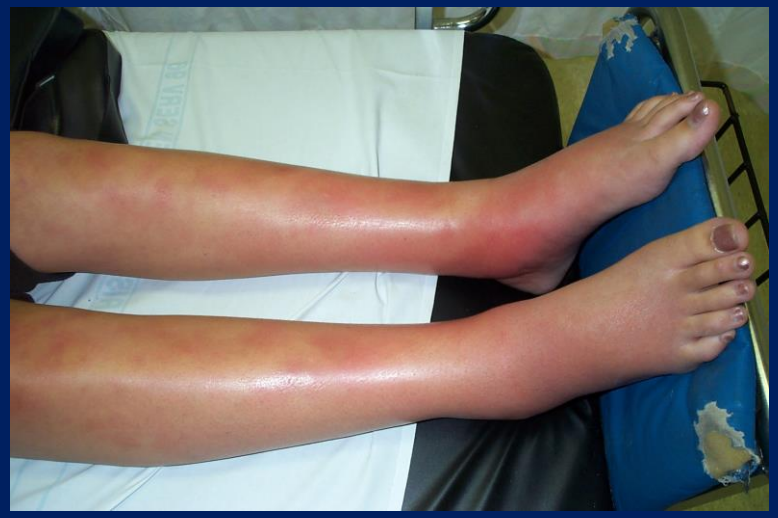
- Sketch and label
- Flowchart
- Diagram
- Cutting and pasting pictures or tables ***does not*** count!

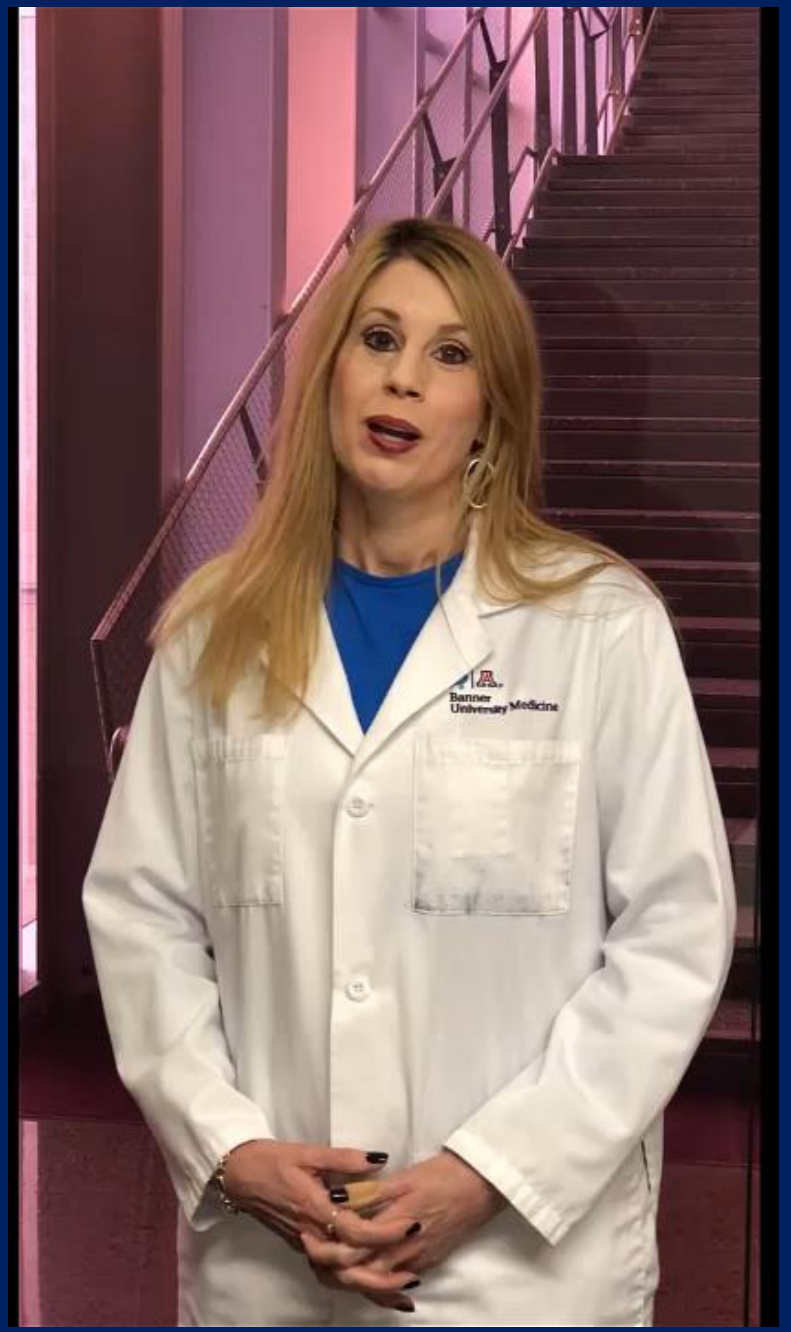
5. Rhyme, Rhythm, Chant

- Familiar Tune
- Rap/Chant
- Poem
- Mneumonic

6. Movement

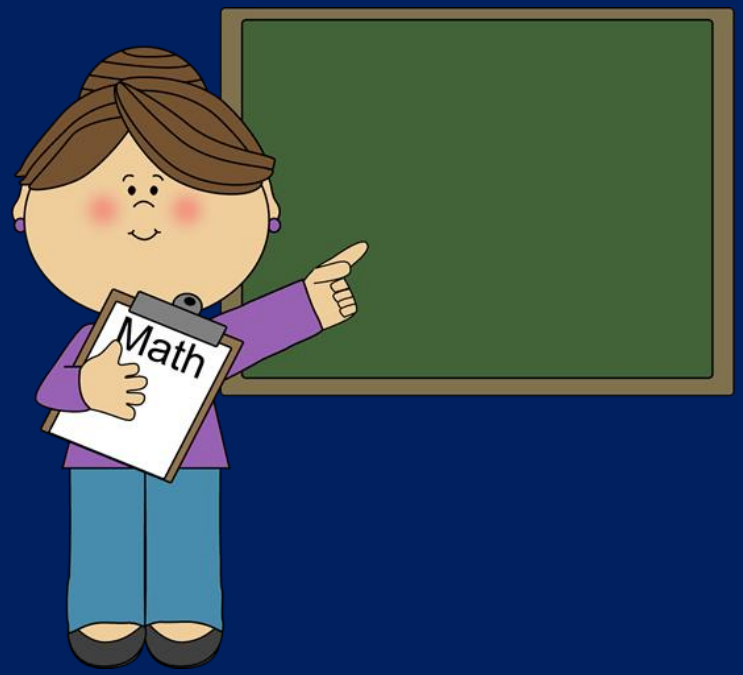
- Make up motions
- Change positions
- Take stretch breaks





7. Teach/Talk Out loud

- Partner share
- Video yourself
- Narrate PP slides
- Call your Grandma!



8. Repeat, Review, Reflect

- Repetition required
- Review your notes and correlate with patient care
- Reflect back to reinforce
- Wonder about what you know and what you don’t know- this requires reflection

Question 4.

Studying requires dedicated time, organization and planning, and active participation for durable knowledge retention.

Which of the following study strategies best helps you recognize deficits in your knowledge so that you can work to correct them?

A. Setting a goal for each study session
B. Drawing a flowchart
C. Making up a chant or jingle
D. Making a movement or gesture
E. Teaching a concept out loud

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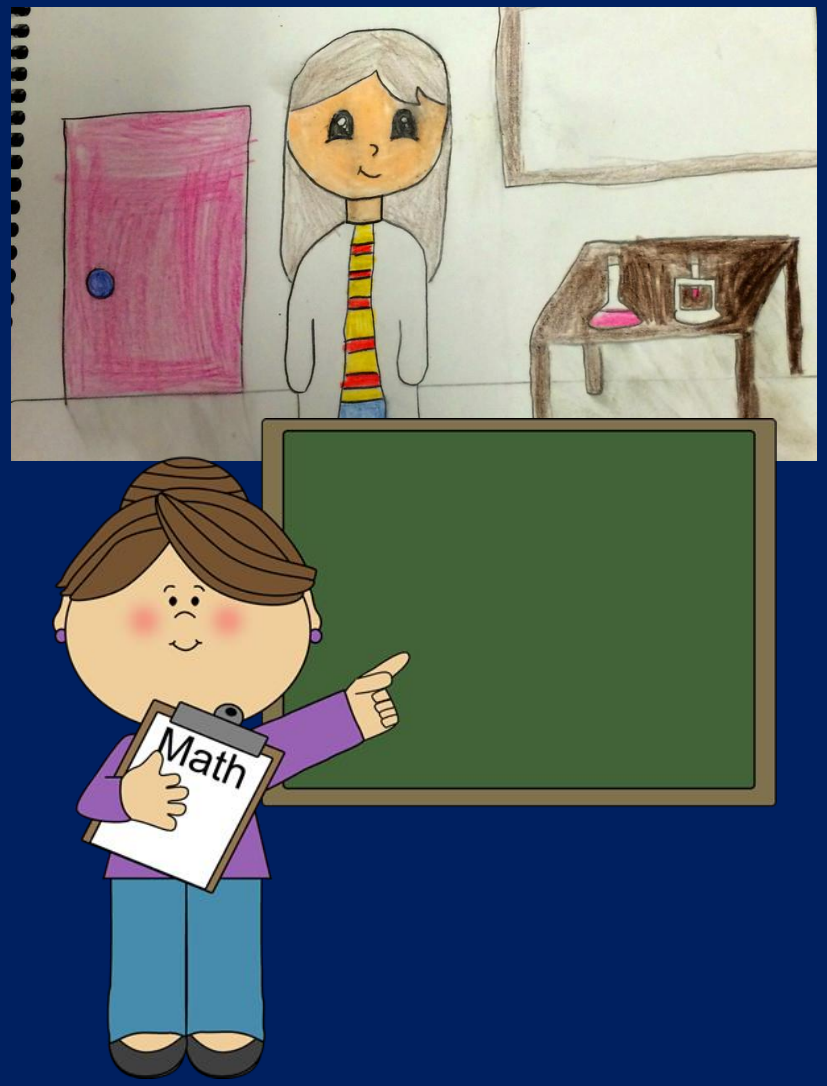
Table 1: Established Risk Factors and Risk Modifiers for Sudden Cardiac Death (SCD) in Patients with Hypertrophic Cardiomyopathy (HCM)

Risk	Details
Established Factor*	
Prior SCD event due to ventricular tachycardia or ventricular fibrillation	This is the highest-risk group
Unexplained syncope	A history of unexplained syncope occurring 6 months prior to clinical evaluation is associated with an increased risk of SCD
Maximal left ventricular-wall thickness	There is a linear relationship between left ventricular-wall thickness and SCD. A thickness ≥30 mm is an independent risk factor for SCD, with a 20 % increase in the relative risk of death at 10 years compared to the general HCM population
Nonsustained ventricular tachycardia	In select patients, especially young patients, a history of nonsustained ventricular tachycardia on ambulatory monitoring is a marker for increased risk of SCD. There may be value in longer-term monitoring to assess the burden of nonsustained ventricular tachycardia in unclear cases
Abnormal blood pressure response to exercise	Many patients with HCM have an abnormal blood pressure response to exercise, defined as a decrease in systolic pressure of 20 mmHg or a failure to increase systolic blood pressure by 20 mmHg while exercising. A normal response to exercise has a high negative predictive value. An abnormal response to exercise is useful in conjugation with other risk factors
Family history of SCD	A history of documented SCD in at least one first-degree family member is associated with an increased risk of SCD. There is currently no conclusive evidence that a history of SCD in second-degree and more distantly-related family members should influence the decision to place an implantable cardioverter-defibrillator
Modifier§	
Late gadolinium enhancement on cardiac magnetic resonance imaging	This is a marker of cardiac fibrosis and recent data have supported its role in identifying patients at elevated for SCD. Any late gadolinium enhancement places patients at increased risk of SCD. If found, a thorough evaluation for other risk factors should be performed
Apical aneurysm	While rare, a dilated and thinned left ventricular apex is associated with significant scarring. Patients with HCM and apical aneurysms often present with monomorphic ventricular tachycardia
Genetic mutations	Genetic mutations targeting the myosin heavy chain (*MYH7* gene) appear to increase the risk of SCD, however the mutation has poor positive predictive value

*These six risk markers are the best studied predictors of SCD in patients with HCM. They are the cornerstone in determining a patient's risk of SCD.

Indications for ICD in patients with HOCM

- Groups 1-8
  - Flowchart/Table
- Groups 9-16
  - Picture/Diagram
- Groups 17-23
  - Rhyme/Rhythm/Chant
- Groups 24-33
  - Movement/Dance
- Groups > 33
  - Pick a category



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Testimonials

- Iza Aguayo
- Hospitalist, Banner MD Anderson, Gateway
- ITE scores increased by 35% from PGY 2-3

- One Note
- U World Questions

Testimonials

- Dan Hannon
- Hospitalist, Scottsdale Thompson Peak
- ITE scores increased by 50%

- Organized a plan for studying
- Wrote out and reviewed notes in his own words

Testimonials

- Mayur Patel
- Cardiology Fellow PGY-5
- Board Certified
- ITE scores increased by 60%

- Start with a broad foundation
- Teach others!

What’s YOUR plan?