PA LIKE A PRO

DRINKING OUT OF A FIRE HYDRANT:

HOW TO STUDY EFFECTIVELY IN PA SCHOOL

Savanna Perry, PA-C - The PA Platform

Seems exaggerated.

It is, but it isn't.

Tons of information. Really fast.

KEEP IN MIND

Study for the PANCE from day 1.

You might fail.

FIRST SEMESTER

C on first Anatomy test (barely)

Failed first pharmacology test (barely)

Learning Style

OLD METHODS MIGHT NOT WORK.

VARK LEARNING TEST

Gain insights to study better

VISUAL

Diagrams, charts, designs, NOT words

AURAL

Lectures, group discussion, speaking, talking

READ/WRITE

Words, Powerpoints, lists

KINESTHETIC

Simulation, video, demonstration, case studies, practice

Multiple times in multiple ways

@MASAA98

Be choosy about what resources you use, but go for a multimodal approach to make sure you're seeing that information in different ways.

Study Techniques

REPETITION

Re-listen, write things out, review the day's material and repeat in 24 hours

POMODORO

Take breaks! Set a timer for study time vs rest time and take away distractions

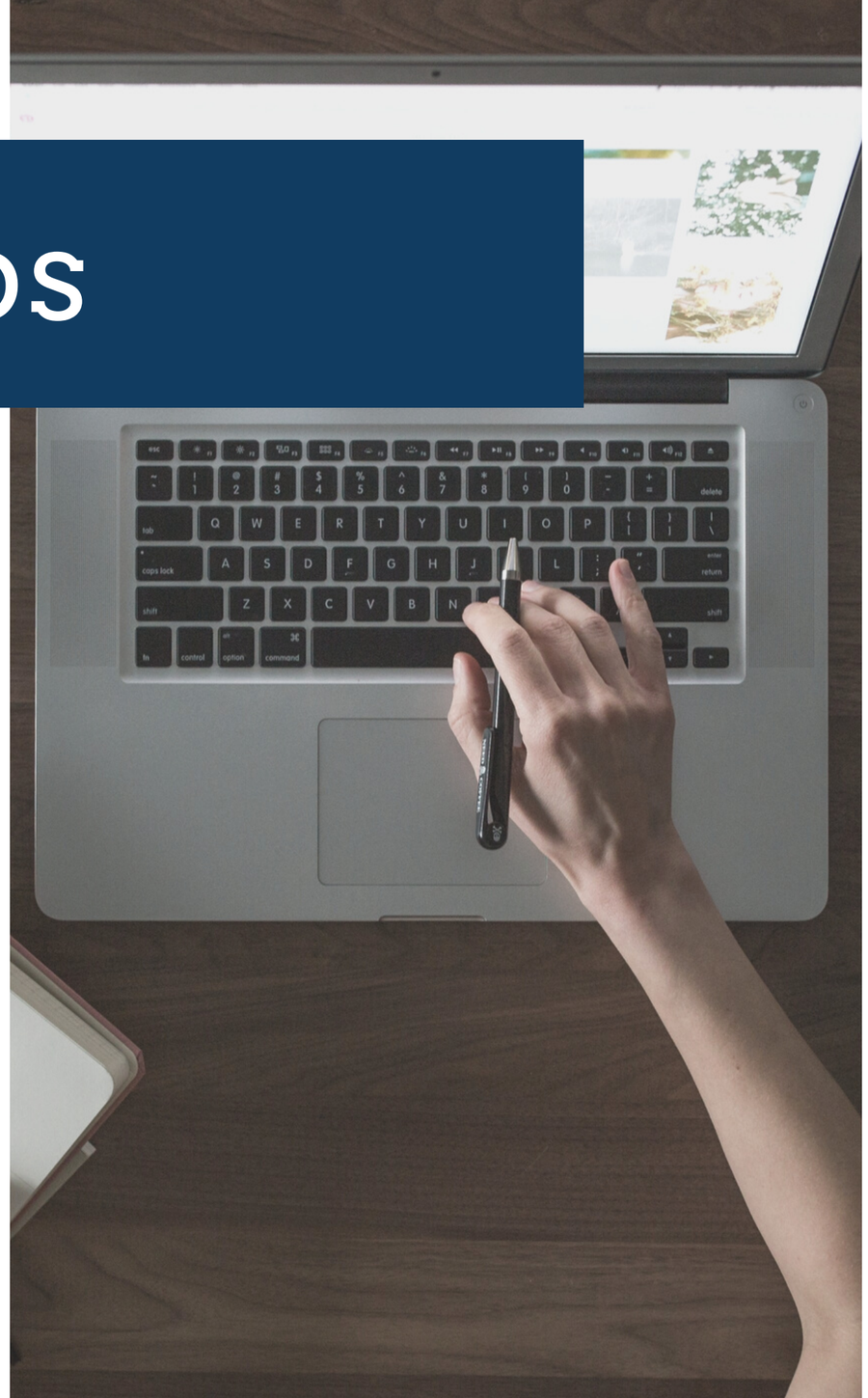
GROUP STUDY

Talk through concepts, quiz each other, teach each other

APPLICATION

Practice questions, create charts with highlights, teach someone else

Tips



SWITCH IT UP

Recognize when a study method isn't effective, and try something new.

ASK FOR HELP

Classmates, advisors, faculty, friends, family, internet

STICK TO WHAT WORKS

Once you do find a resource or method, use it until it doesn't work.

Don't sacrifice yourself!

SLEEP

Keep a regular bedtime. Will studying another 2 hours to sacrifice really impact your grade?

EAT WELL

Choose healthy foods, and take breaks for meals. Your brain needs fuel!

EXERCISE

Get up and active. Sitting in a classroom and studying all day can be exhausting. Wake up your muscles!

How I Studied

NOTE TAKING

Staying engaged in lecture by taking notes on important points and pearls

STUDY GUIDES

Putting notes into study guide form and sharing with my classmates

REVIEW PANCE RESOURCES

Make sure I was focusing on the most important topics

APPLICATION

Practice questions with explanations and discussing with others

PANCE ENT

rs of the Ears

aring Impairment/loss = acute or gradual onset
- Conductive or sensorineural
- Weber test — sensorineural lateralizes to good ear
  - Lateralization to affected ear = conductive hearing loss
- Rinne test (sign)
  - Bone conduction > air conduction on affected side = conductive hearing loss
  - Sensorineural defects will have impairment of air conduction and bone conduction, but AC > BC
- Conductive hearing loss = impaired transmission of sound along external canal, across ossicles, and through the oval window
  - Often temporary
  - Increased threshold for perceived sound intensity
  - Possible causes
    - Cerumen impaction = may require removal by irrigation of use of wire loop or cerumen spoon
    - Acute otitis externa = exudate in central canal
    - Otosclerosis = abnormal new bone formation in oval window = surgery
    - Otitis media
- Sensorineural hearing loss = hearing loss secondary to disruption in nerves or mechanics of hearing
  - Presbycusis = most common cause of sensorineural hearing loss
    - Occurs with age in most people
    - Men > women
    - Probably genetic predisposition
    - May be caused or exacerbated by noise exposure
    - Usually involves higher frequencies
    - May be associated with tinnitus
    - Complaint = difficulty in sound discrimination
    - Tx = may or may not be helped by hearing aids
  - Meniere's disease
    - Recurrent, usually progressive group of symptoms = acquired hearing loss, tinnitus, and dizziness or vertigo
    - Unknown cause
    - Symptoms from distention of endolymphatic compartment of the inner ear
    - Clinically = hearing loss with episodes of tinnitus, vertigo, and nausea and vomiting
      - Attacks last from minutes to hours, and unsteadiness may last longer
      - Hearing loss may abate with each attack, but hearing rarely returns to pre-attack level
    - Tx – diuretics and salt restriction
      - Possibly surgery
  - Acoustic trauma/chronic noise exposure can cause sensorineural hearing loss
  - Acoustic neuroma (vestibular Schwannoma) = neoplastic cause of hearing loss
    - Females > males
    - Usually unilateral
    - Patient may present with insidious hearing loss
    - With progressive growth patient may develop tinnitus, vertigo, ataxia, and brainstem dysfunction
    - Dx = CT or MRI
    - Tx = surgical
- Drug-Induced hearing loss

Side Effects

Renin Angiotensin System Blockers	
ACE inhibitors: **Captopril** **Fosinopril** **Enlapril** **Lisinopril** **Quinapril** **Ramipril**	~ Postural hypOtension (requires careful monitoring) ~ Renal insufficiency ~ Hyperkalemia ~ Angioedema ~ Persistent dry cough ****Fetotoxic ****– do not use ACE inhibitors in pregos **CAPTOPRIL** = Cough Angioedema Proteinuria Taste changes hypOtension Pregnancy problems (teratogenic) Rash Increase renin Lower angiotensin II
ARBs: **Candesartan** **Telmisartan** **Losartan** **Valsartan**	~ Similar side effects to **ACE** ~ NO cough **Contraindicated in pregos**
Beta Blockers	
Carvedilol **Acebutolol** **Atenolol** **Metaprolol** **Propranolol**	Contraindicated in asthma, diabetes, severe bradycardia, PVD, COPD Taper off to avoid rebound angina or HTN ***Propranolol** = Combine carefully w **Verampamil** & **Diltiazem** bc additive negative ionotropic effects lead to bradyarrhthmias*
Diuretics	
Metolazone **Bumtanide** **Furosemide**	HypOvolemia (overdose on loop diuretics) **Furosemide:** Hyper/HypO - Calcemia Hyper – Glycemia, Uricemia HypO – Kalemia, Natremia, Magnesemia
Hydrochlorothiazide	Hyper – Calcemia, Glycemia, Lipidemia, Magnesemia, Uricemia HypO – Kalemia, Natremia Metabolic Alkalosis Muscle weakness Pancreatitis Vasodilation
Direct Vasodilators	
Diazoxide	Reflex tachycardia
Hydralazine	Toxicity in slow acetylators Tachycardia HA Dizziness Nausea Sweating Flushing Nasal Congestion Lupus-like-syndrome
Minoxidil	Tachycardia Massive fluid retention Hypertrichosis (excessive hair) EKG changes Pericarditis
Sodium Nitroprusside	By-product of metabolism is cyanide, which is metabolized by rhodanase to thiocyanate Cyanide poisoning in pts w poor diets (alcoholics) Drug broken down by UV light – wrap in foil

Renin-Angiotensin System Blockers

ACE inhibitors (vasodilators) – pril: Captopril, Enlapril, Quinapril, Ramipril, Lisinopril, Fosinopril QAM

Angiotensin receptor antagonists – sartan: Candesartan, Losartan QD, Telmisartan, Valsartan

Beta Blockers – lol
- Carvedilol (B & α blocker)
- Acebutolol
- Atenolol (selective B1)
- Metoprolol (selective B1)
- Propranolol (nonselective B & sympathetic antagonist)
- Labetalol (nonselective B & selective α1)
- Nadolol (nonselective B)
- Timolol (nonselective B)

Diuretics

Loop Diuretics (vasodilators): Metolazone, Bumtanide, Furosemide

Thiazide Diuretics: Hydrochlorothiazide

Direct Vasodilators

Arterial: Diazoxide, Hydralazine, Minoxidil — Arteries Hyd mal Di

Arterial & Venous: Sodium Nitroprusside

Ionotropic Agents

cardiac glycosides (dig): Digitoxin, Digoxin

PDE inhibitors/vasodilators (inotrope): Amrinone, Milrinone

sympathom (Do): Dobutamine, Dopamine

Aldosterone Antagonists

Spironolactone

Organic Nitrates

(vasodilators): Isosorbide Dinitrate, Isosorbide Mononitrate, Nitroglycerin, Amyl Nitrate

Ca2+ Channel Blockers – dipine

Dihydropyridines: Amlodipine, Nitredipine, Nicardipine

Diphenylamide: Verapamil

Benzothiazepine: Diltiazem

Dihydropyridine 1: Nifedipine

Dihydropyridine 2: Felodipine

DIGOXIN

Lanoxin, Lanoxicaps

CLASS: Positive Inotropic agents – Cardiac glycosides

ADMINISTRATION: IV or PO – only good oral inotropic agent
Digitalis compounds must be given slowly and in small doses

BINDING: 25% protein bound

METABOLISM: Small amount metabolized in liver

ELIMINATION: Majority excreted unchanged in urine

MOA: Inhibit Na+/K+ ATPase = increase in IC Na+ = decrease in activity for Na+/Ca2+ exchanger = increase in IC Ca2+ = higher amount of Ca2+ present during AP = more forceful contraction
Direct electrical effects on heart = decrease in AP duration, ectopic beats, and arrhythmias

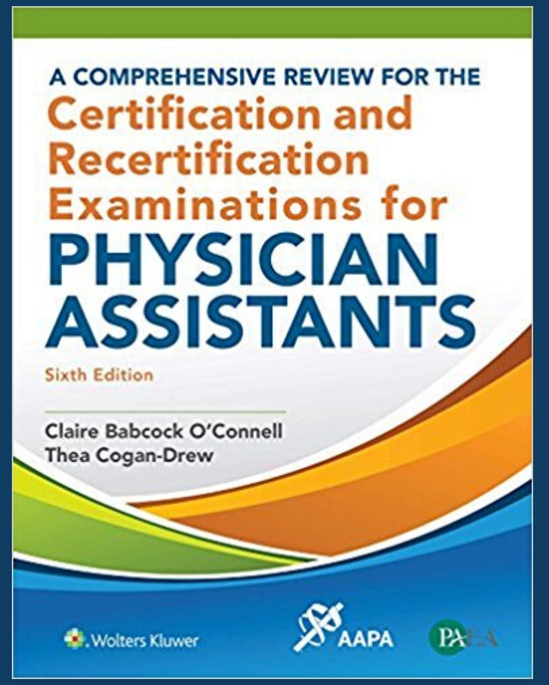
CLINICAL USES: Tx of chronic CHF
Pts w/ severe left-ventricular systolic dysfunction after initiation of diuretic, ACE inhibitor, & B blocker therapy
NOT in pts with diastolic or right-sided HF

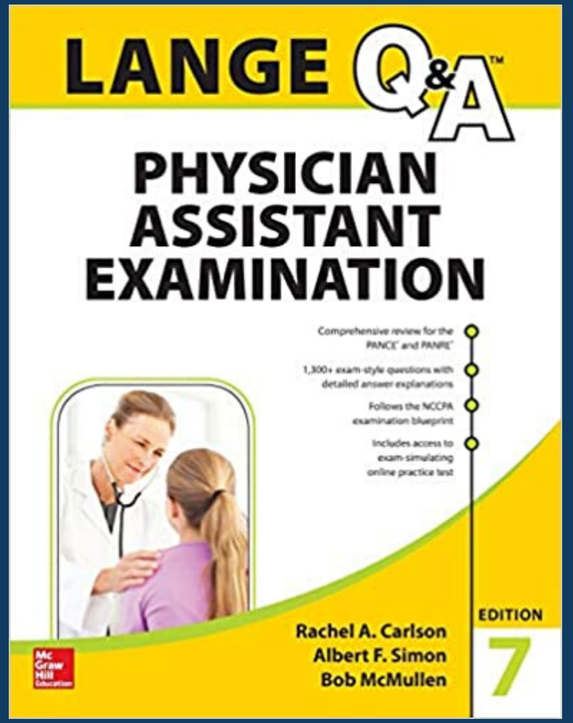
AE'S: Low therapeutic index = high risk
Cardiac = AV junctional rhythm, premature ventricular depolarization, AV blockade
Noncardiac = nausea, color vision abnormality, anorexia, diarrhea, disorientation, gynecomastia
Hypokalemia can increase the risk of toxicity by worsening arrhythmia
Monitor levels in renal insufficiency and adjust dosage if needed
Severe toxicity with ventricular tachycardia = give antiarrhythmic drugs and antibodies to digoxin (digoxin immune fab) to bind and inactivate drug

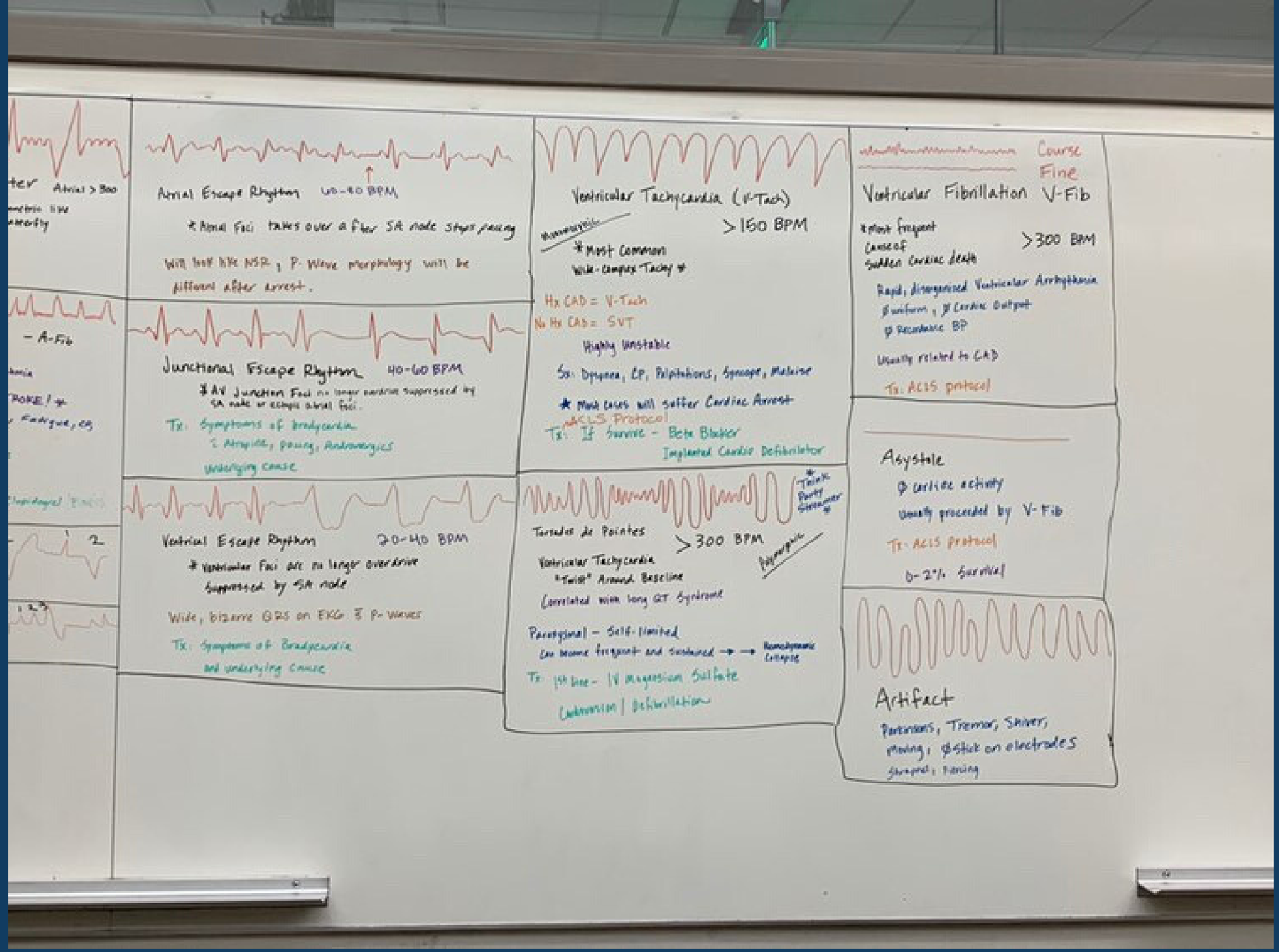
T1/2: 1.5 days = short = better Tx of toxic reactions

DRUG REACTIONS: Digoxin levels in plasma double when coadministered with quinidine
Digoxin intoxication can occur with quinidine, verapamil, amiodarone
K+-depleting diuretics, corticosteroids, and other drugs can increase digoxin toxicity

ONSET: 20 minutes – more rapid = useful in emergency situations





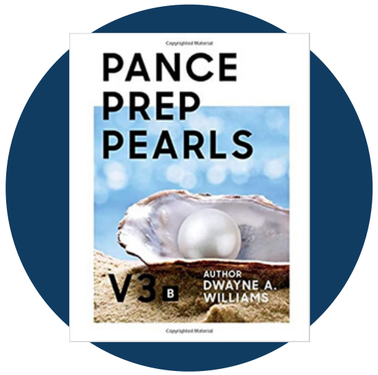


WHITE BOARDS

A time proven crowd favorite

Favorite Resources

CROWDSOURCED ON INSTAGRAM



PANCE Prep Pearls

Rosh Review, Lange

Picmonic, Osmosis, SketchyPharm

PACKRAT, Quizlet

Always here to help!

@THEPAPLATFORM

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