Getting the MOST out of YOUR Academic Half Day

Brenda Shinar
Cheryl O’Malley

10,000 hours of deliberate practice

Do the math….

- Intern call months:
- 65 hours/wk X 8* months (35 weeks) as an intern = 2275 hrs
- Intern non-call months:
- 40 hours/wk X 13 weeks = 520 hrs

- Senior call months: Next 2 years 52 weeks of call X 65 hrs/wk= 3380 hrs
- Senior non-call months:
- 46 weeks X 40 hrs = 1840 hrs

TOTAL hours in residency = 8015 hrs in 3 years

More Math…

- 10,000 hours – 8015 hours
  = 1985 hours left to be an expert

- Over 3 years = 661 hours per year

  - = 108 minutes per day of deliberate reading!

108 minutes

It is a job!


Charged if I do.
Charged if I don't.
TALK TO CHUCK
charles SCHWAB
$2.50 INITIAL FARE
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GET THE
JOB
YOU WANT
10 secrets to a successful job search

Job Description

Your Job

- To provide excellent care to patients today
- To engage in deliberate practice to prepare yourself to take excellent care of patients when unsupervised in the future.

Dr. Bob Raschke

Dr. Edwin Yu

Top 8 Barriers (and solutions) to being the SMARTY pants you dream of…

Dr. Amandeep Khurana

Dr. Your Name Here!

The Top 8 Barriers

8. I’m just a bad test taker no matter how much I study.

“I’m a bad test taker.”

- **Test Anxiety or Attention**
  - Evaluation and treatment is available!
  - If this is NOT your problem, then its one of the following…

- **Poor Study Skills and Habits**
  - I’m a bad test taker = I don’t know the answers
  - Many subjects are hard to learn and take considerable effort with appropriate study strategies

- **Inflated self-assessment of knowledge, skill, or ability**
  - **Most students are very poor at “meta-cognition” or knowing what they know!**
  - **It is very hard to correct an inflated sense of knowledge…**
  - **The key indicator of this problem is that you don’t do well on the test!**

The Top 8 Barriers

7. My fellowship goals are more important than learning internal medicine.
8. I’m just a bad test taker no matter how much I study.

“My fellowship goals are more important than learning internal medicine.”

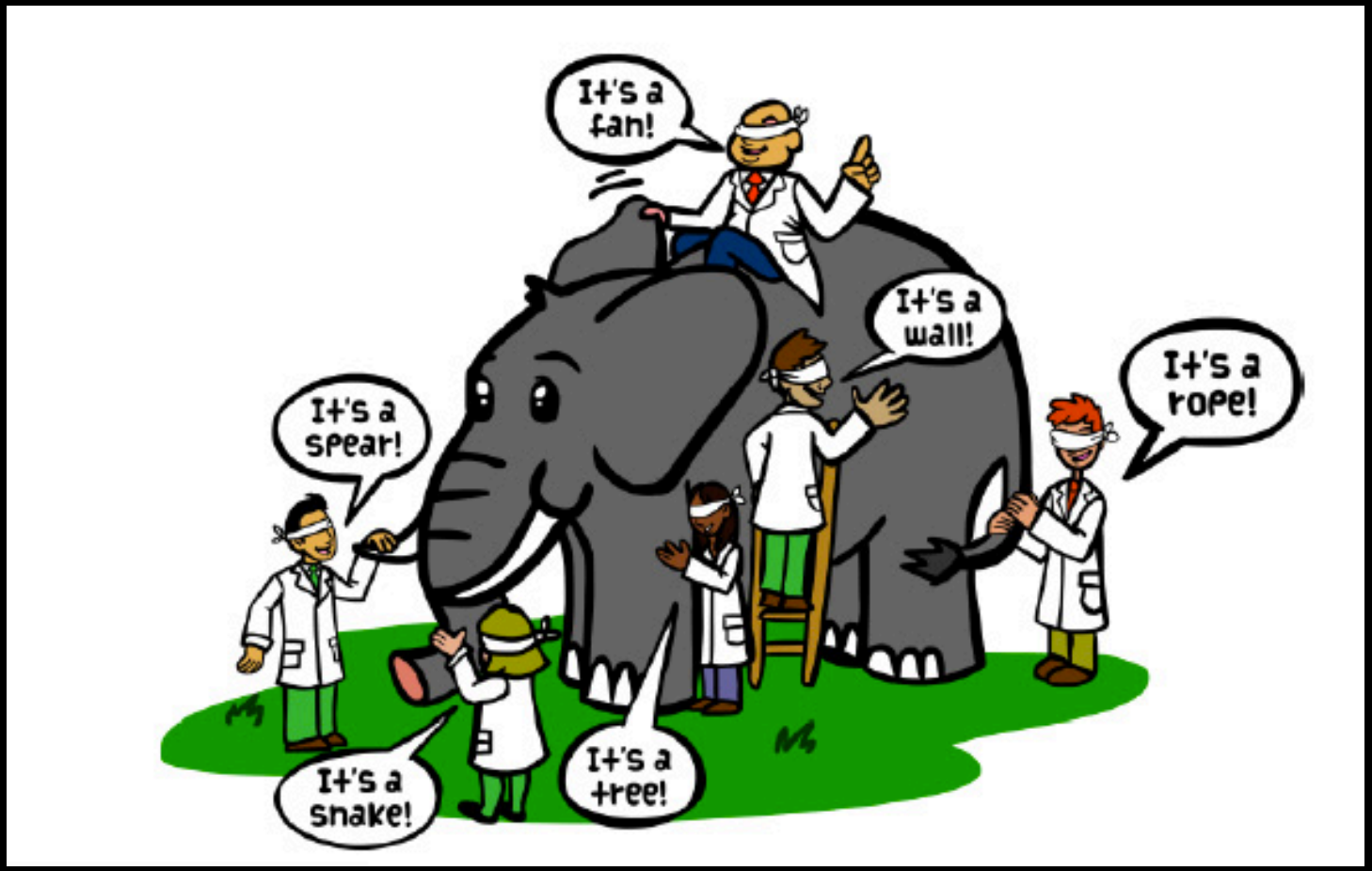
- Study and take an elective in your WEAKEST area.
- Fellowships are looking for well rounded fellows with excellent baseline GIM knowledge.
- Study it when you are on that rotation, be engaged and ask thoughtful questions.

- “Discipline is doing something you hate with the zeal and tenacity as if you loved it…”

The Top 8 Barriers

6. I learn best by doing questions rather than reading.
7. My fellowship goals are more important than learning internal medicine.
8. I’m just a bad test taker no matter how much I study.

“I learn best by doing questions rather than reading”



The Top 8 Barriers

5. The board exam is far away and I work best under pressure.
6. I learn best by doing questions rather than reading.
7. My fellowship goals are more important than learning internal medicine.
8. I’m just a bad test taker no matter how much I study.

“The Board Exam is far away and I work best under pressure”

- You always have a deadline!
- Weekly AHD articles/objectives
- Monthly MKSAP questions on electives (due 7 DAYS after finishing elective rotation)
- Timely completion is linked to your day off at BUMC-P

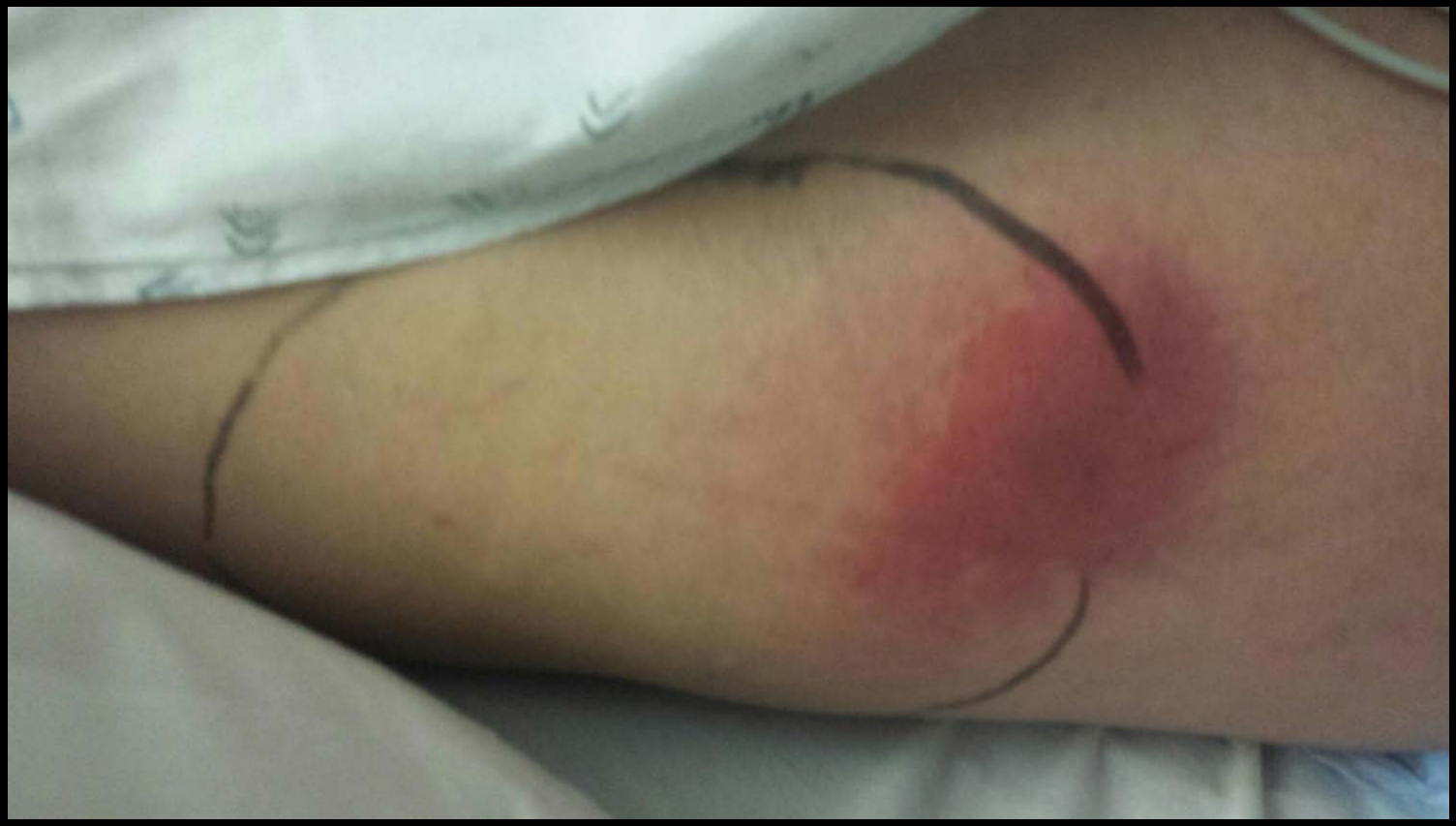
The Top 8 Barriers

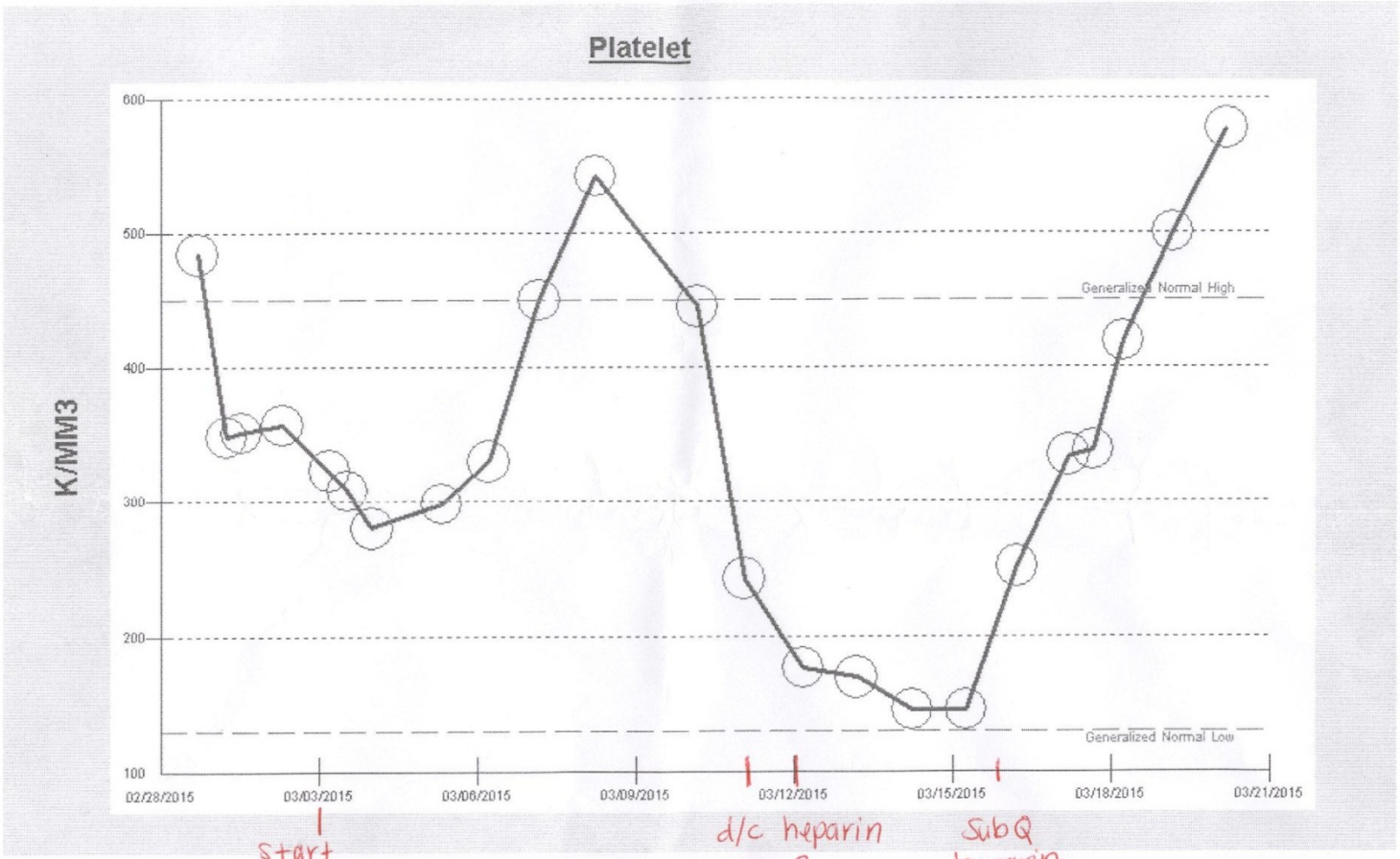
4. My medical knowledge is fine. (I don't know what I don't know).
5. The board exam is far away and I work best under pressure.
6. I learn best by doing questions rather than reading.
7. My fellowship goals are more important than learning internal medicine.
8. I'm just a bad test taker no matter how much I study.

“The faculty haven’t said anything bad about my medical knowledge, it must be fine.”

Board Pass Calculator

http://www.r-calc.com/administrator/calculatorPreview.aspx?isGrid=0&mobile=0&isTemp=0&calculator_id=de409368-643f-4f71-b96e-b040cb7478bf

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03/15/2015
03/18/2015
03/21/2015
start heparin infusion d/t DVT/PE
d/c heparin ~3/11 ? IVC filter on 3/12
SubQ heparin 5,000 u

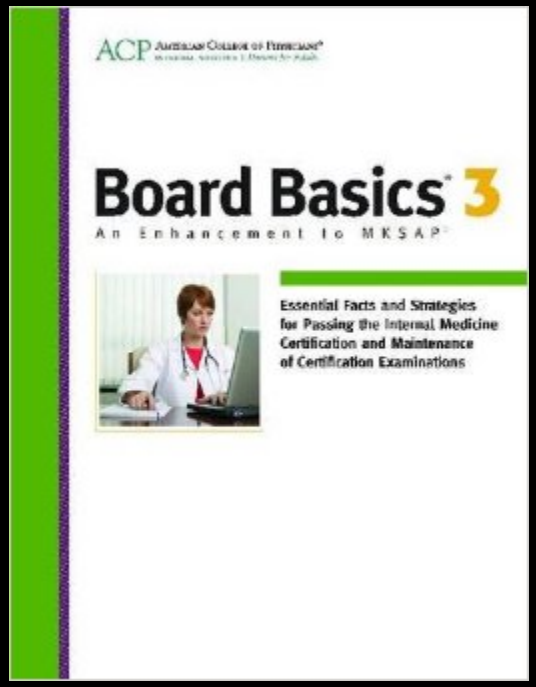
The Top 8 Barriers

3. I don’t know how to make the knowledge stick.
4. My medical knowledge is fine. (I don’t know what I don’t know).
5. The board exam is far away and I work best under pressure.
6. I learn best by doing questions rather than reading.
7. My fellowship goals are more important than learning internal medicine.
8. I’m just a bad test taker no matter how much I study.

“I don’t know how to make the knowledge stick.”

- Make your reading active!
  - *What do I already know about this topic?*
  - *What should I get out of this? (objectives)*
- Apply it- anything!
- Teach it!
- Take notes!
- Study your notes!
  - Before the end of the month test…
  - Before the ITE exam…

Review and grow your notes



The Top 8 Barriers

2. There is too much to read and I am not sure where to start or how to get organized.
3. I don't know how to make the knowledge stick.
4. My medical knowledge is fine. (I don't know what I don't know).
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“There is too much to read and I don’t know how to get organized”

Call Months	Non-Call Months
Your patients- Read whatever is handy/answers your ?. What else do you need to consider in the differential? Does this fit the typical illness script? Does our treatment match the current recommendations? If not, why?	Your patients- • MKSAP content for that topic • Daily discussions with the subspecialty attendings
Other: am report prep (seniors), teaching topics on rounds **	Rotation assignments
	Clinic articles
Academic ½ day weekly readings – What do you already know about the topic? What is the minimum that should you know (objectives)?	AHD readings: Objectives due?
Journal Club Grand Rounds Articles	Journal Club Grand Rounds Articles

Medical knowledge

Context

Experience

Patient's story

Data acquisition

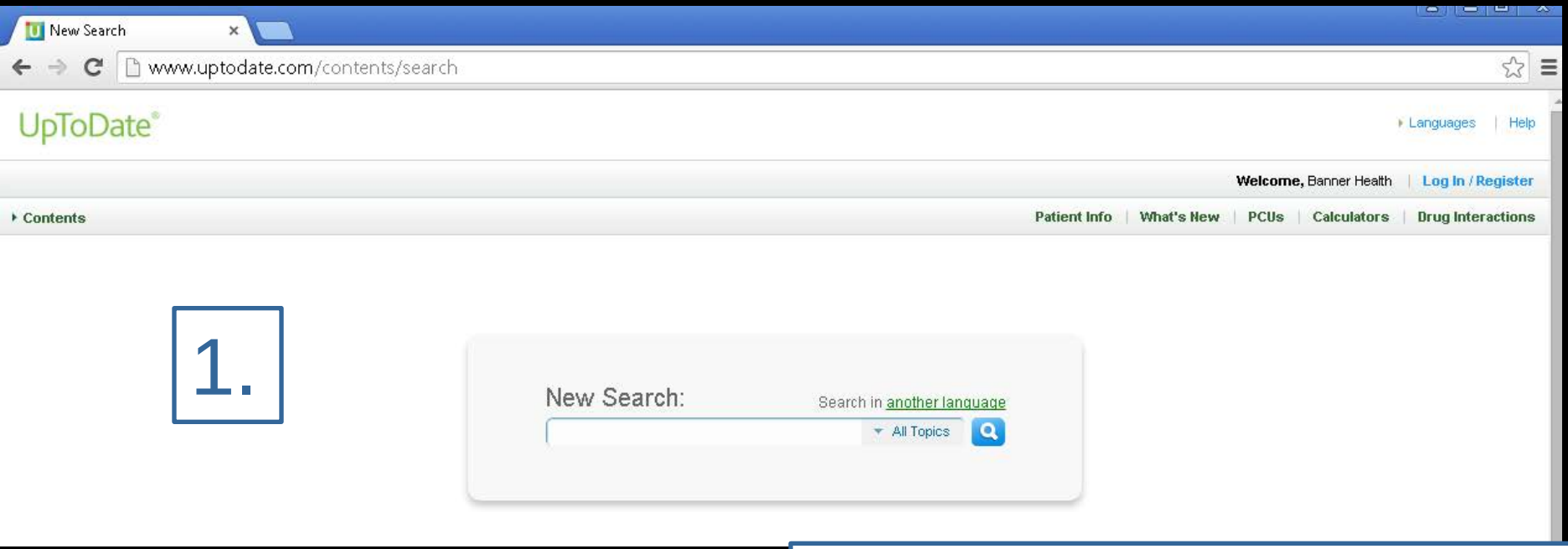
Accurate problem representation

Generation of hypothesis

Search for and selection of illness script

Diagnosis

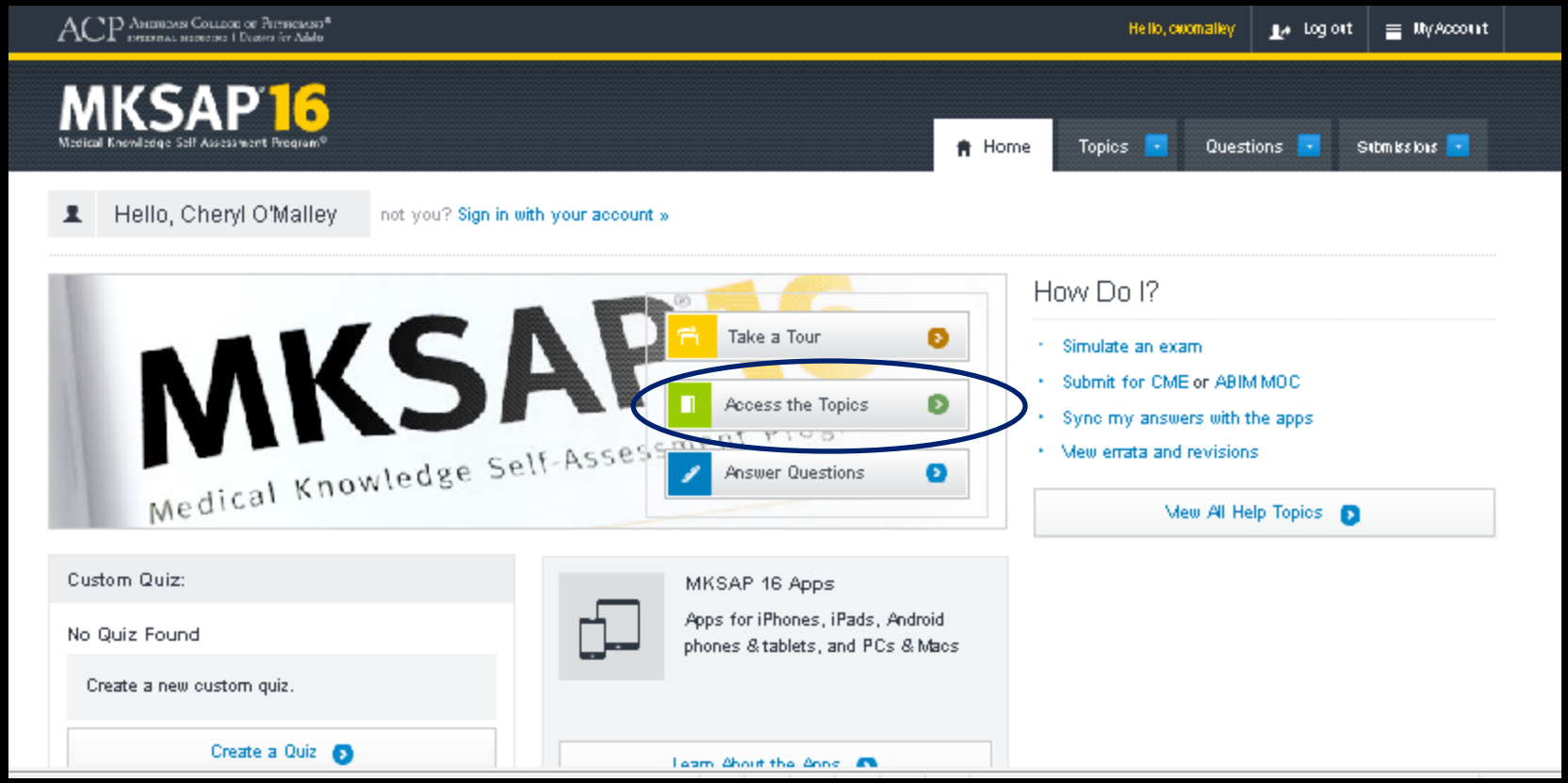
Read about your patients



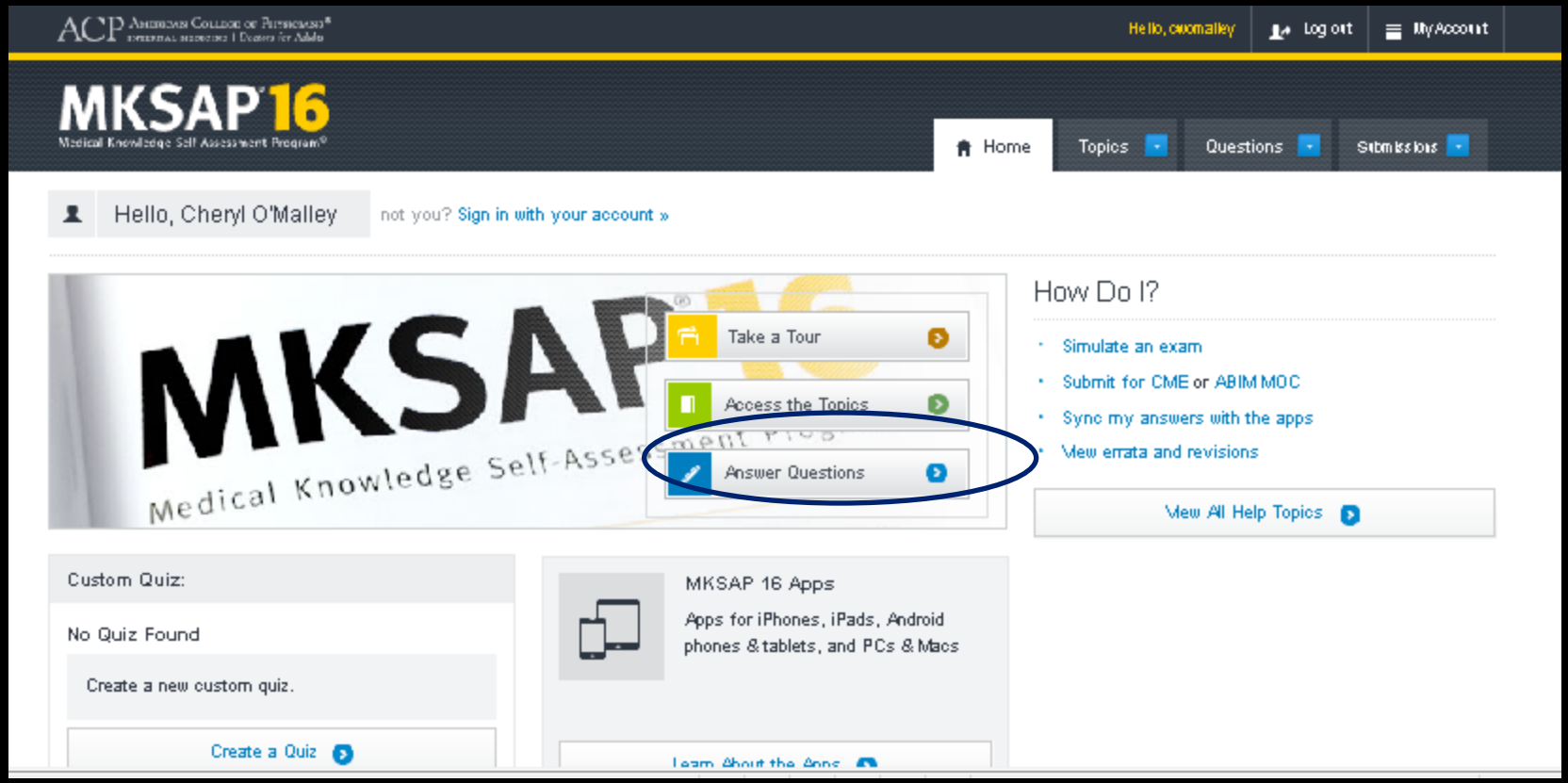
2. Review referenced studies

3. Look up your own search

Read the content



Answer 100 Questions →Submit



Academic Half Day

- Review the objectives
- Read the articles *(new “Read by QxMD” App folder “B-UMCP Academic Half Day”)
- Prepare answers to the objectives
- Review and talk about during your daily work
- Attend conference and listen
- Review and talk about in your daily work
- Return to the objectives, articles and video as needed

Goodsamim.com

“Gsphoenixim”

My Study Schedule 2015-2016

- Graduating Seniors need to have FINISHED by the time of the Awesome review in May!
- PGY1 and 2→ just make progress every month!
- MKSAP questions are due 7 days after a non-call month is over.

My Study Calendar

	July	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun
My rotation												
AHD topic												

The Top 8 Barriers

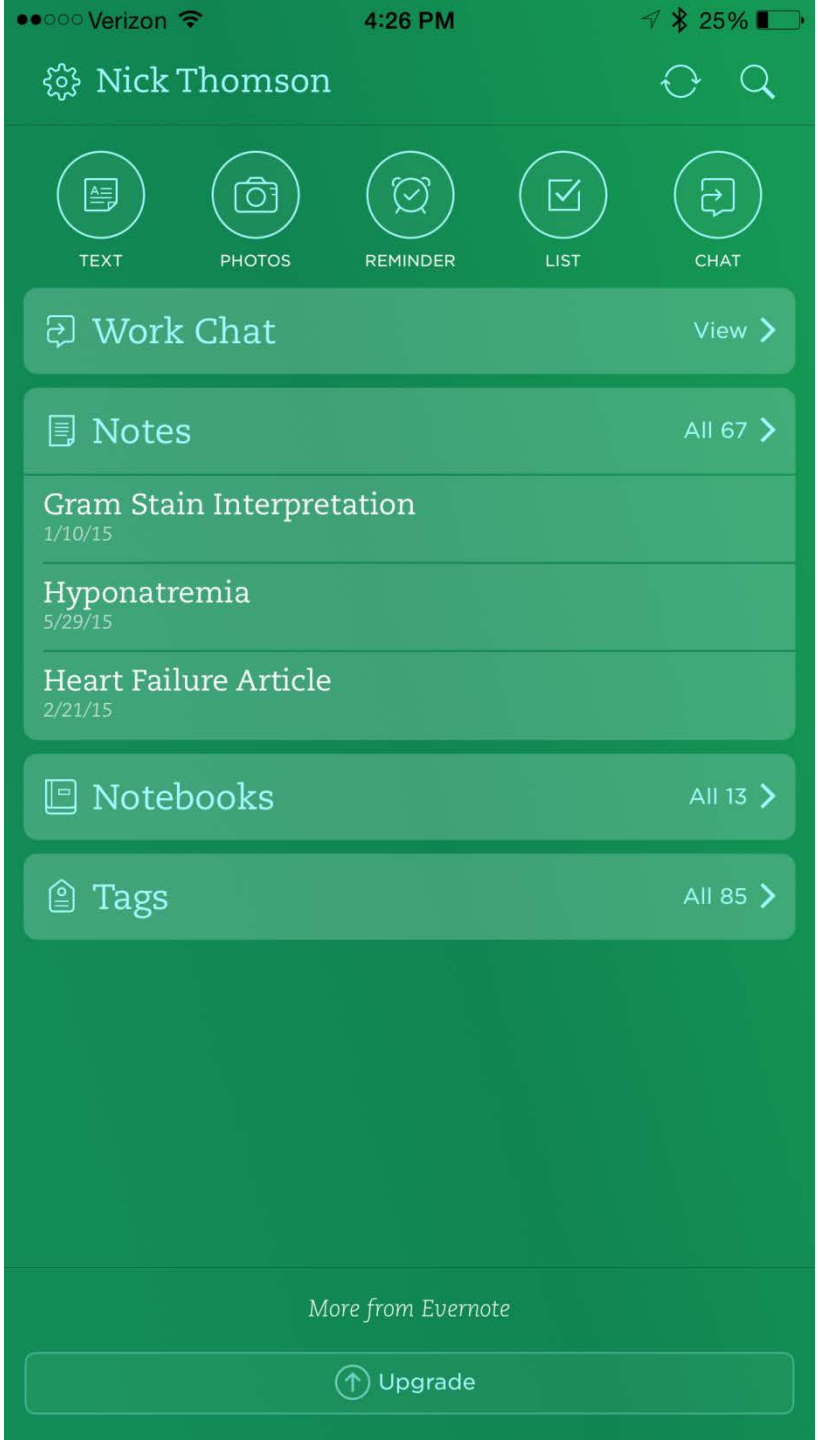
1. Patient care counts as studying and I just worked a 12+ hour day.
2. There is too much to read and I am not sure where to start or how to get organized.
3. I don't know how to make the knowledge stick.
4. My medical knowledge is fine. (I don't know what I don't know).
5. The board exam is far away and I work best under pressure.
6. I learn best by doing questions rather than reading.
7. My fellowship goals are more important than learning internal medicine.
8. I'm just a bad test taker no matter how much I study.

“Patient care counts as studying and I just worked a 12+ hour day”

- We know!!
- Find little islands of time in your day
- Have something handy all the time
- Use it and refer to it
- Slow and steady WINS the race!
- You ALWAYS have time for what is important to you.

Study Journal Using **Evernote**

By Nick Thomson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Gram Stain Interpretation
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Hyponatremia
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Heart Failure Article
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Tags
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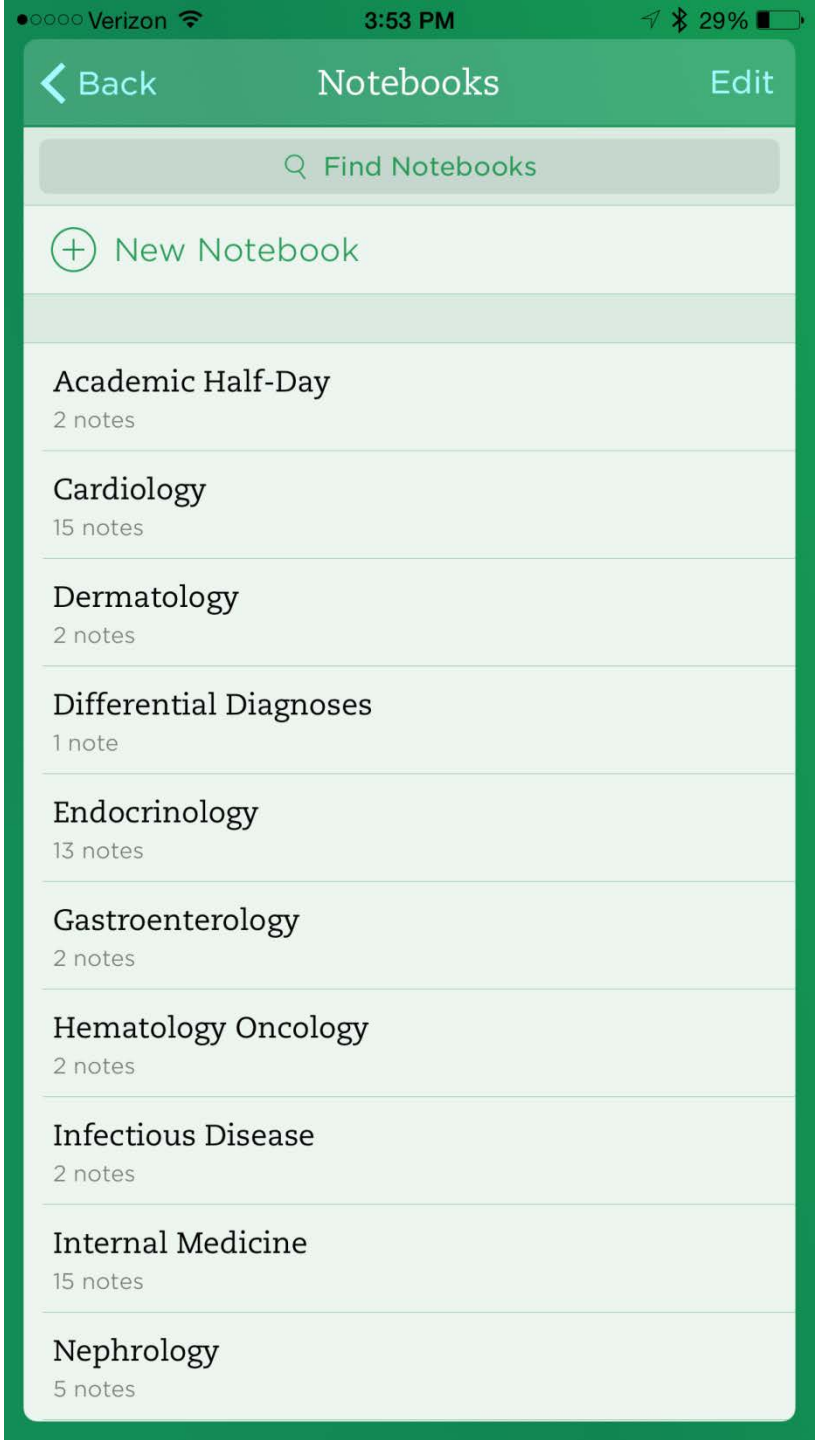
More from Evernote
Upgrade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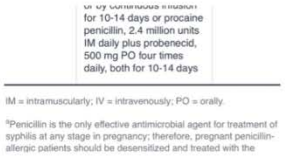
Academic Half-Day
2 notes
Cardiology
15 notes
Dermatology
2 notes
Differential Diagnoses
1 note
Endocrinology
13 notes
Gastroenterology
2 notes
Hematology Oncology
2 notes
Infectious Disease
2 notes
Internal Medicine
15 notes
Nephrology
5 notes

APRIL 2015 Options

STD Treatment
4/27/15



JANUARY 2015

Gram Stain Interpretation
1/10/15

Gram Stain Interpretation

pdf

Gram Stain Interpretation.pdf

BACTERIAL MORPHOLOGIES
LEVEL I
MINIMUM COMPETENCY-EVERYONE

MORPHOLOGY REPORTED	ORGANISM IMPLIED
• Gram-positive cocci clusters	Staphylococcus
• Gram-positive coccci pairs/chains	Streptococcus
• Gram-positive cocci	Staphylococus/Streptococcus
• Gram-positive rod	Any Gram-positive rod
• Gram-negative diplococci	Neisseria/Moraxella
• Gram-negative coccobacilli	Haemophilus/Bacteroides
• Gram-negative rod	Any Gram-negative rod
• Yeast cells	Yeast, usually Candida
• Yeast cells with pseudohyphae	Candida, not C. glabrata
• OTHER FINDINGS-LEAVE FOR REVIEW	

interpretation [microbiology | generalist]

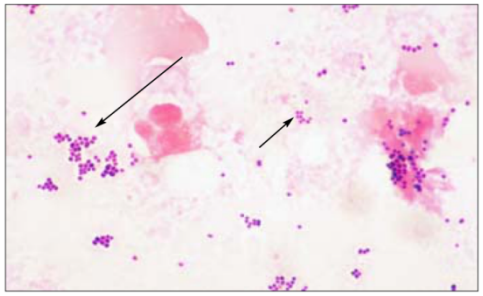
Interpretation of Gram Stains for the Nonmicrobiologist

Joan Barenfanger, MD, MMB, ABMM, and Cheryl A. Drake, SM(ASCP)
From the Department of Laboratory Medicine, Memorial Medical Center, Springfield, IL

- Guidelines for the interpretation of Gram stains
- Normal flora in respiratory secretions
- Normal flora in the female genital tract
- Presumptive identification of microorganisms from Gram stain
- Correlation of findings

Laboratories everywhere are being asked to do more with less. To enable the laboratory to offer increased services over an expanded period of time, many technologists with little experience in microbiology are now asked to perform and read Gram stains. Interpretation of Gram stains is notoriously difficult for nonmicrobiologists because such interpretation requires multiple observations and the judgment that comes with years of experience. This article offers objective criteria for interpreting the most commonly encountered Gram-stained specimens.

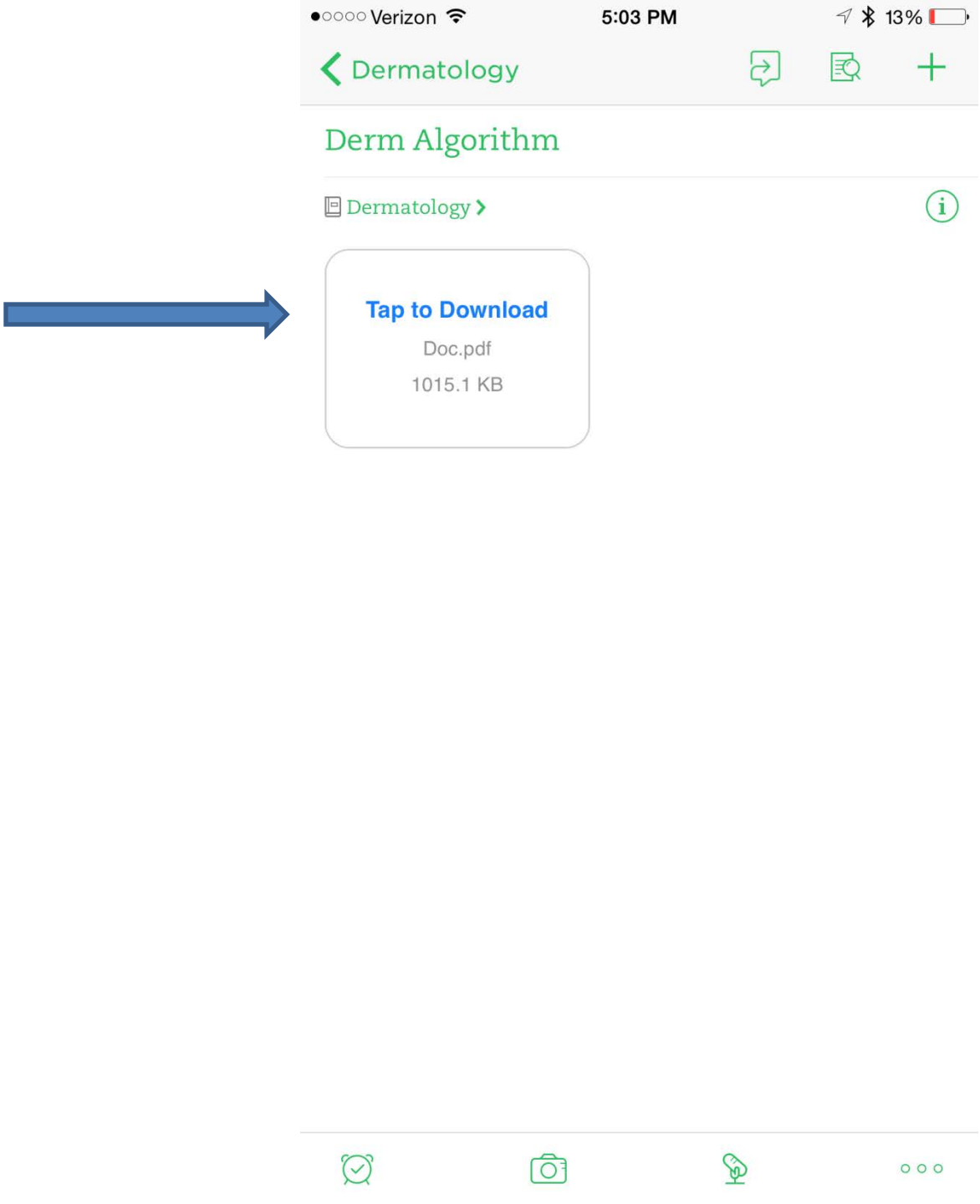
An adequate examination of a Gram-stained smear includes observing numerous representative fields. The fields containing neutrophils yield the most useful information. A minimum of 1 minute should be spent examining a smear; after that, judgment is needed. Obviously, a smear from the cerebrospinal fluid (CSF) with neutrophils deserves more time than an acellular smear. Similarly, more time should be spent on a specimen that was obtained by an invasive technique than on one that was easy to obtain.



[I1a] Staphylococci: gram-positive cocci in the tetrads (short arrow) and clusters (long arrow) as well as the nonspecific singles and pairs.

Generally, only 1 morphotype (bacteria with a certain Gram stain and shape, eg, gram-negative bacilli) is seen in a sterile site. For instance, only gram-negative diplococci are expected in a patient's CSF, not gram-negative diplococci as well as gram-positive cocci. If both of these morphotypes are seen, most likely the problem is over- or underdecolorization.

The problem with decolorization must also be considered when the shape of the organism does not "fit" the Gram stain. Please refer to Images 1a through 1j for Gram stains of characteristic morphotypes (all images ×100 oil except G and J). For instance, gram-positive cocci seen in tetrads and clusters or in long chains are highly characteristic of staphylococci and streptococci, respectively [T1] [I1a] [I1b], but may appear gram-negative because of overdecoloration. Similarly, classic gram-positive diplococci are lancet-shaped or pointed at the outside ends [T1] [I1c]. If gram-positive diplococci flattened at the out-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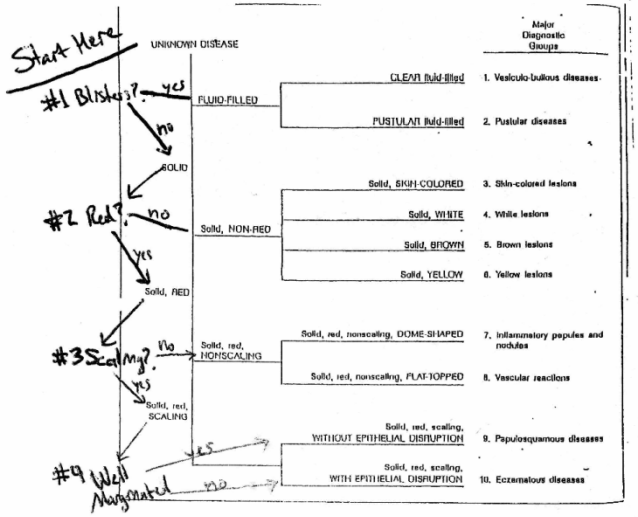
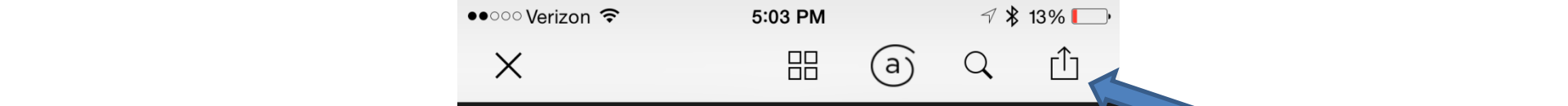
EVALUATING A SKIN LESION



Macule — < 2 cm diameter area of color change, with no palpable substance.
Patch — > 2 cm diameter macule.
Papule — Palpable mass < 1.5 cm diameter.
Nodule — Spherical enlargement of a papule > 1.5 cm diameter.
Plaque — Flat-topped palpable lesion > 1.5 cm diameter. Papule that is enlarged in 2 dimensions.
Wheal — Edematous papule or plaque. Nonlocalized fluid.
Vesicle — Fluid-filled papule < 1-1.5 cm diameter.
Bulla — > 1-1.5 cm vesicle.
Pustule — Vesicle packed full of polys (may or may not be sterile).
Excoriation — Scratchmarks.
Lichenification — Thickening secondary to chronic rubbing or scratching (seen only in eczematous diseases).

3

- Location / Distribution
- Size / Color
- Border / Margination
- Primary Lesion
- Secondary characteristics (scale, crust, excoriation)



I. Vesiculo-Bullous Diseases
- A. Vesicular disease
  1. Herpes simplex
  2. Varicella-zoster
  3. Vesicular tinea pedis
  4. Dyshidrosis (pompholyx)
  5. Scabies
  6. Dermatitis herpetiformis
- B. Bullous disease
  1. Poison-ivy-type contact dermatitis
  2. Bullous impetigo
  3. Erythema multiforme bullosum (Steven Johnson syndrome)
  4. Pemphigoid
  5. Pemphigus

II. Pustular Diseases
- A. True (soft pustules)
  1. Acne vulgaris
  2. Rosacea (acne rosacea)
  3. Bacterial folliculitis
  4. Fungal folliculitis
  5. Candidiasis
  6. Systemic bacterial infection (eg, gonorrhea)
- B. Pseudo-pustular (See white papules, Group IV)

III. Skin-Colored Lesions
- A. Keratotic (rough-surfaced lesions)
  1. Warts: verruca vulgaris, paronychial and plantar warts
  2. Actinic keratoses
  3. Seborrheic keratoses
  4. Corns and calluses
- B. Nonkeratotic (smooth lesions)
  1. Warts: genital warts, flat warts
  2. Basal and squamous cell carcinoma (with or without ulceration)
  3. Epidermoid ("sebaceous") cysts
  4. Lipomas
  5. Molluscum contagiosum
  6. Nevi: intradermal

IV. White Lesions
- A. White patches and plaques
  1. Pityriasis alba
  2. Pityriasis (tinea) versicolor
  3. Vitiligo
  4. Postinflammatory hypopigmentation
- B. White papules
  1. Milia
  2. Keratosis pilaris
  3. Molluscum contagiosum
  4. Sebaceous gland hyperplasia

V. Brown Lesions
- A. Brown macules
  1. Freckles
  2. Lentigenes
  3. Nevi: junctional
- B. Brown papules and nodules
  1. Nevi: compound and intradermal
  2. Seborrheic keratoses
  3. Melanoma
- C. Brown patches and plaques
  1. Cafe-au-lait patches
  2. Postinflammatory hyperpigmentation
  3. Giant congenital nevi
- D. Generalized hyperpigmentation
  1. Secondary to systemic disease
  2. Secondary to medication
  3. Postinflammatory hyperpigmentation

VI. Yellow Lesions
- A. Smooth yellow lesions
  1. Xanthelasma
  2. Necrobiosis lipoidica diabeticorum
  3. Sebaceous gland hyperplasia
- B. Rough yellow lesions
  1. Actinic keratoses
  2. Any crusted lesion (see vesiculo-bullous diseases, eczematous and insect bites)

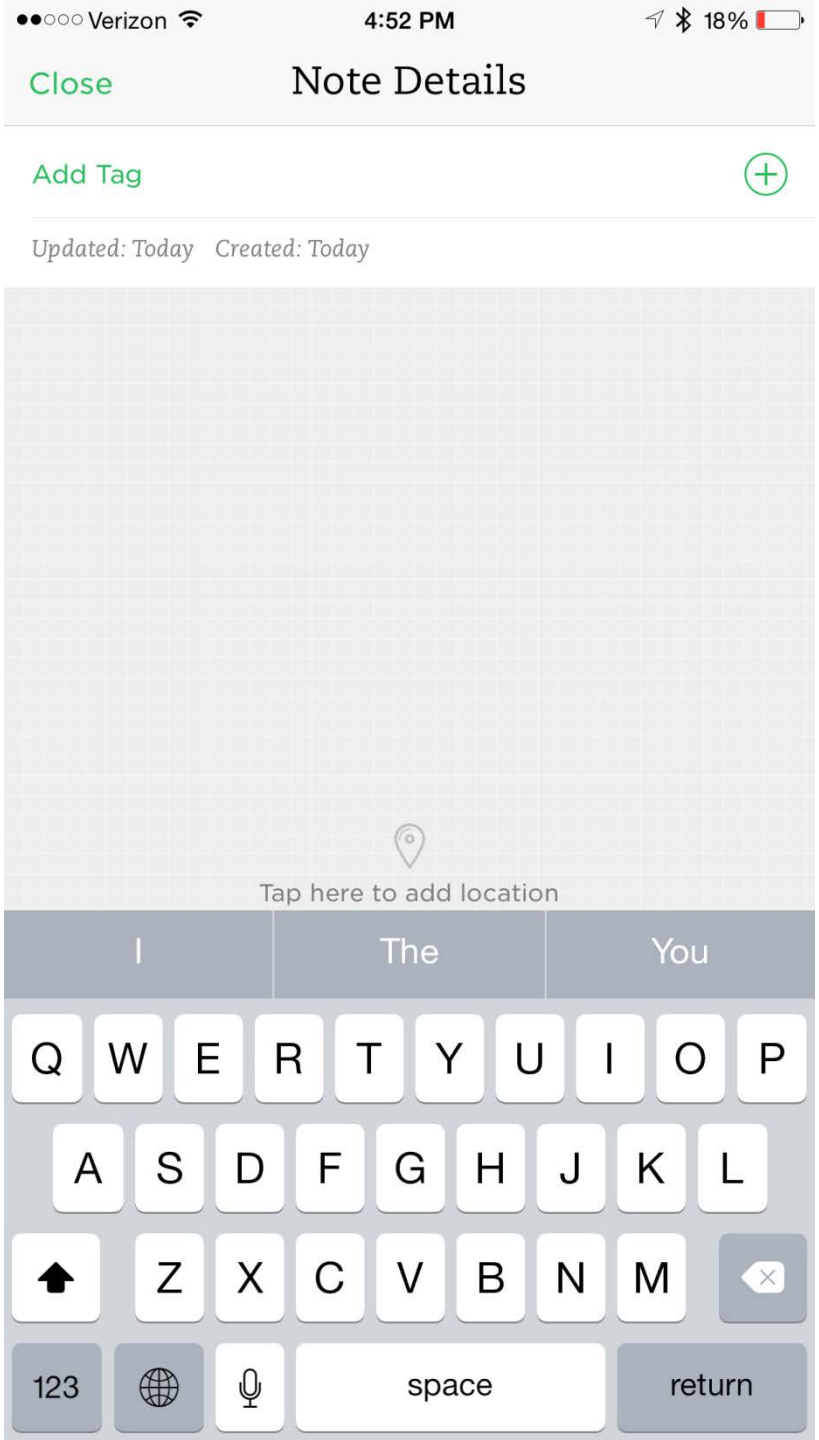
VII. Inflammatory Papules and Nodules
- A. Nonscaling red papules
  1. Insect bites
  2. Cherry angiomas
  3. Spider angiomas
  4. Granuloma annulare
  5. See nonconfluent papules, Group IX
- B. Nonscaling red nodules
  1. Furuncles
  2. Inflamed epidermoid cysts
  3. Hidradenitis suppurativa
  4. Erythema nodosum

VIII. Vascular Reactions
- A. Nonpurpuric (blanchable) lesions
  1. Toxic erythema: exanthems, medications, photosensitivity
  2. Urticaria: infection, medications
  3. Erythema multiforme
  4. Cellulitis (erysipelas)
- B. Purpuric lesions
  1. Vasculitis (PMN type, palpable purpura)
  2. Actinic ("senile") purpura
  3. Petechia and ecchymoses secondary to medications

IX. Papulosquamous Diseases
- A. Prominent plaque formation
  1. Psoriasis vulgaris
  2. Tinea: corporis, capitis, pedis and cruris
  3. Lupus erythematosis: discoid type
  4. Parapsoriasis-mycosis fungoides
- B. Nonconfluent papules
  1. Pityriasis rosea
  2. Lichen planus
  3. Syphilis: secondary
  4. Psoriasis: guttate type

Continued

X. Eczematous Diseases
- A. Excoriations prominent
  1. Atopic dermatitis (neurodermatitis, lichen simplex chronicus, infantile eczema)
  2. Dyshidrotic eczema
  3. Stasis dermatitis
  4. Tinea: cruris, capitis, pedis
  5. Psoriasis in atopic individuals
  6. Candidiasis
- B. Little or no excoriation
  1. Seborrheic dermatitis
  2. Contact dermatitis
  3. Xerotic (asteatotic) eczema
  4. Impetigo
- C. Eczematous reaction patterns (seen with more than one of the above eczematous diseases)
  1. Hand and foot eczema
  2. Diaper dermatitis
  3. Nummular eczema
  4. Exfoliative erythrodermatitis
  5. Autoeczematization (autosensitization, "id" reaction)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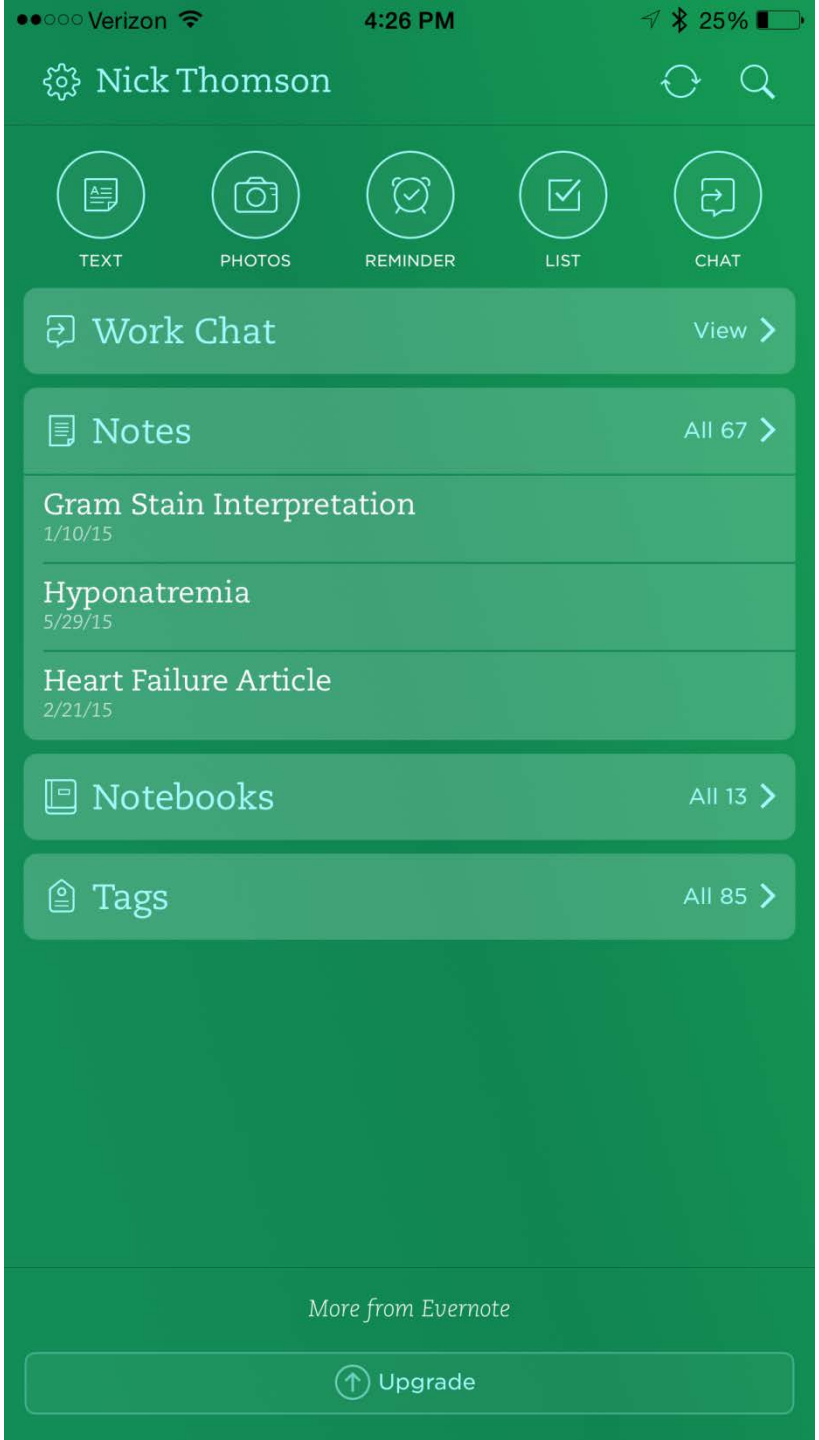
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Hyponatremia
5/29/15
Heart Failure Article
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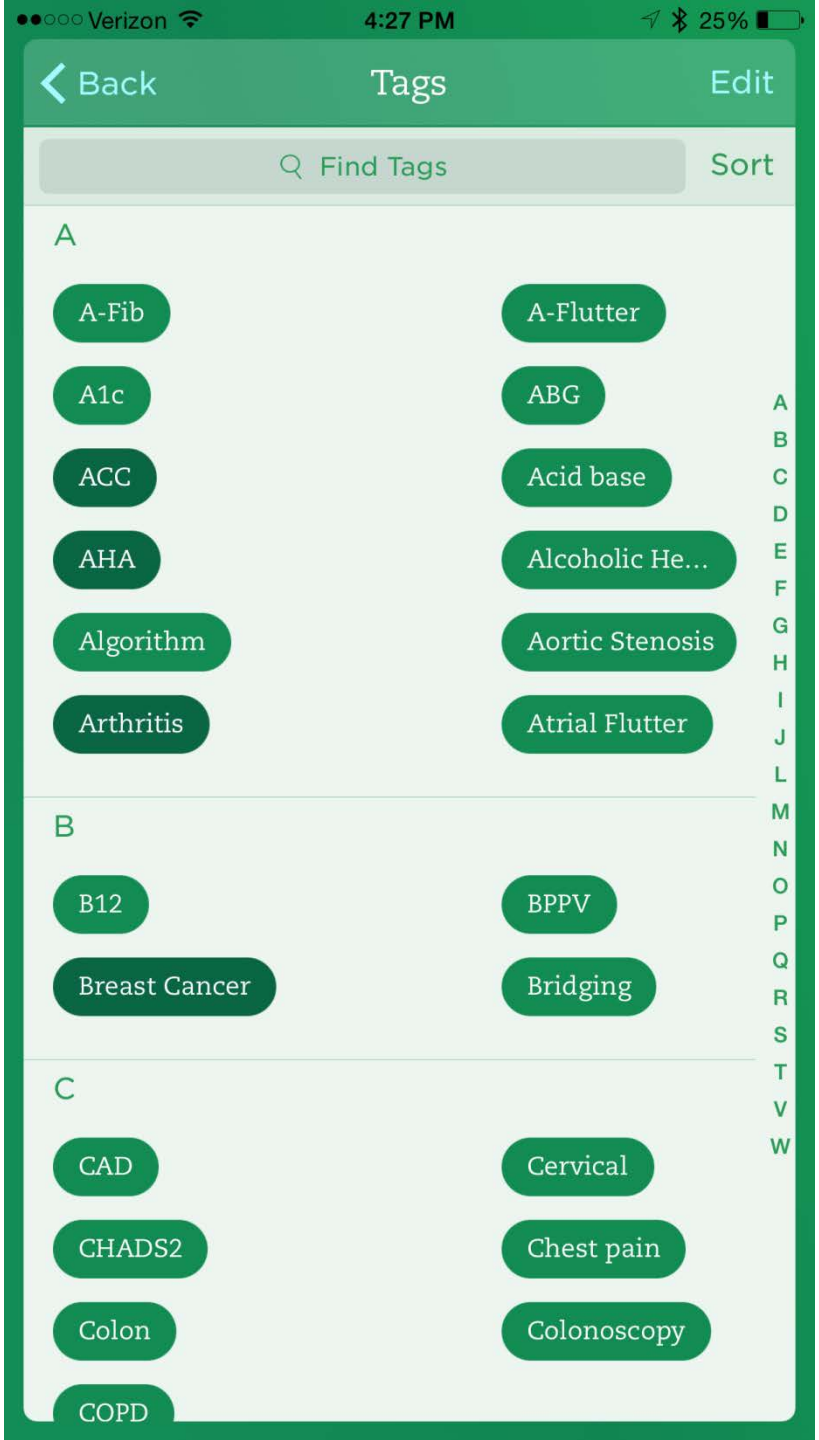
More from Evernote
Upgrade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Algorithm
Aortic Stenosis
Arthritis
Atrial Flutter
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B12
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Breast Cancer
Bridging
C
CAD
Cervical
CHADS2
Chest pain
Colon
Colonoscopy
COPD
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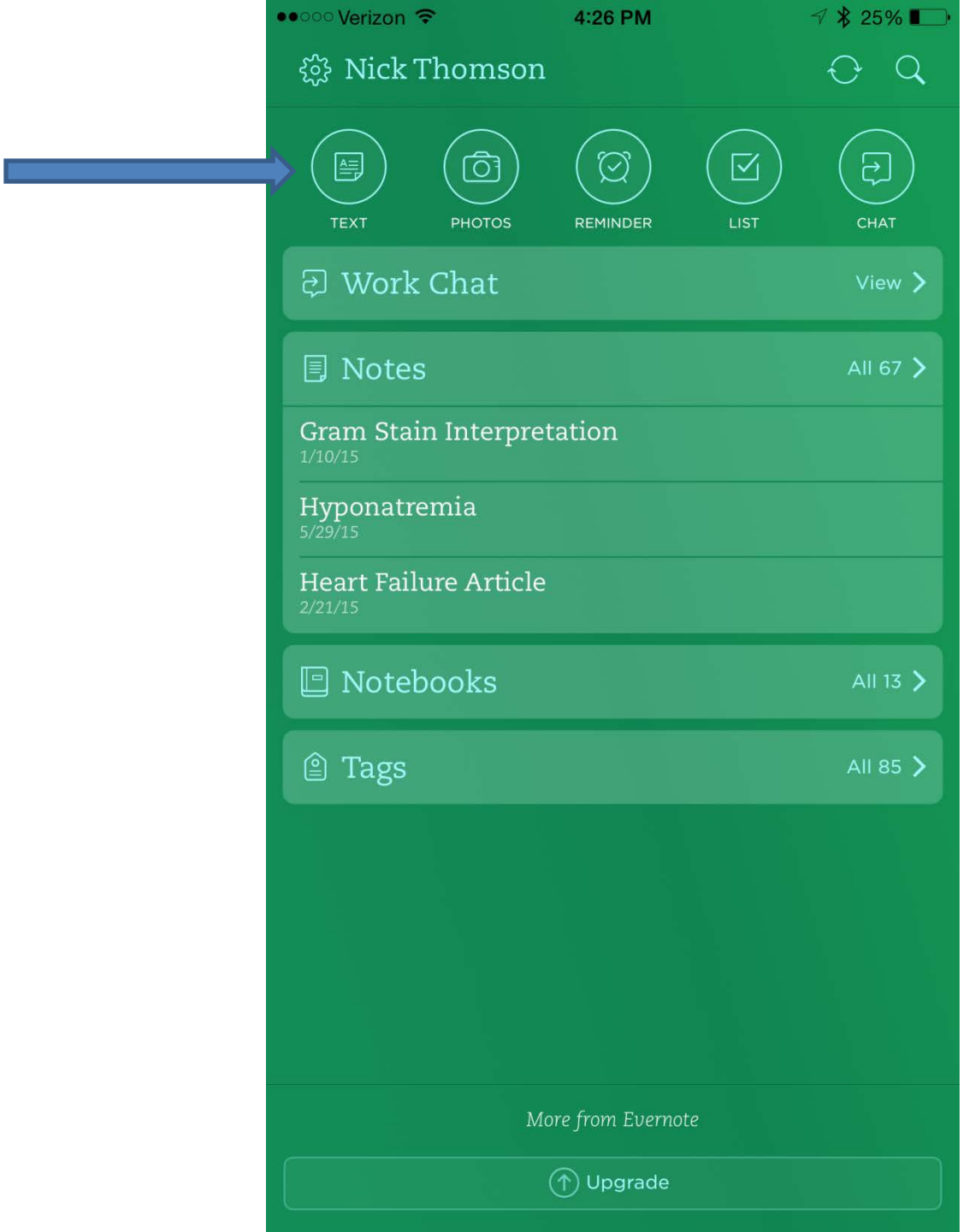
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Gram Stain Interpretation
1/10/15
Hyponatremia
5/29/15
Heart Failure Article
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Notebooks
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Tags
All 85
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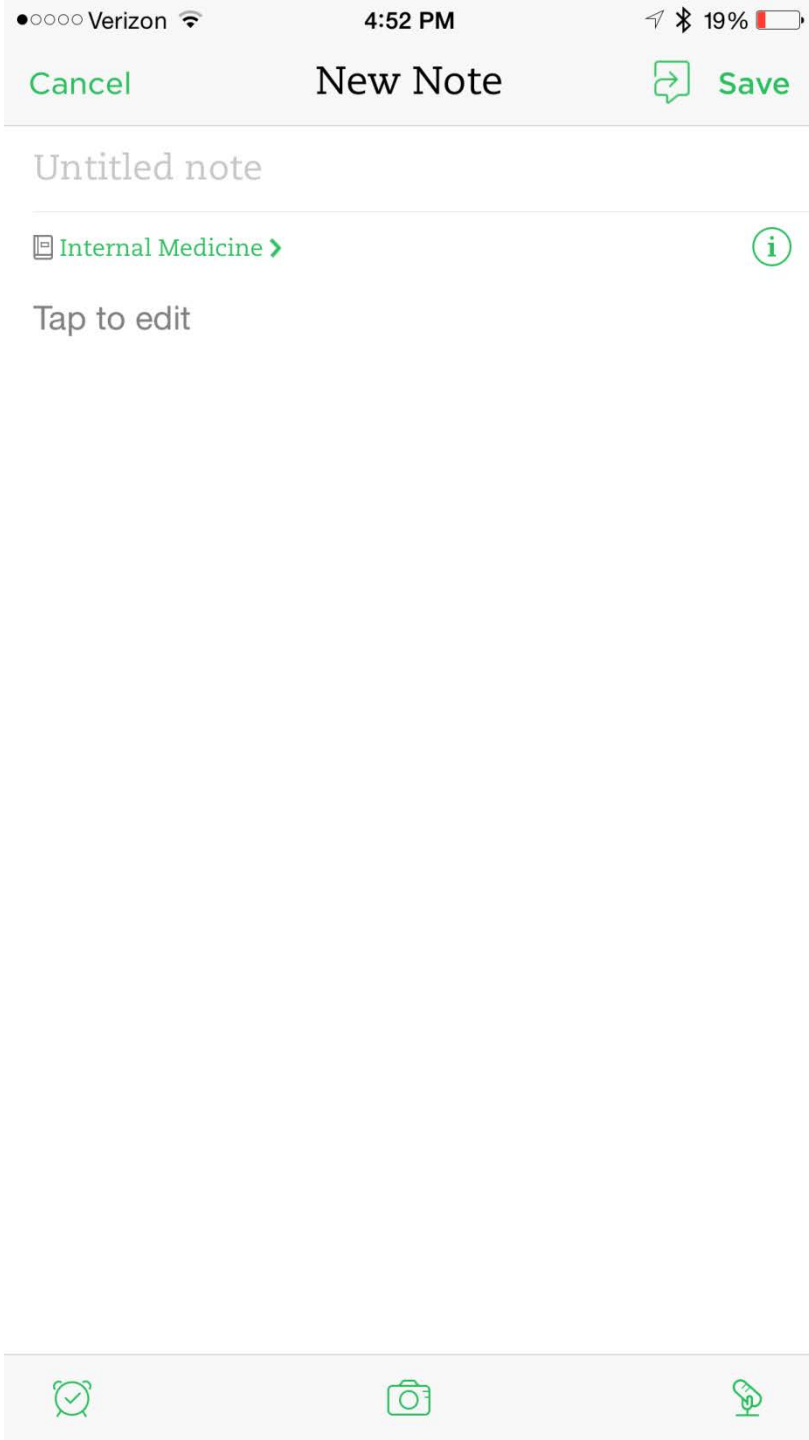
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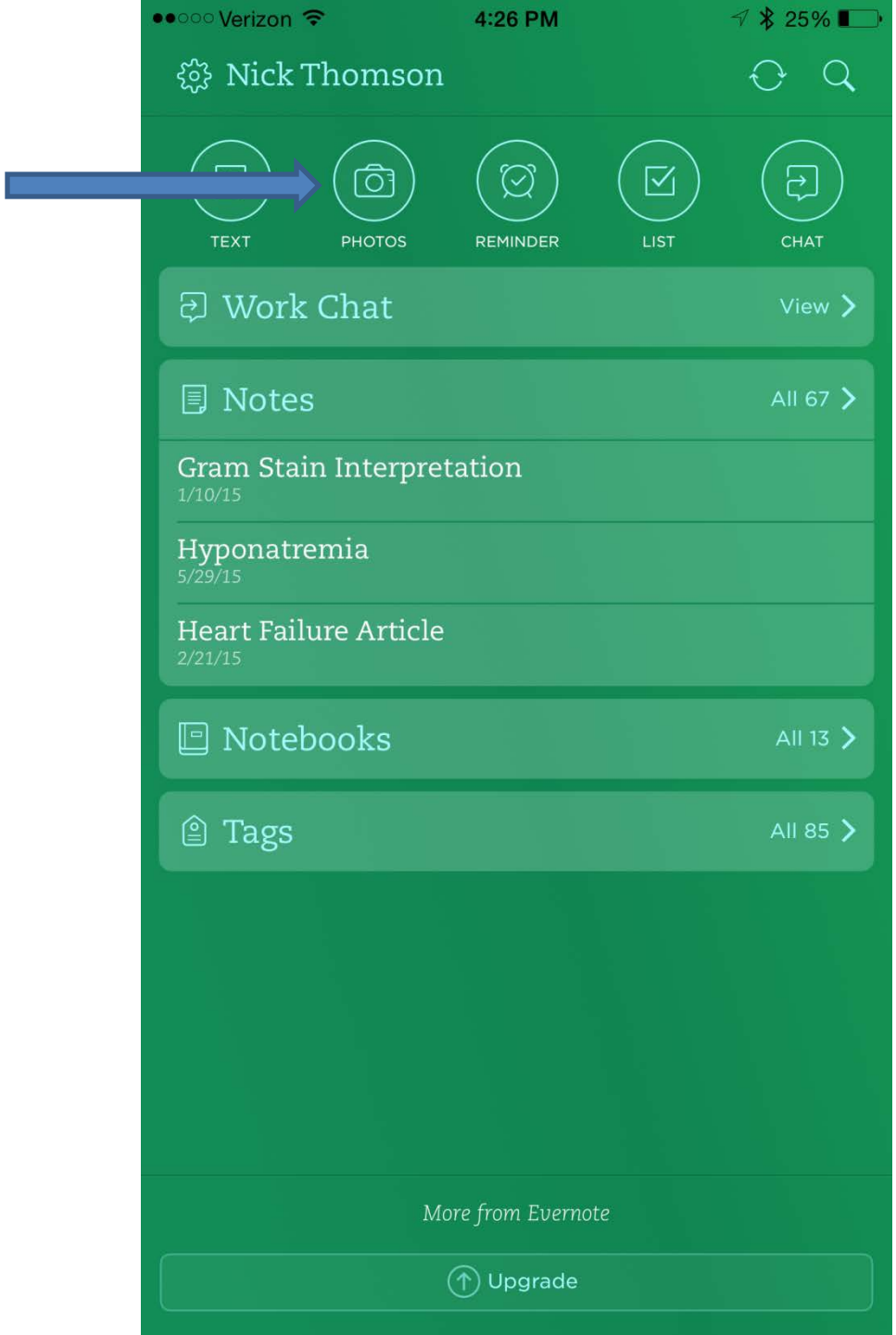
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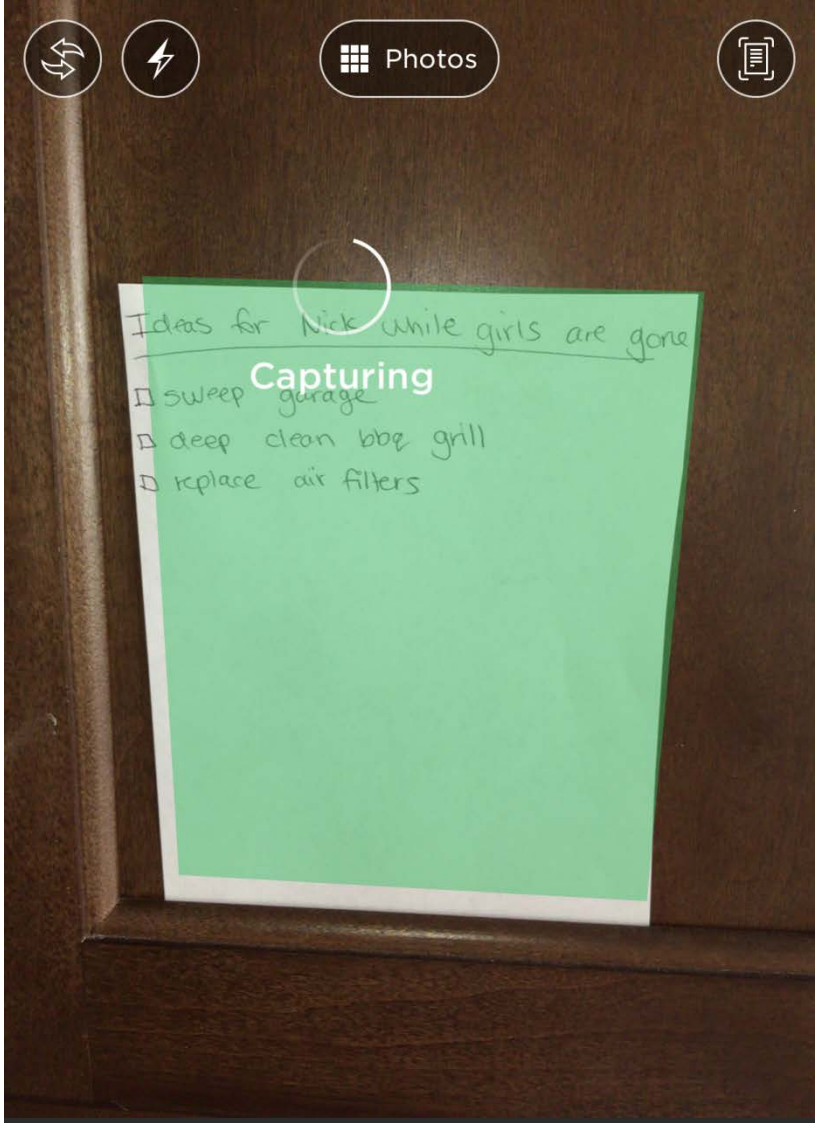

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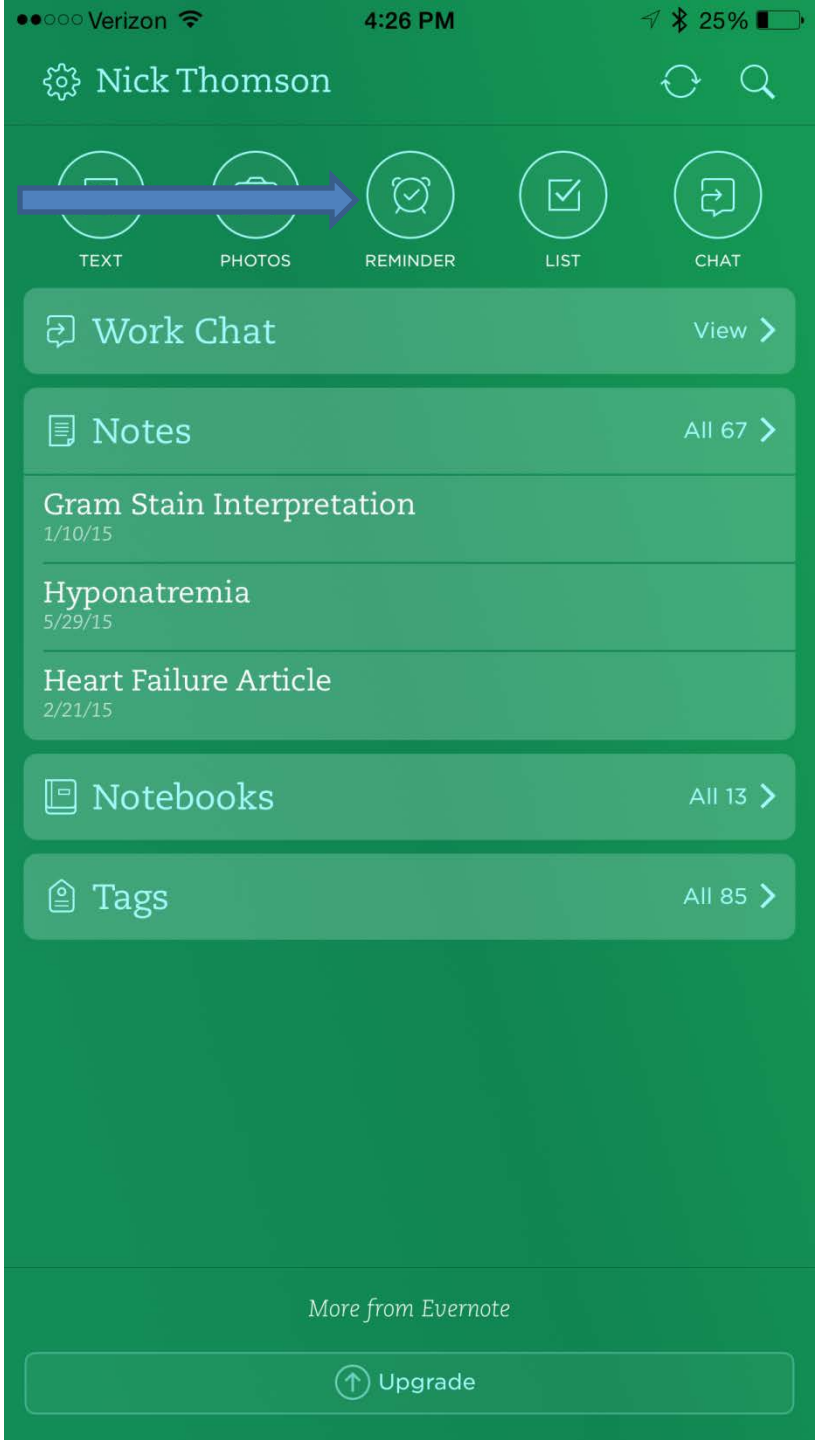
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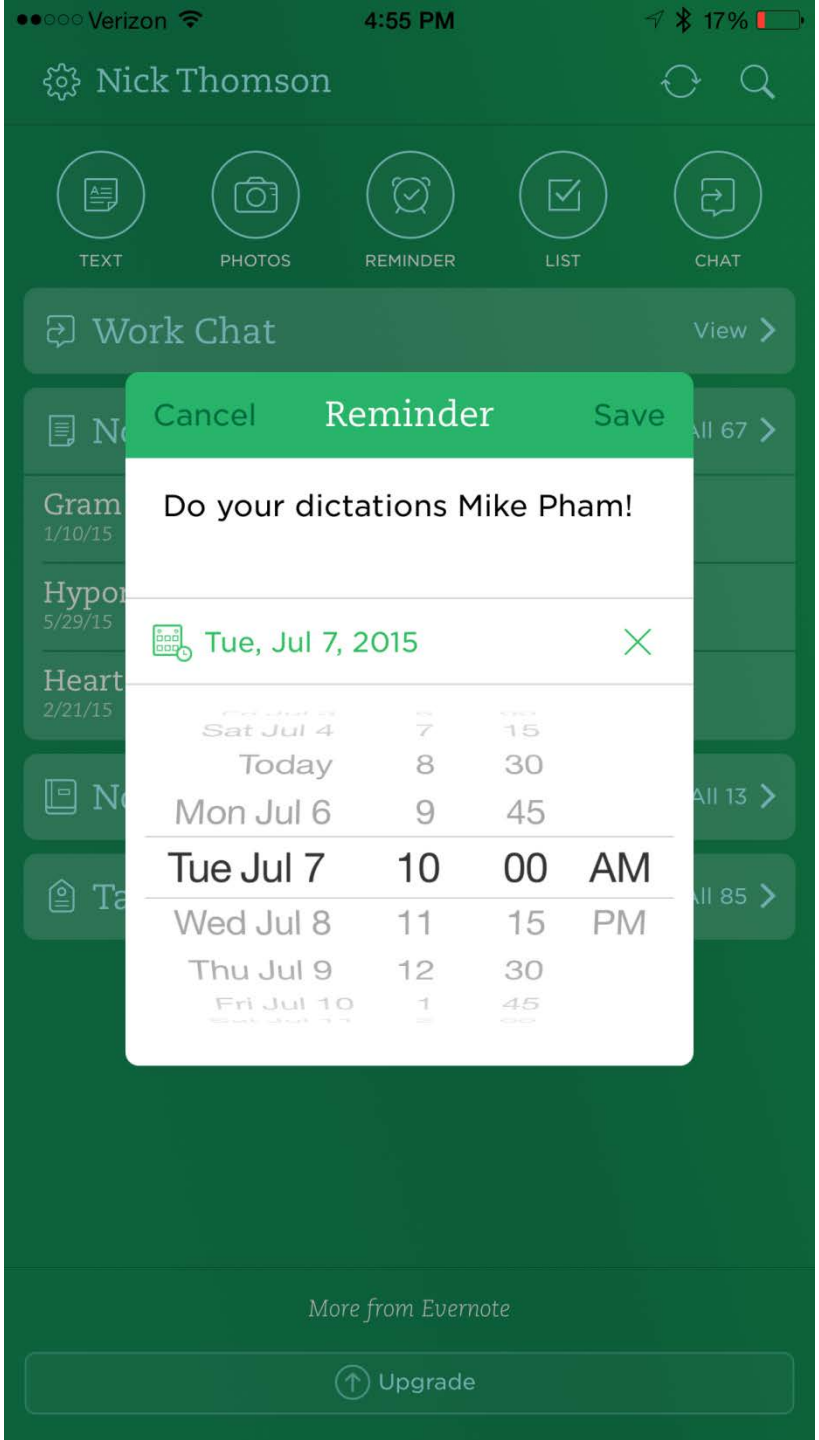
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Do your dictations Mike Pham!
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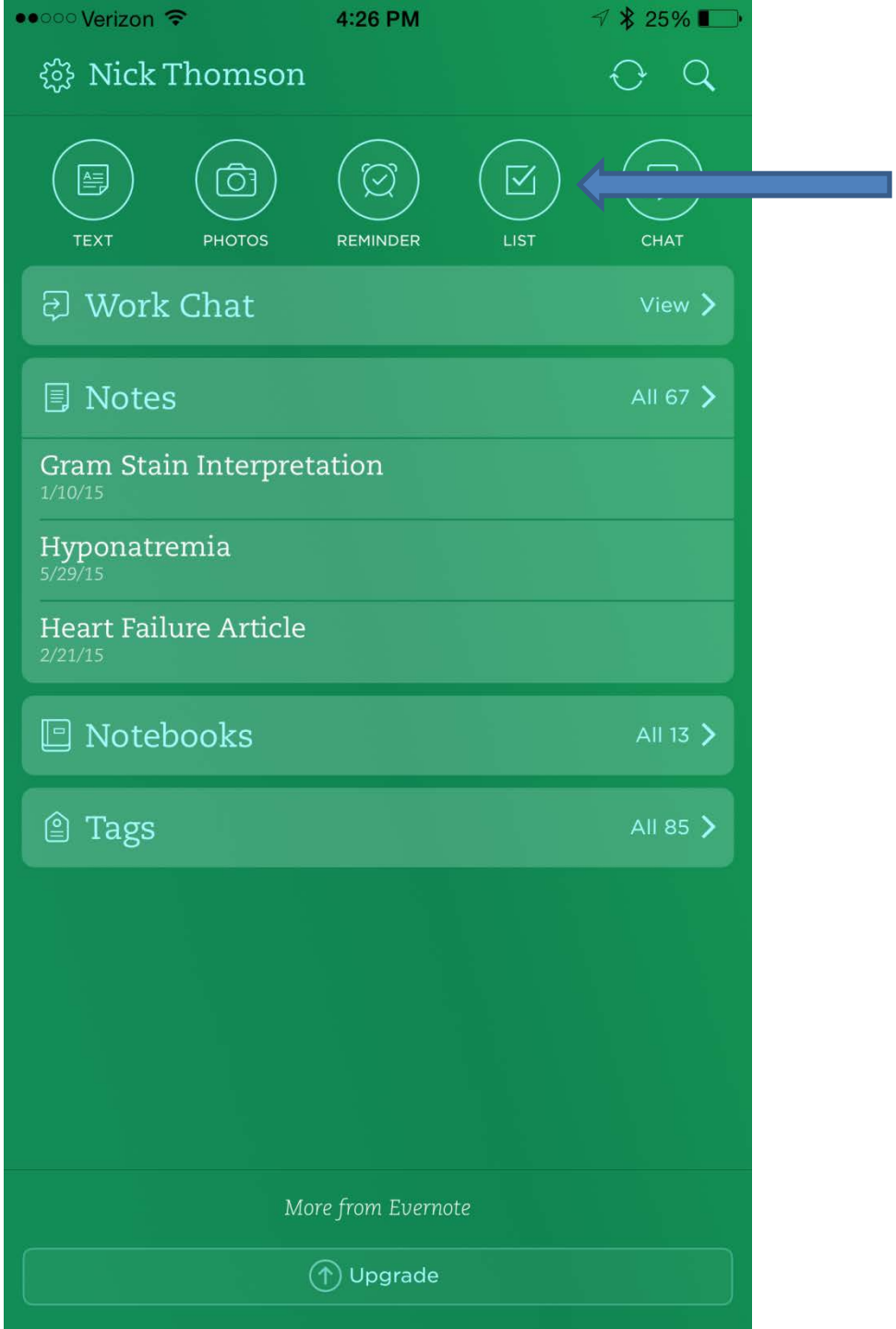
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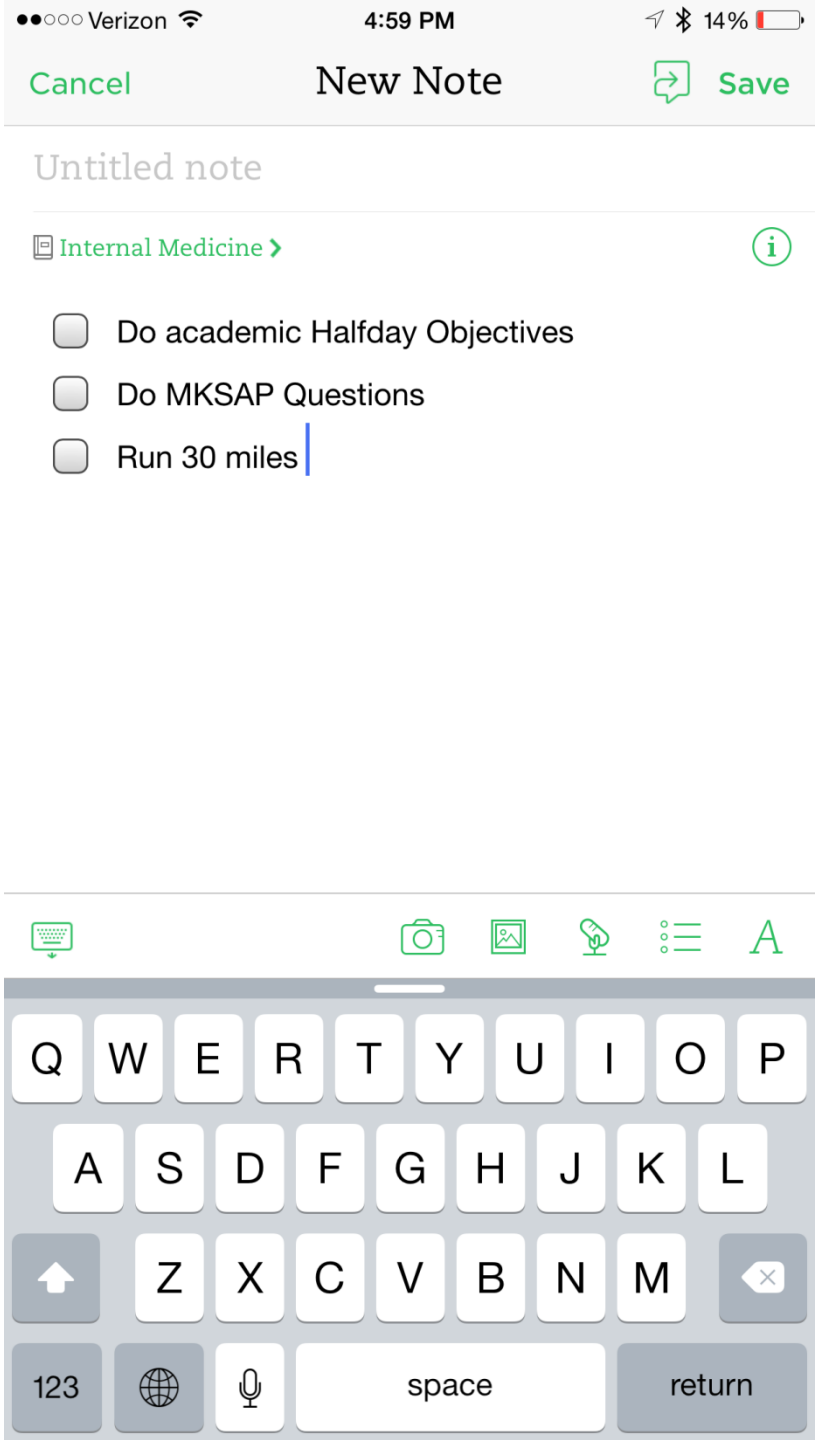
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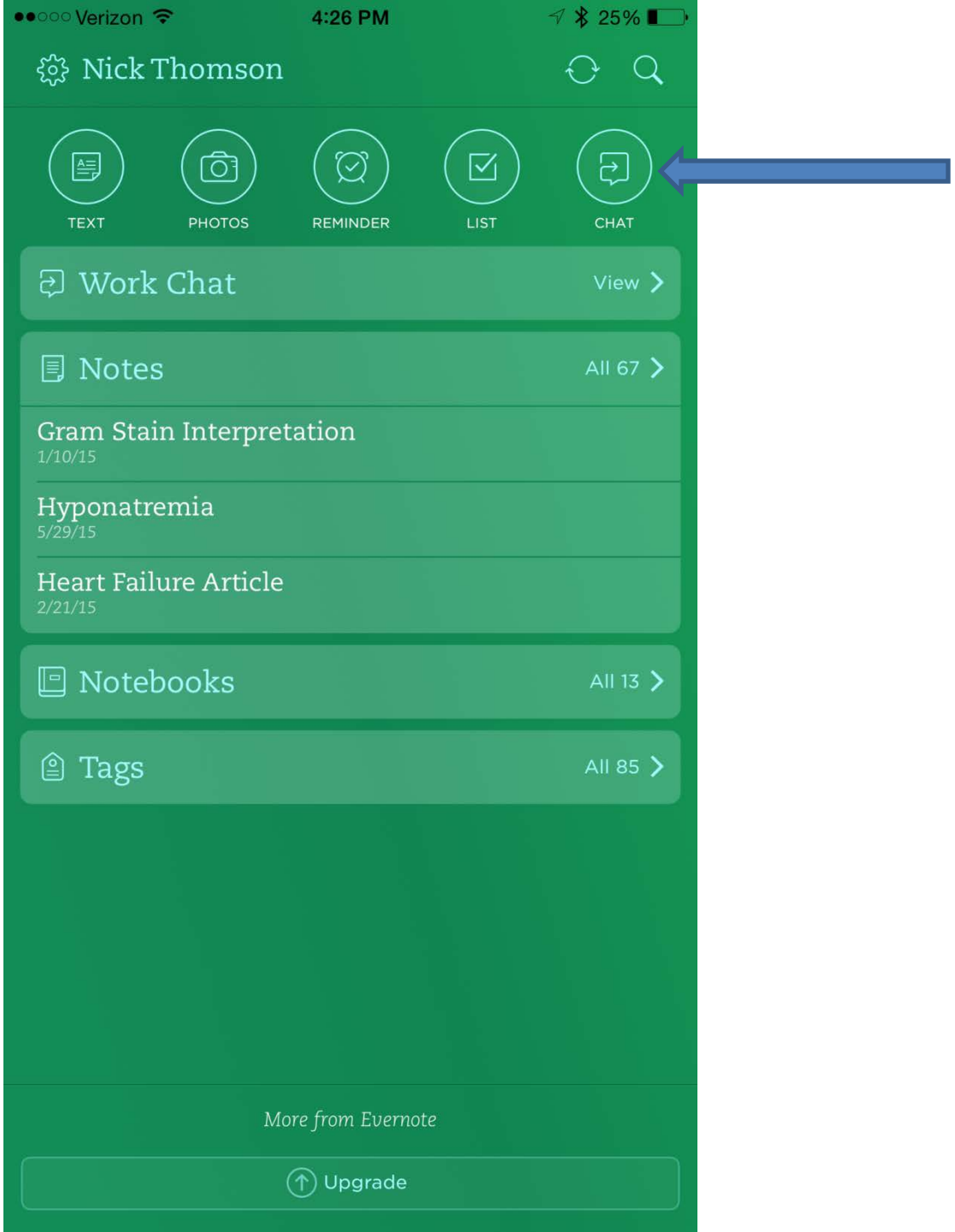
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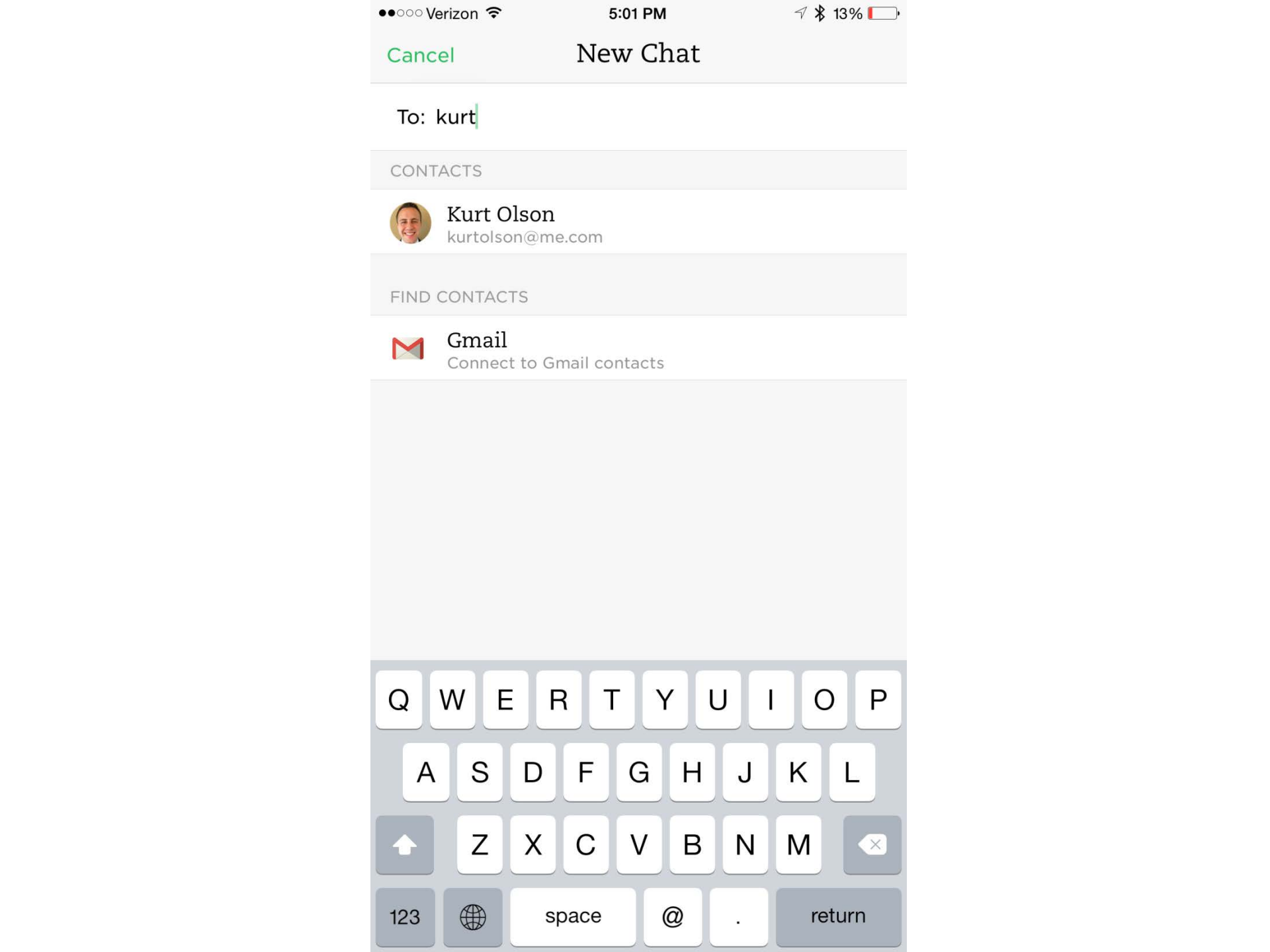
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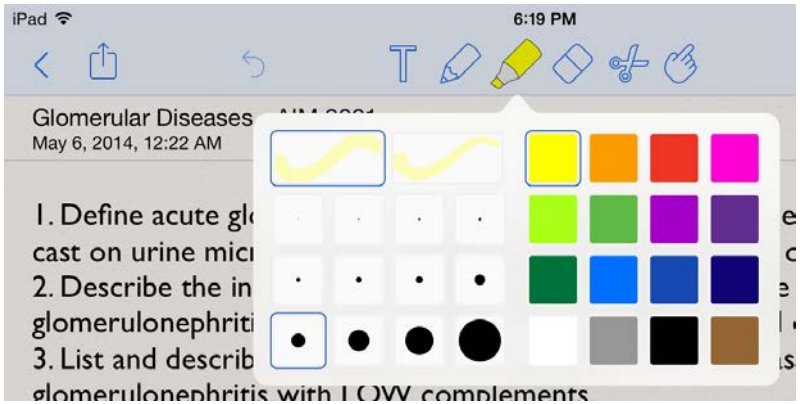


REVIEW ARTICLE

The Diagnosis of Glomerular Diseases

Acute Glomerulonephritis and the Nephrotic Syndrome

Michael P. Madaio, MD; John T. Harrington, MD

Rapid and efficient diagnosis of diseases presenting as acute glomerulonephritis and/or nephrotic syndrome is critical for early and appropriate therapy aimed at preservation of renal function. Although there may be overlap in clinical presentation, and some patients present with clinical features of both syndromes, this analysis serves as an initial framework to proceed with serologic testing and/or pathologic confirmation en route to final diagnosis. Efficient and timely diagnosis is essential in these situations because progression to end-stage renal disease may result if the underlying disease is not promptly treated.

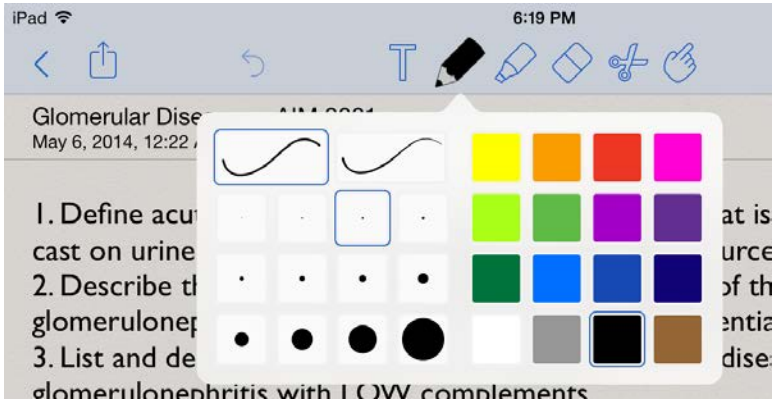
Arch Intern Med. 2001;161:25-34

Glomerular injury leads to impairment of the selective filtering properties of the kidney and reduction in the glomerular filtration rate (GFR).[1-3] Consequently, blood constituents normally excluded from the urinary space pass into the urine and are excreted. The nature and severity of the defect (ie, underlying disease and pathologic lesion) determine the quantity of red blood cells (RBC), white blood cells, and proteins lost in the urine and the extent of functional impairment.[4] These variables determine the clinical presentation. While the GFR is reduced initially in many patients, the severity, reversibility, and progression of disease are dependent on many factors, including the nature, location, and extent of the insult and the renal and systemic response to glomerular injury.[3,4] Prompt recognition of the cause of glomerular disease results in a more rational, safer, and effective therapeutic approach. Early diagnosis is especially important in patients with fulminant disease, where delay in treatment greatly reduces the likelihood of a beneficial response.[4,5]

In this review, we delineate our approach to the diagnosis of acute glomerular injury in adults, focusing on glomerulonephritis and nephrotic syndrome. Our intent is to provide a framework that will enable efficient and timely diagnosis. A few introductory points warrant particular emphasis. We do not discuss the evaluation of asymptomatic abnormalities discovered on routine urinalysis (ie, isolated hematuria and/or non–nephrotic-range proteinuria). The clinician should be aware that these manifestations may represent less severe forms of the full-blown entities. However, there are many nonglomerular causes of isolated hematuria and proteinuria that must also be considered in these situations, and the reader is referred to recent reviews of these entities.[2,6-12]

Although our approach distinguishes between nephritic and nephrotic states (the two classic clinical presentations of acute glomerular injury), many of the underlying diseases can produce nephritis or nephrotic syndrome. Furthermore, this distinction is not always easily made in individual patients. For example, some patients present with nephrotic-range proteinuria and active urine sediments, whereas others present with nephrotic-range proteinuria and acute renal failure. In some instances the clinical presentation represents the initial manifestation of an acute disease, whereas in others the physician initially detects a more chronic

From the Renal Electrolyte and Hypertension Division, Department of Medicine, University of Pennsylvania, Philadelphia (Dr Madaio), and the Nephrology Division, Department of Medicine, New England Medical Center and Tufts University School of Medicine, Boston, Mass (Dr Harrington).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2. Describe th
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3. List and de
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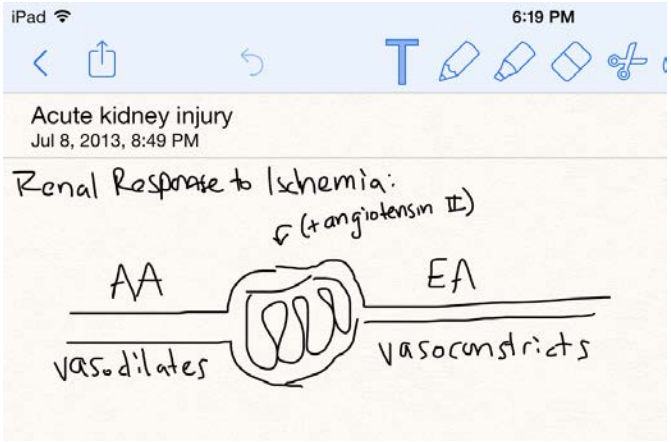
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vasodilates
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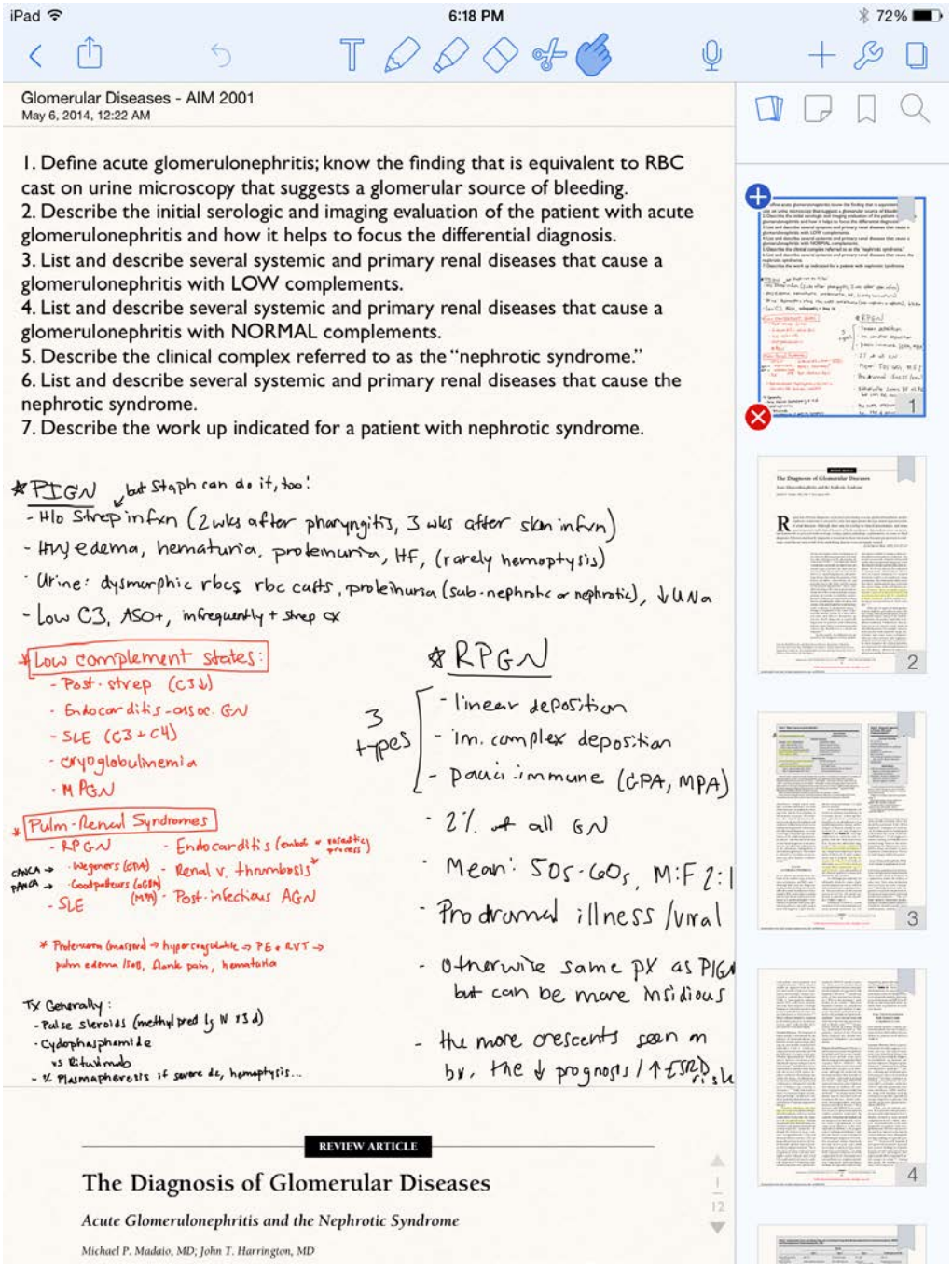
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Glomerular Diseases - AIM 2001
May 6, 2014, 12:22 AM
1. Define acute glomerulonephritis; know the finding that is equivalent to RBC cast on urine microscopy that suggests a glomerular source of bleeding.
2. Describe the initial serologic and imaging evaluation of the patient with acute glomerulonephritis and how it helps to focus the differential diagnosis.
3. List and describe several systemic and primary renal diseases that cause a glomerulonephritis with LOW complements.
4. List and describe several systemic and primary renal diseases that cause a glomerulonephritis with NORMAL complements.
5. Describe the clinical complex referred to as the "nephrotic syndrome."
6. List and describe several systemic and primary renal diseases that cause the nephrotic syndrome.
7. Describe the work up indicated for a patient with nephrotic syndrome.
PIGN
but Staph can do it, too!
- Hx Strep infxn (2 wks after pharyngitis, 3 wks after skin infxn)
- HTN, edema, hematuria, proteinuria, HF, (rarely hemoptysis)
- Urine: dysmorphic rbcs, rbc casts, proteinuria (sub-nephrotic or nephrotic), ↓UNa
- Low C3, ASO+, infrequently + strep cx
Low complement states:
- Post-strep (C3↓)
- Endocarditis-assoc. GN
- SLE (C3+C4)
- cryoglobulinemia
- MPGN
RPGN
3 types
- linear deposition
- im. complex deposition
- pauci-immune (GPA, MPA)
- 2% of all GN
- Mean: 50s-60s, M:F 2:1
- Prodromal illness/viral
- Otherwise same px as PIGN but can be more insidious
- the more crescents seen on bx, the ↓ prognosis / ↑ESRD risk
Pulm-Renal Syndromes
- RPGN
- Endocarditis (subac. + acute)
- Renal v. thrombosis
- Post-infectious AGN
- SLE
ANCA → Wegeners (GPA)
APCA → Goodpastures (GBM) (MPA)
* Proteasome (marrow) → hypercoagulable → PE + RVT → pulm edema (cough, flank pain, hematuria
Tx Generally:
- Pulse steroids (methylpred 1g IV x 3d)
- Cyclophosphamide
vs Rituximab
- ± Plasmapheresis if severe dz, hemoptysis...
REVIEW ARTICLE
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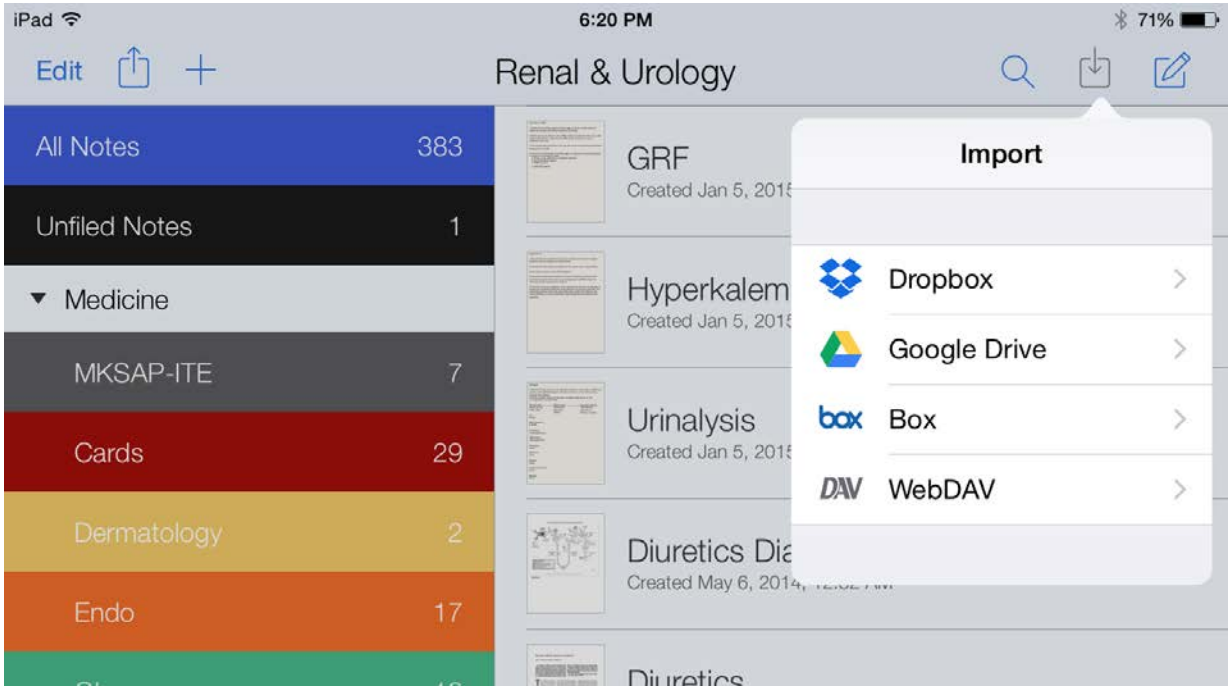
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