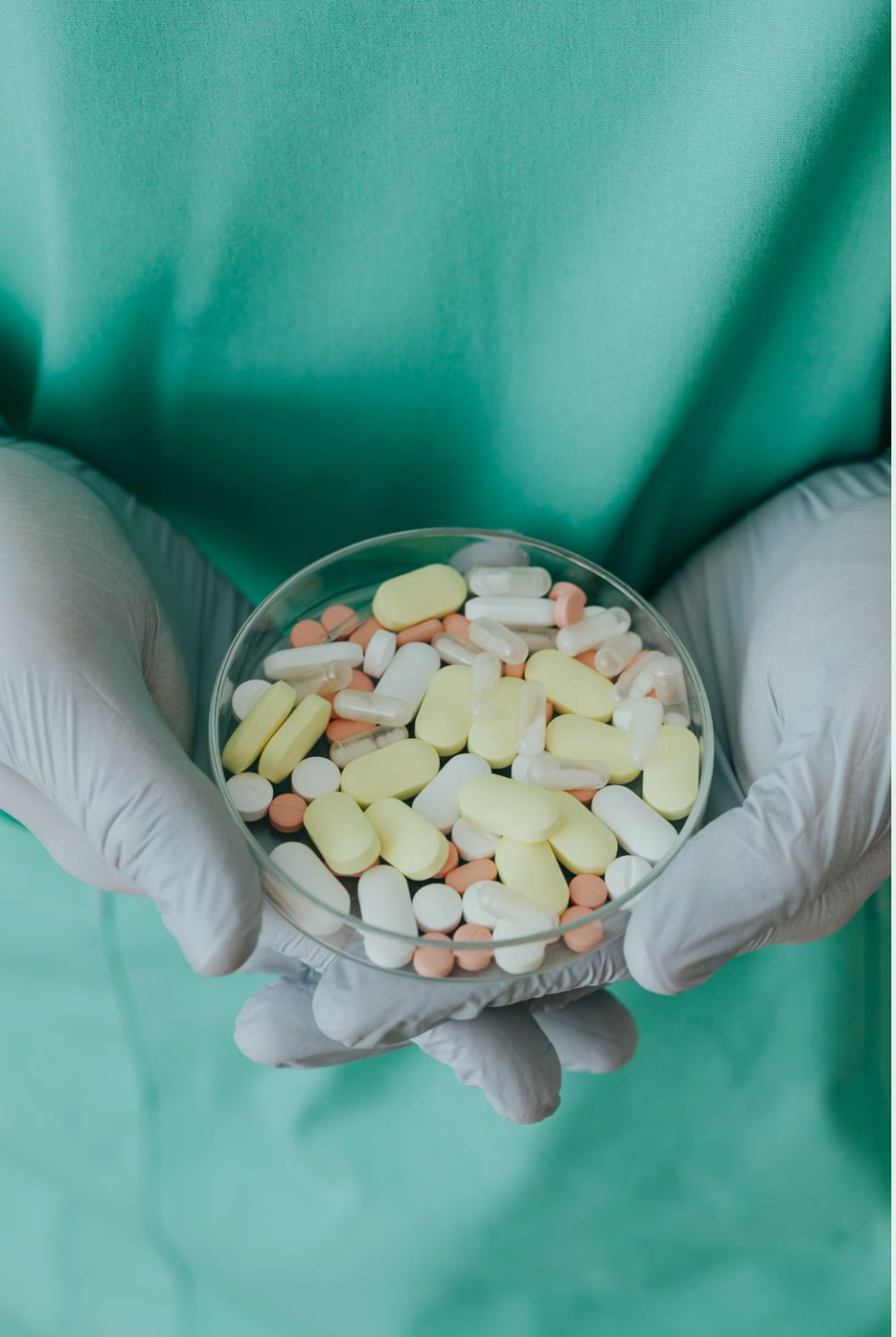

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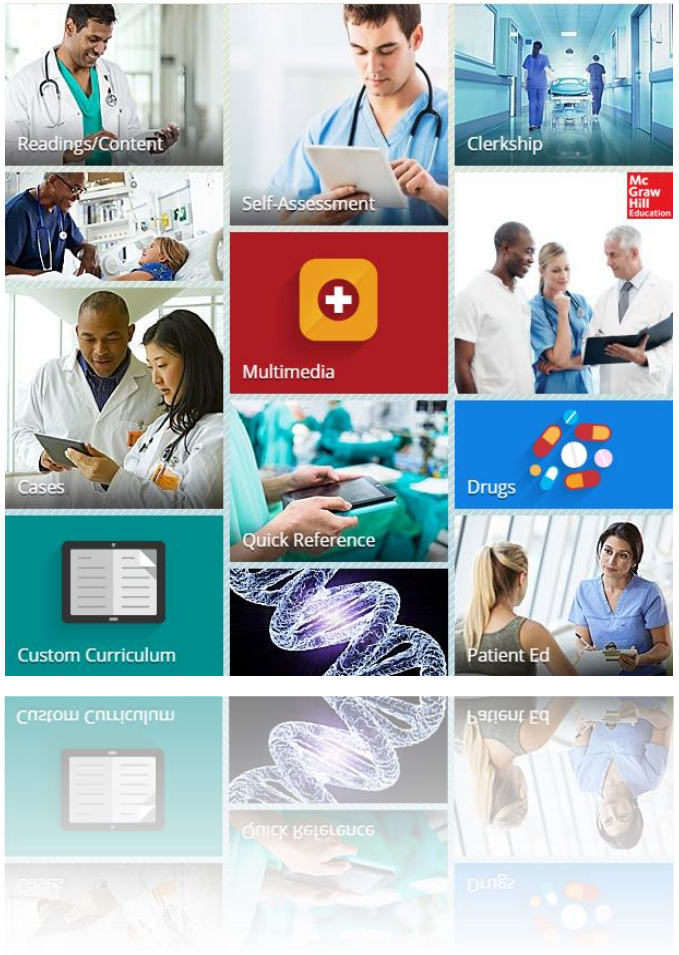

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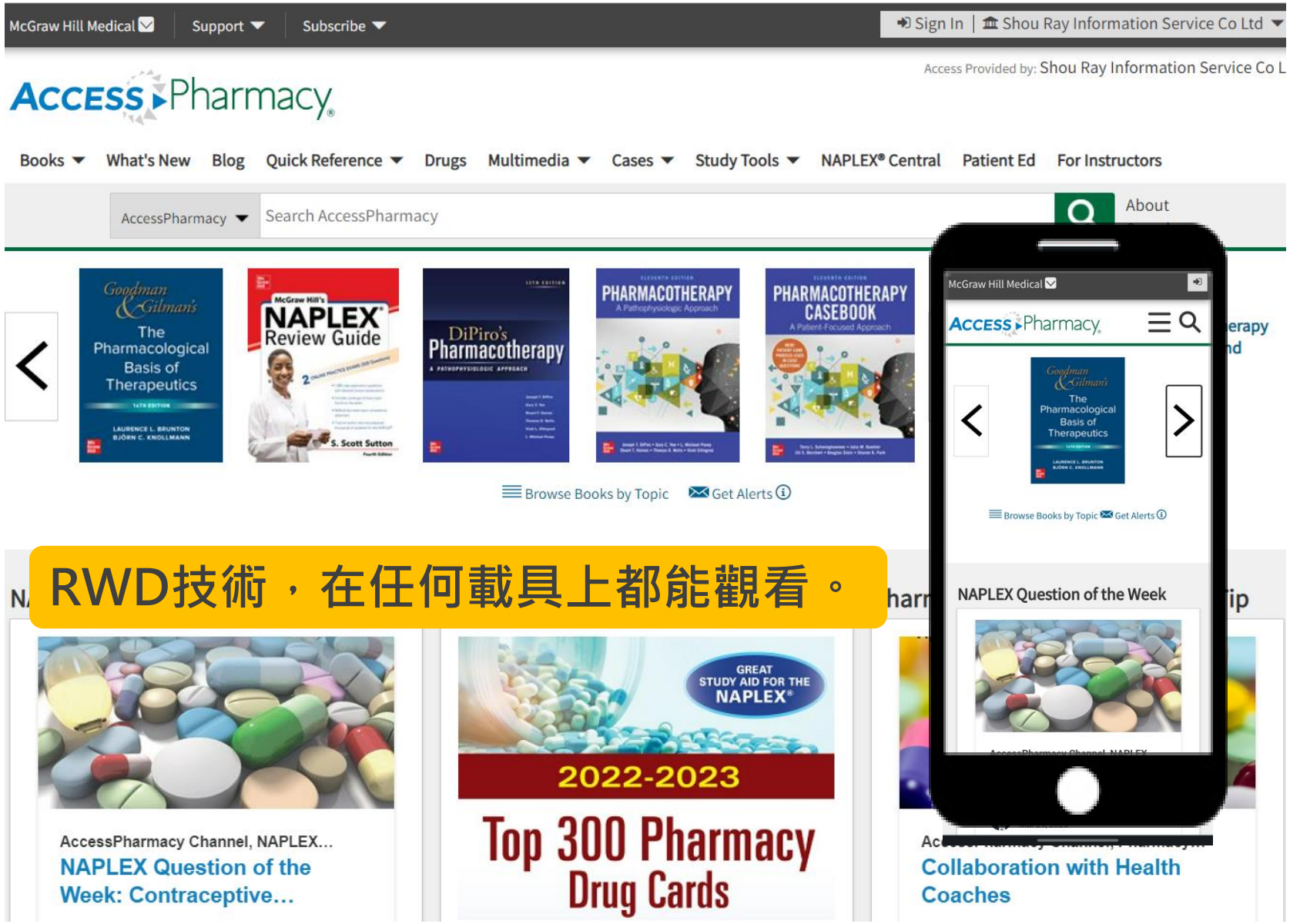

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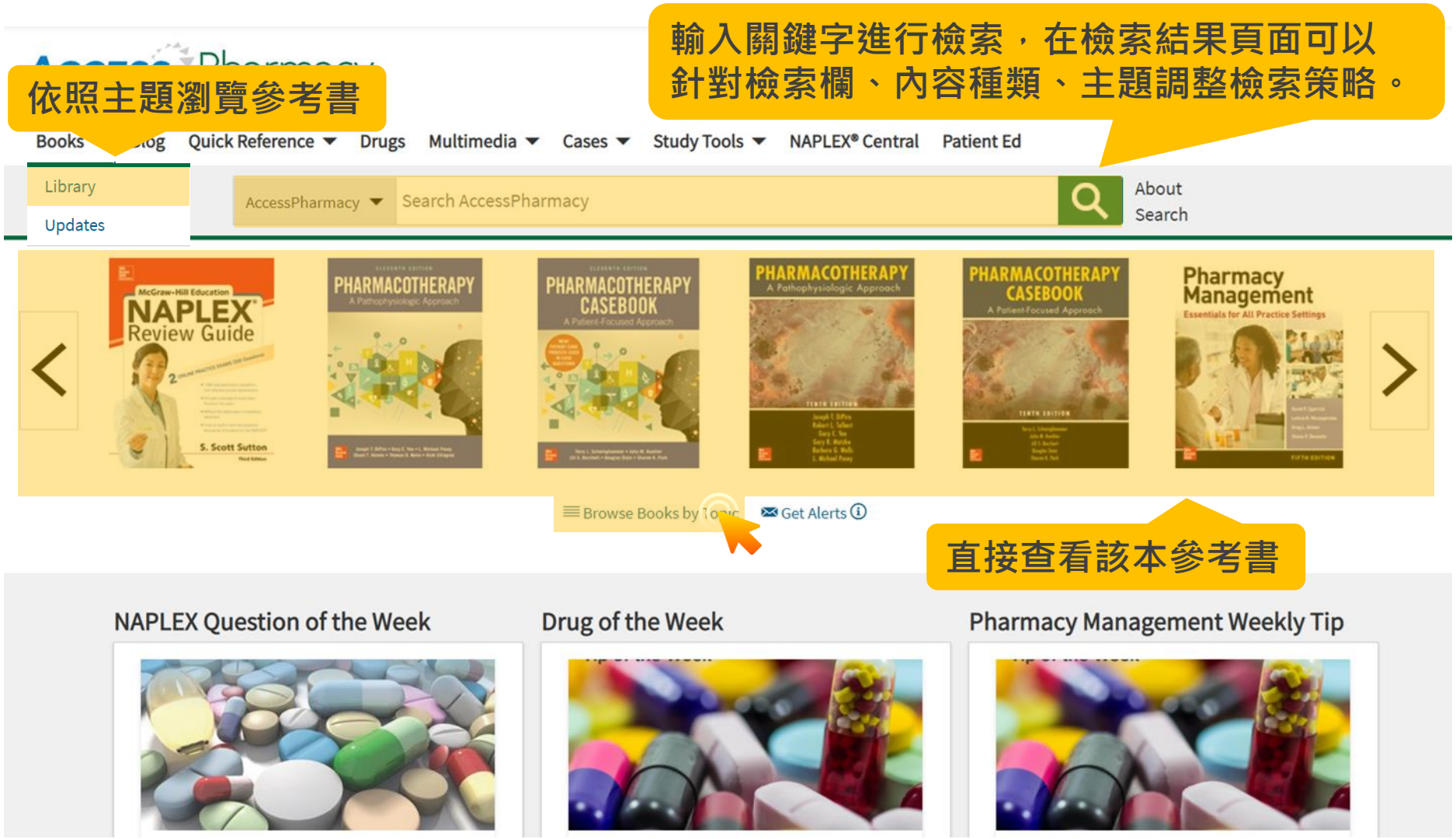
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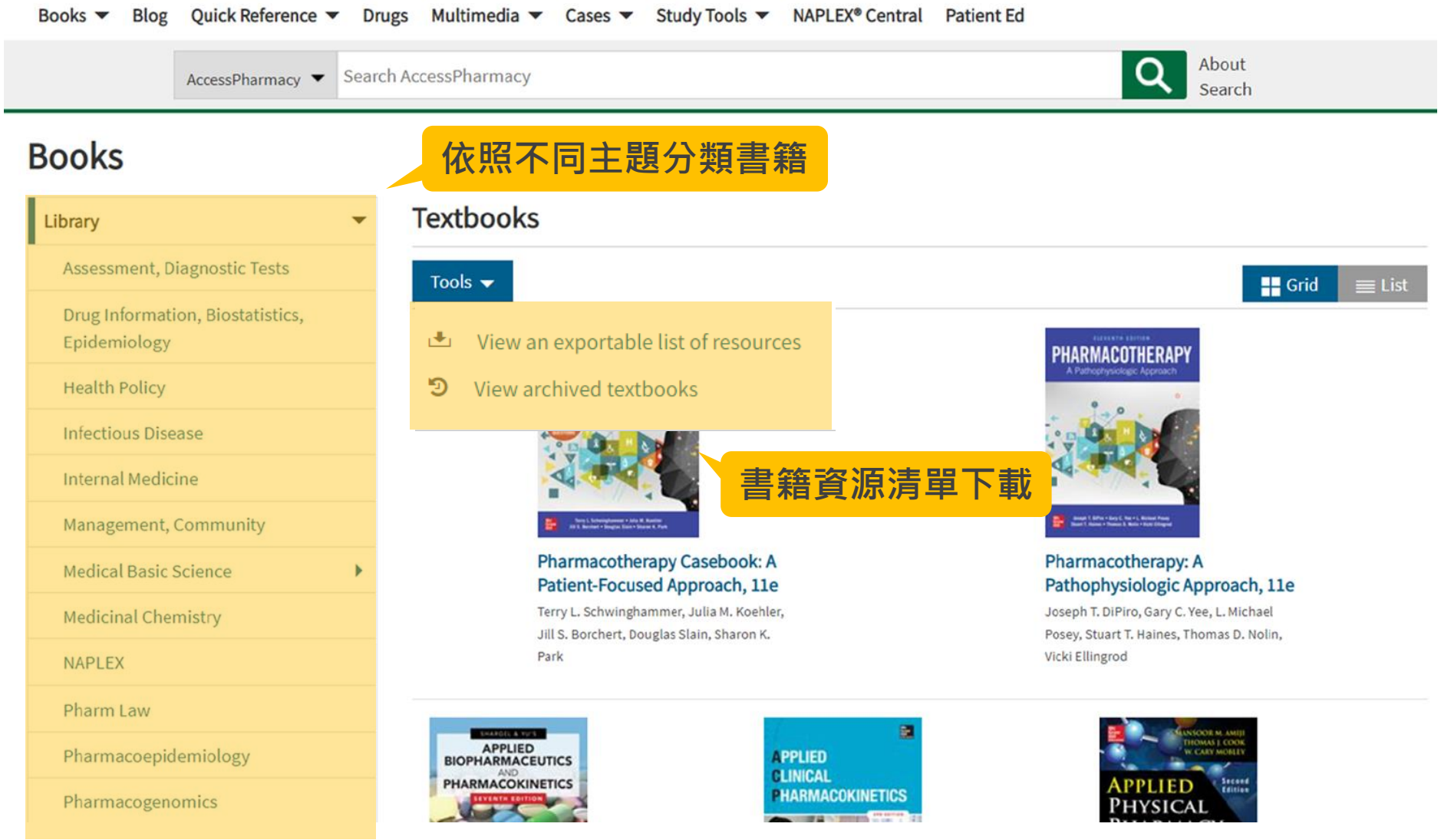

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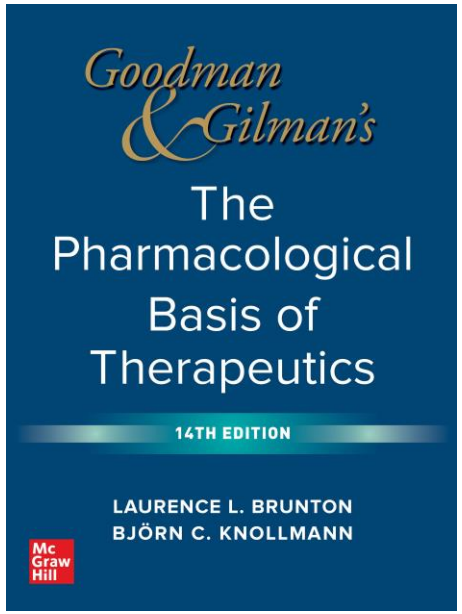

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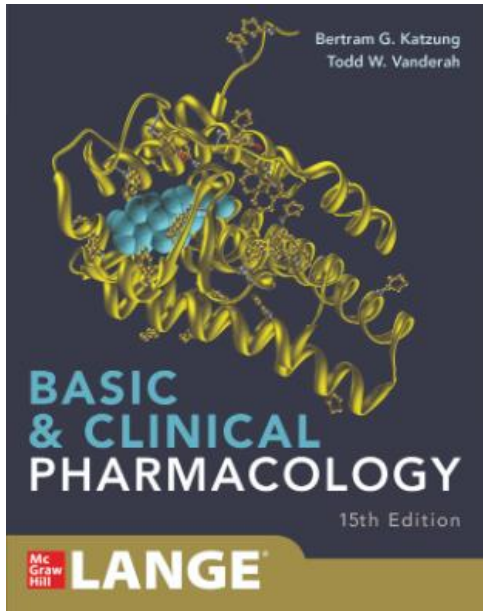

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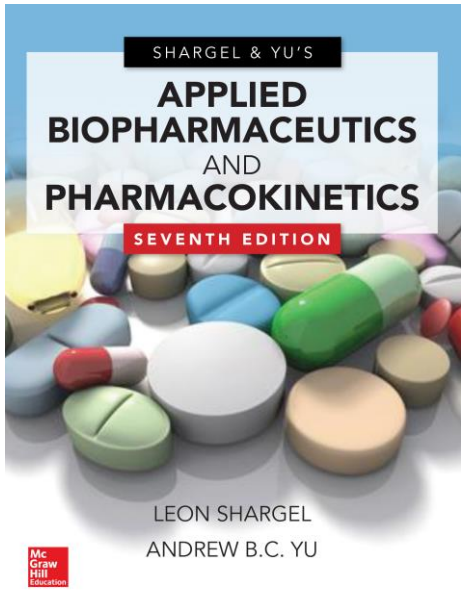

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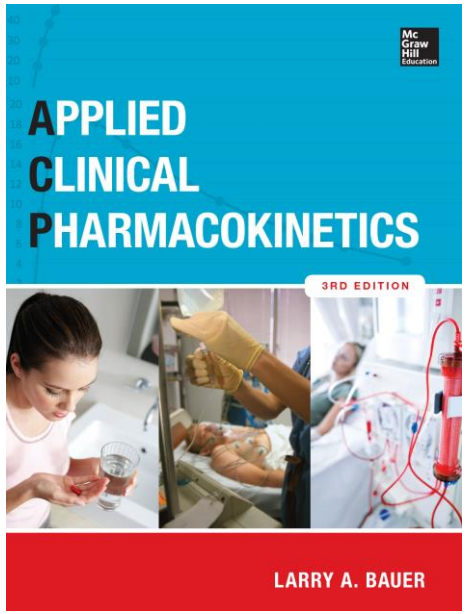

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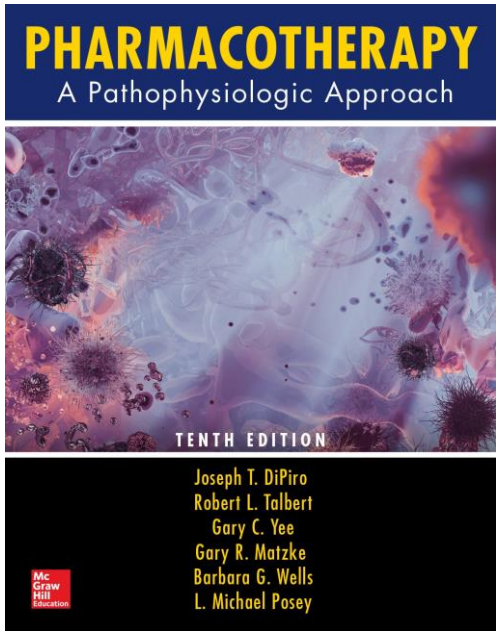

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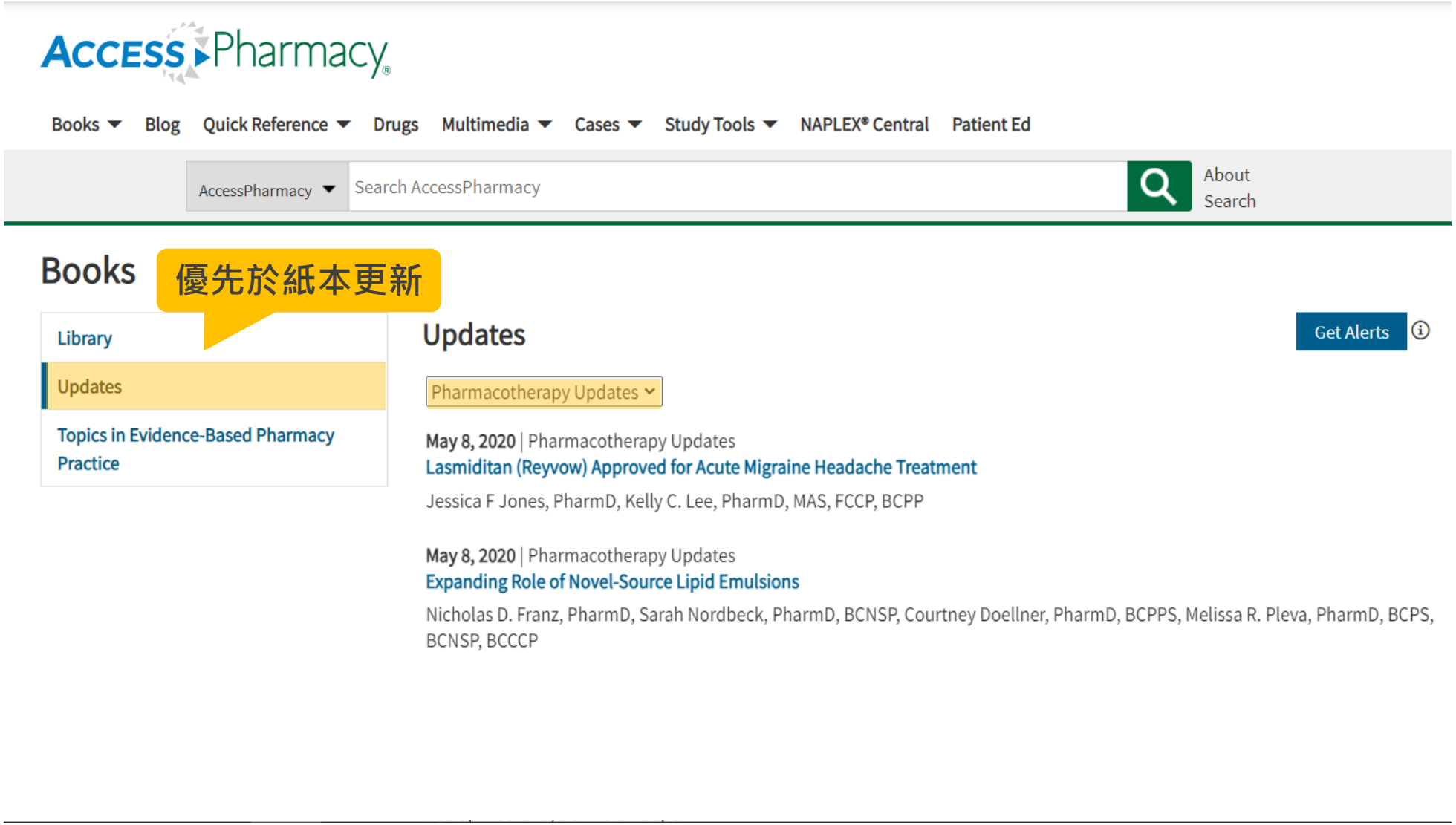

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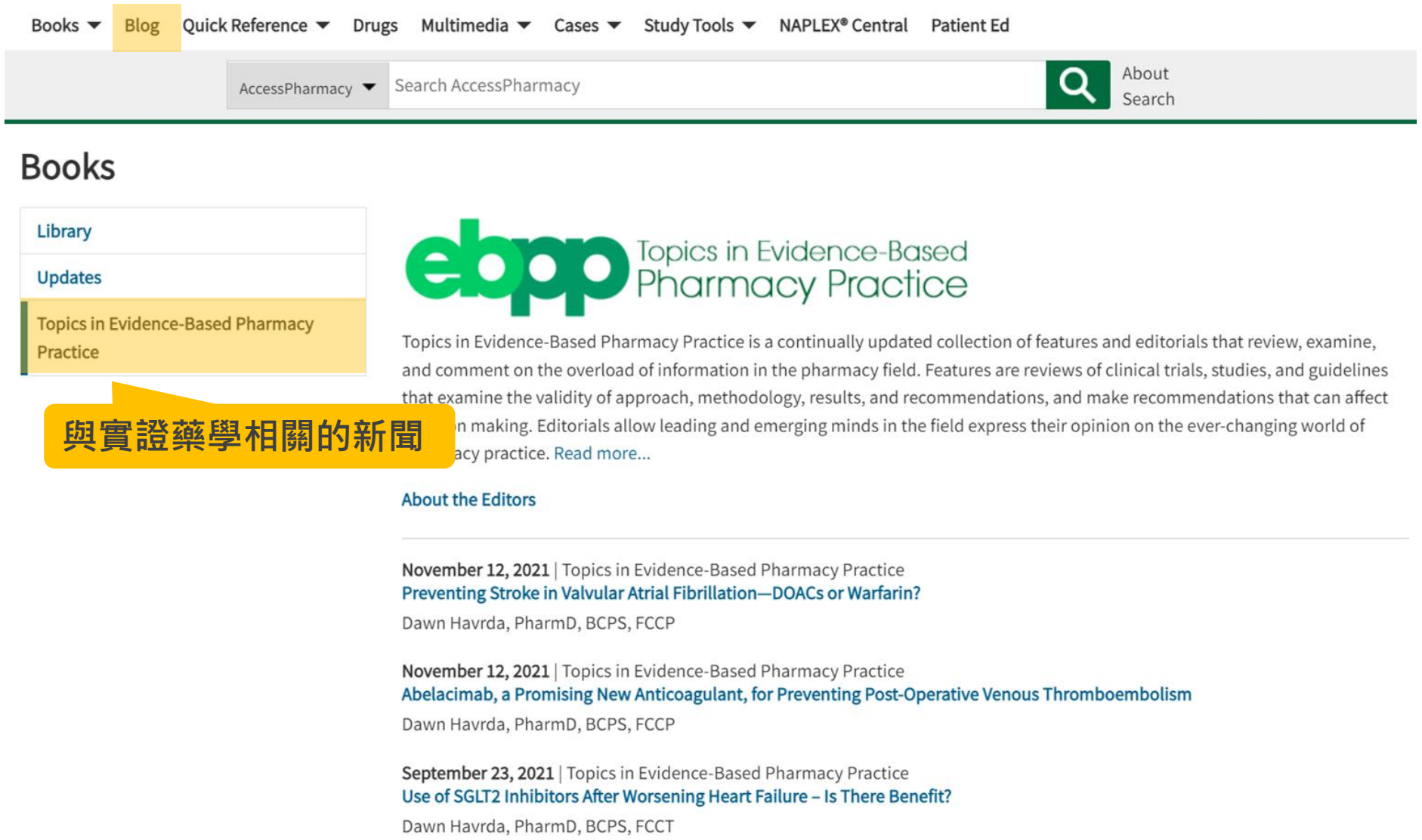

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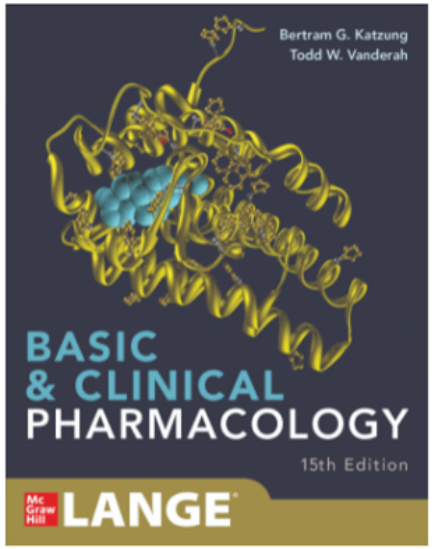

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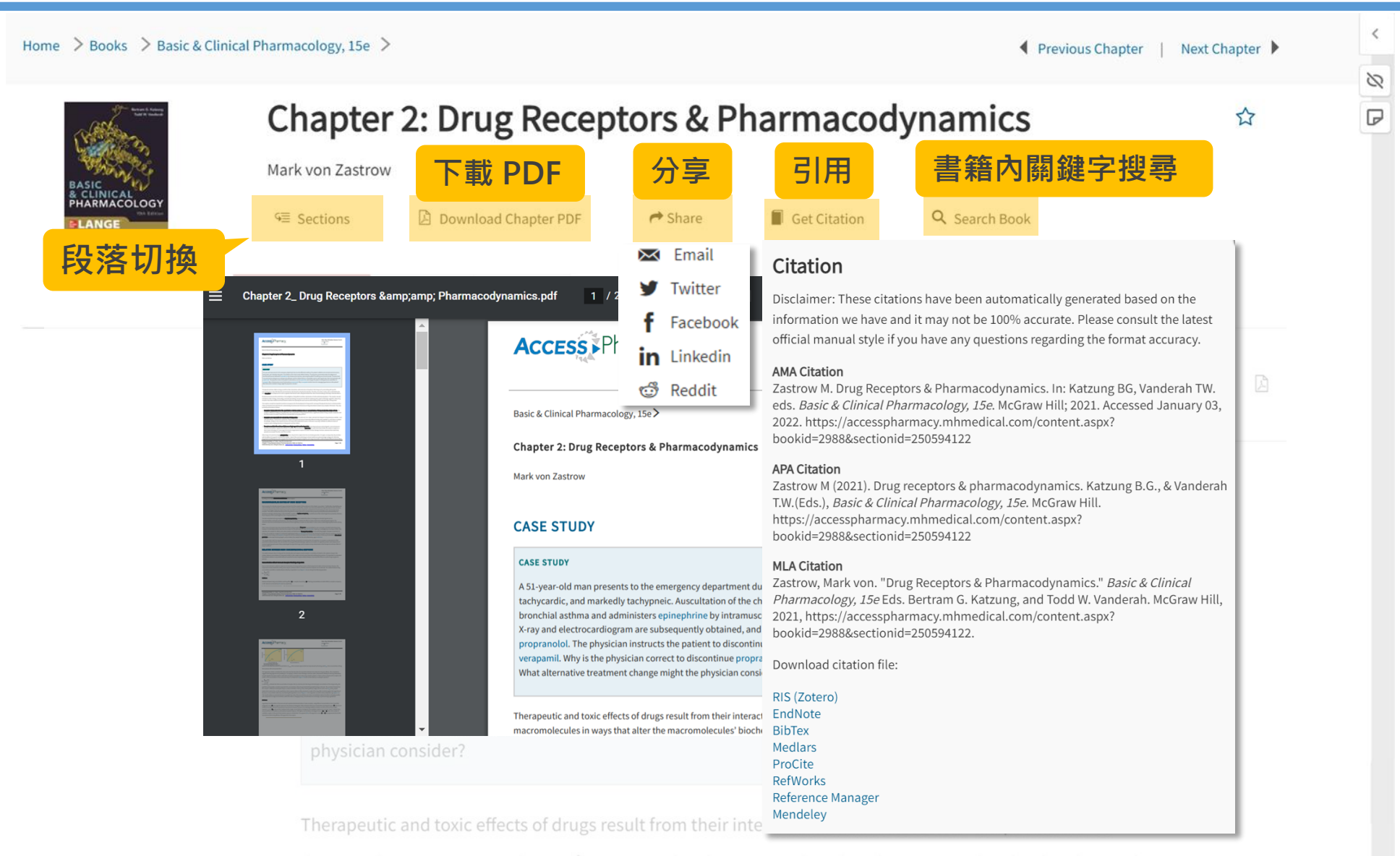
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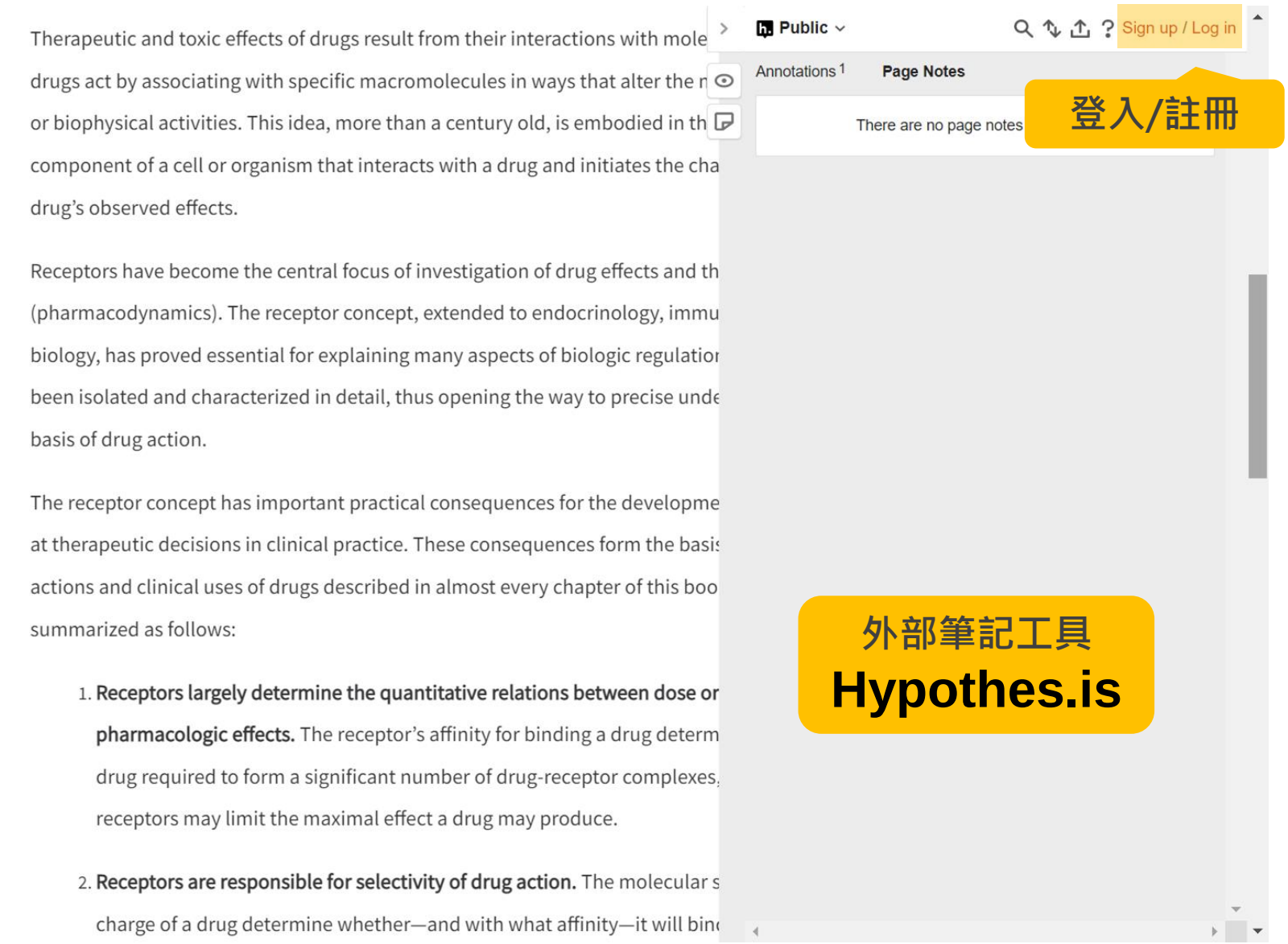

Therapeutic and toxic effects of drugs result from their interactions with mole
drugs act by associating with specific macromolecules in ways that alter the n
or biophysical activities. This idea, more than a century old, is embodied in th
component of a cell or organism that interacts with a drug and initiates the cha
drug's observed effects.

Receptors have become the central focus of investigation of drug effects and th
(pharmacodynamics). The receptor concept, extended to endocrinology, immu
biology, has proved essential for explaining many aspects of biologic regulatio
been isolated and characterized in detail, thus opening the way to precise unde
basis of drug action.

The receptor concept has important practical consequences for the developme
at therapeutic decisions in clinical practice. These consequences form the basis
actions and clinical uses of drugs described in almost every chapter of this boo
summarized as follows:

1. **Receptors largely determine the quantitative relations between dose or pharmacologic effects.** The receptor's affinity for binding a drug determ drug required to form a significant number of drug-receptor complexes, receptors may limit the maximal effect a drug may produce.

2. **Receptors are responsible for selectivity of drug action.** The molecular s charge of a drug determine whether—and with what affinity—it will bin

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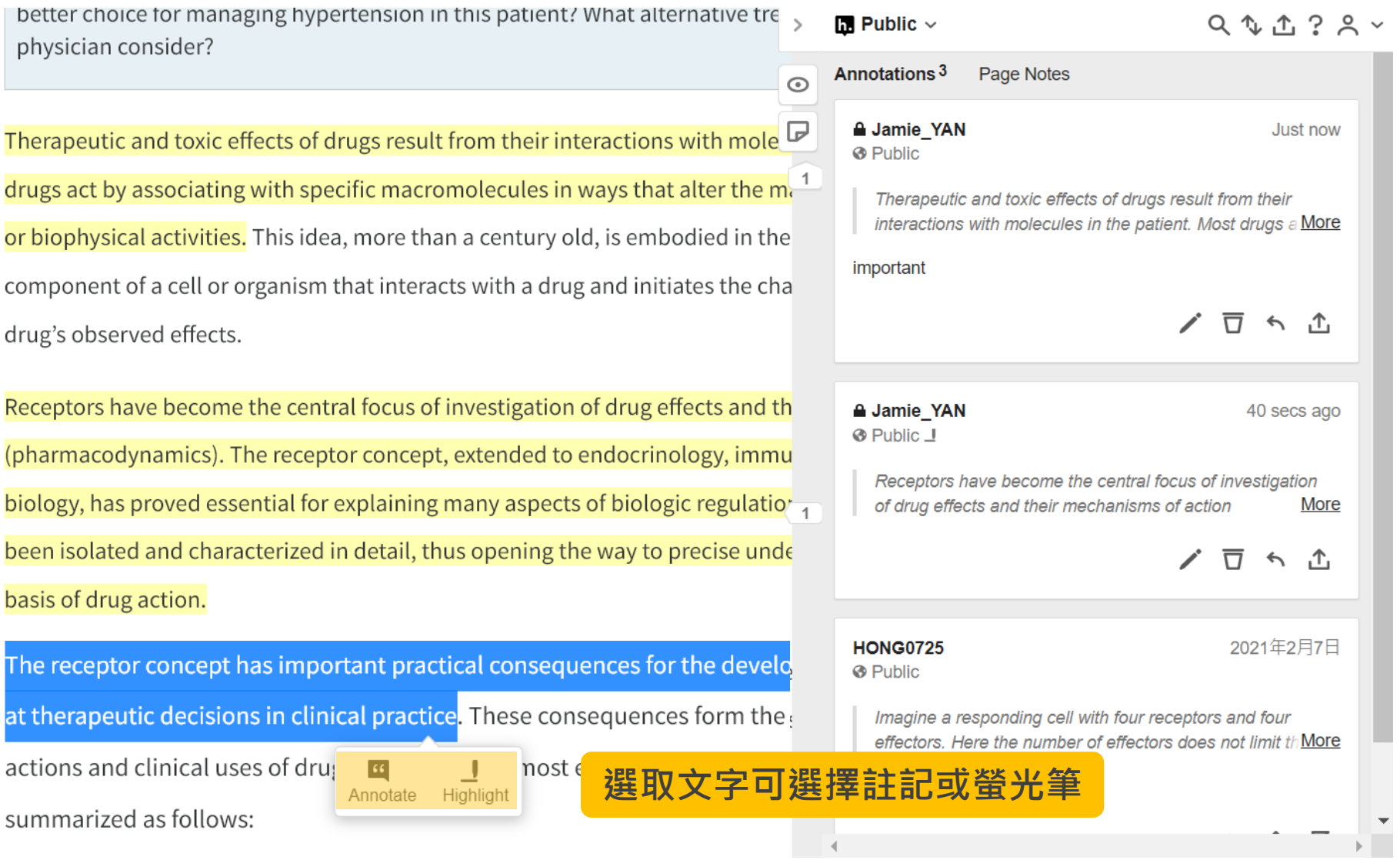

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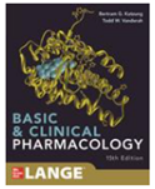
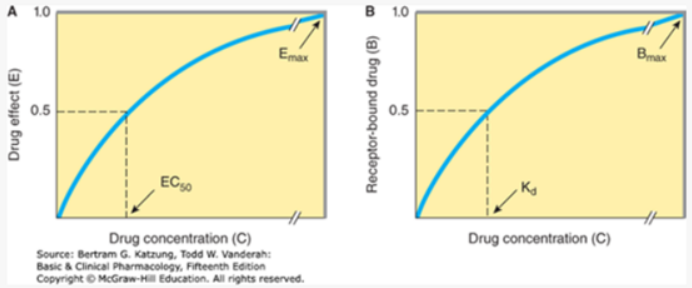


Chapter 2: Drug Receptors & Pharmacodynamics

Mark von Zastrow

FIGURE 2–1

Relations between drug concentration and drug effect (**A**) or receptor-bound drug (**B**). The drug concentrations at which effect or receptor occupancy is half-maximal are denoted by EC_{50} and K_d, respectively.



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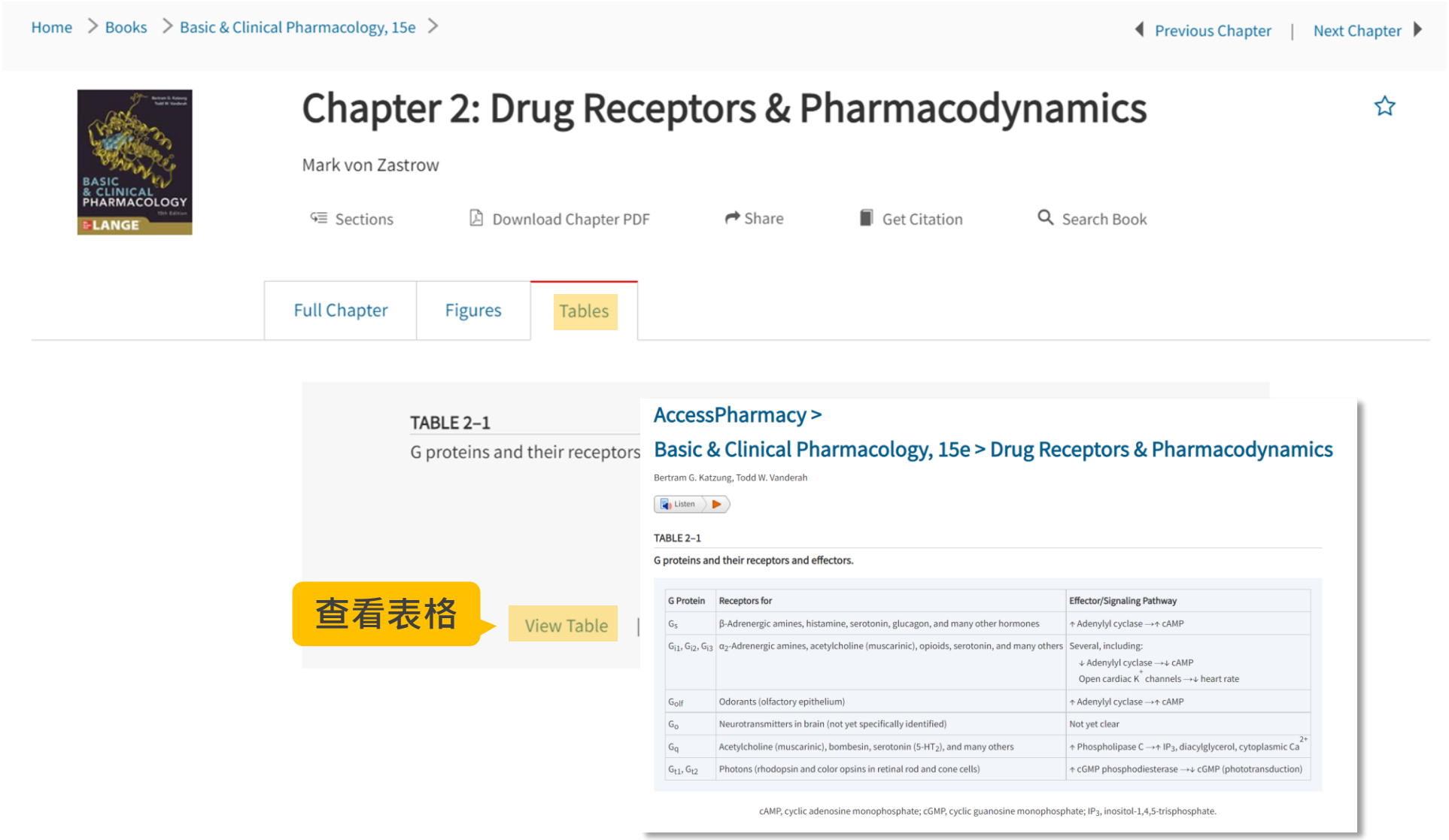

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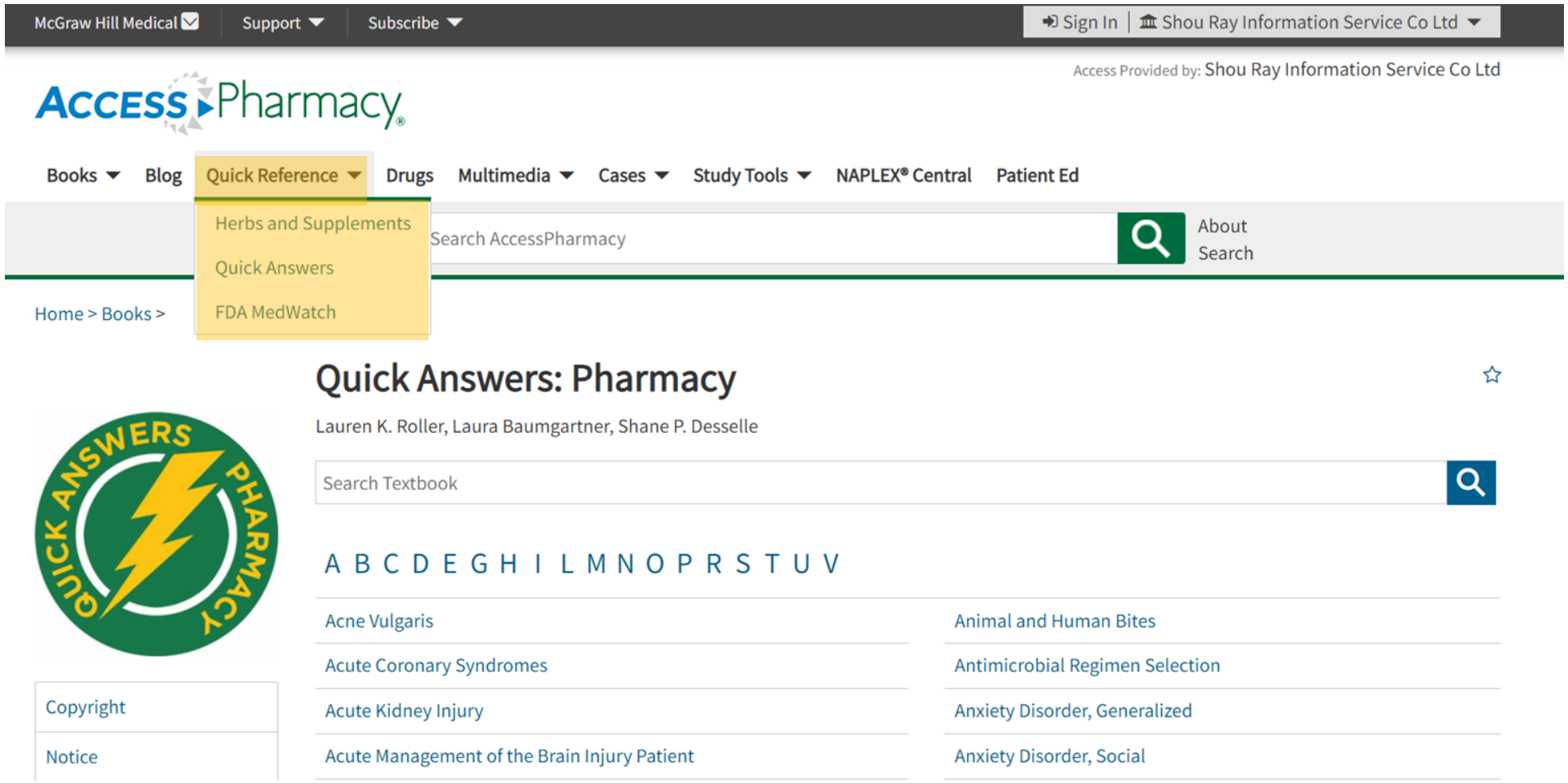

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Acidosis, Respiratory	Anemia, Iron Deficiency (IDA)
Acne Vulgaris	Anemia, Vitamin B12 Deficiency
Acute Coronary Syndromes	Antimicrobial Regimen Selection
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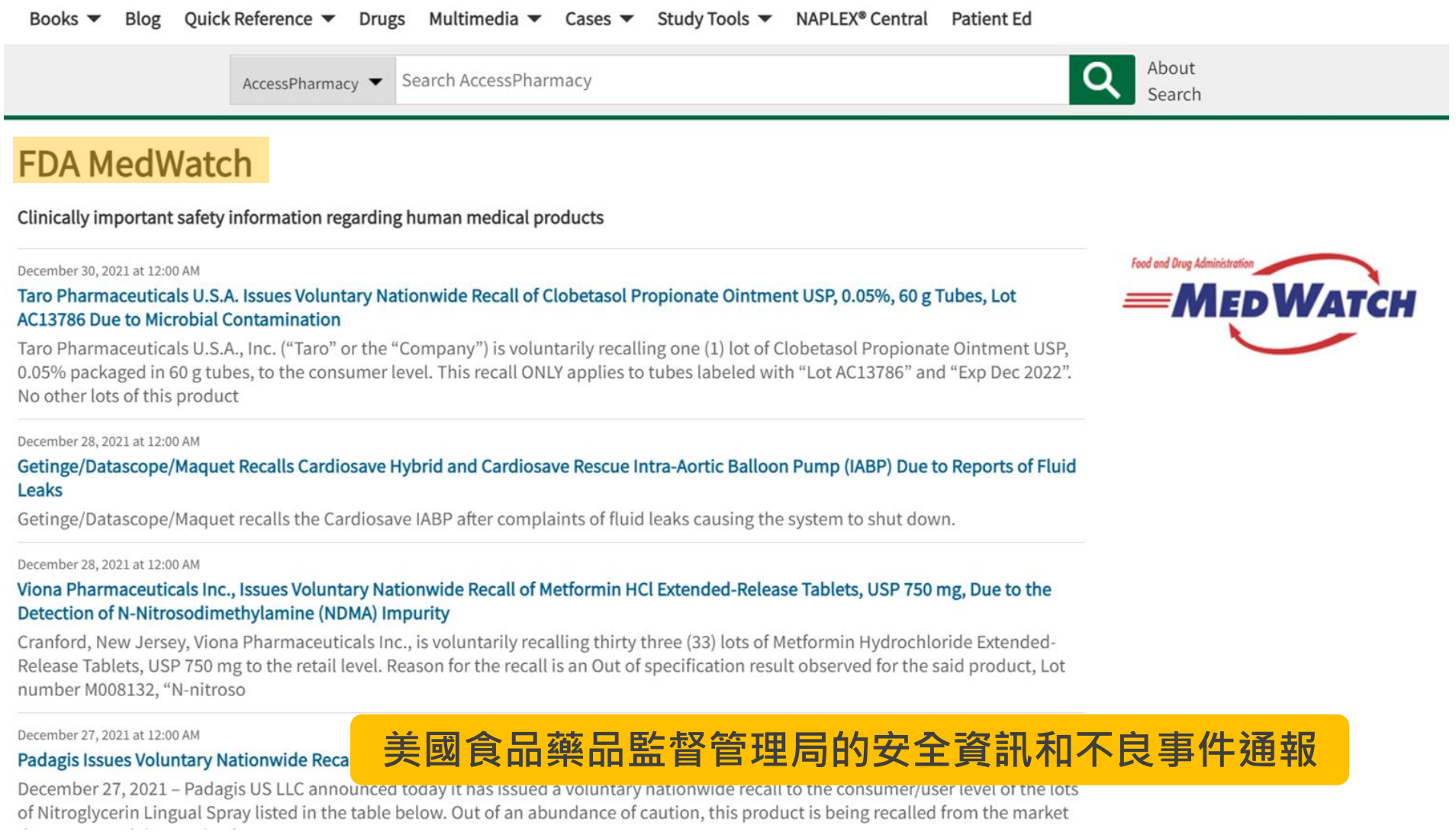

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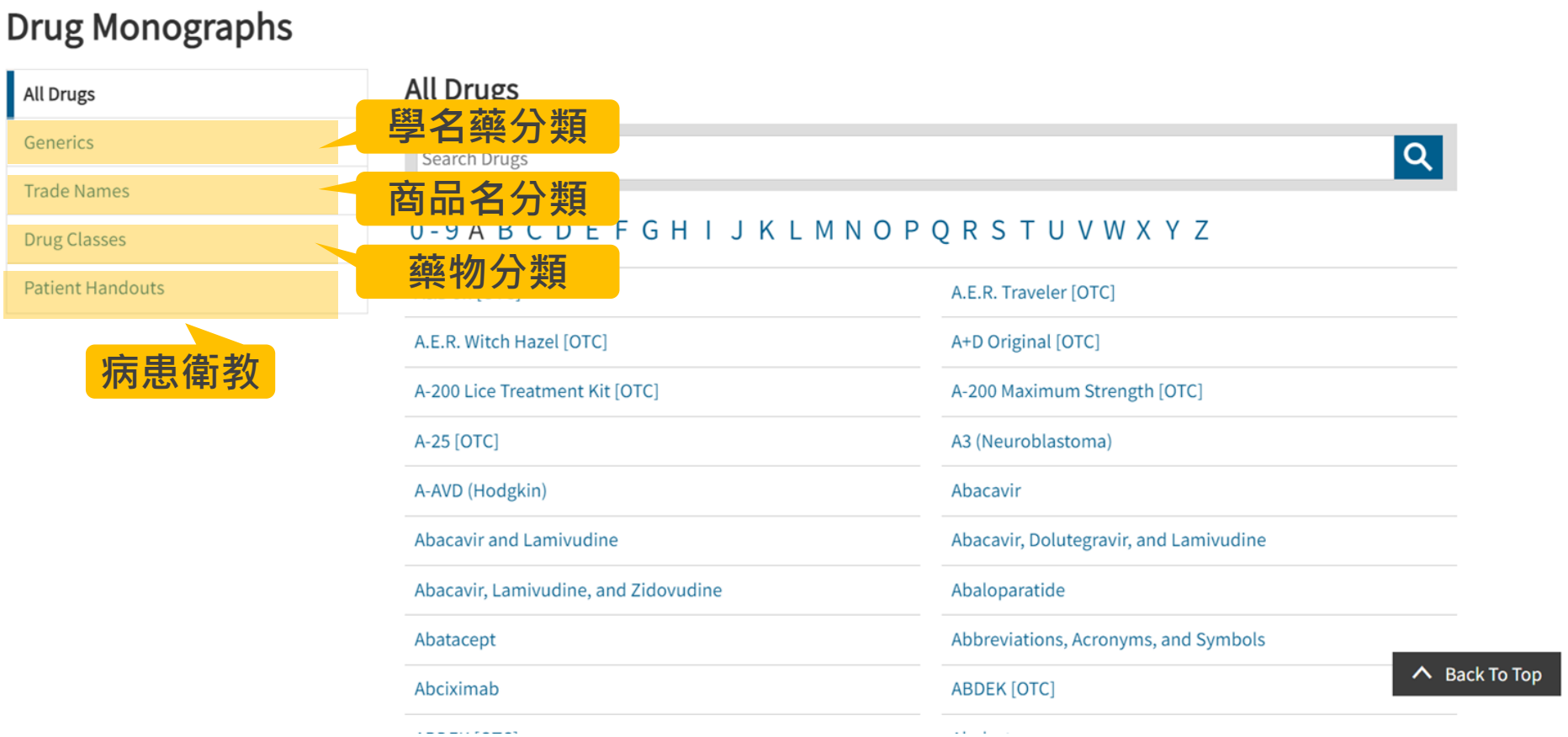

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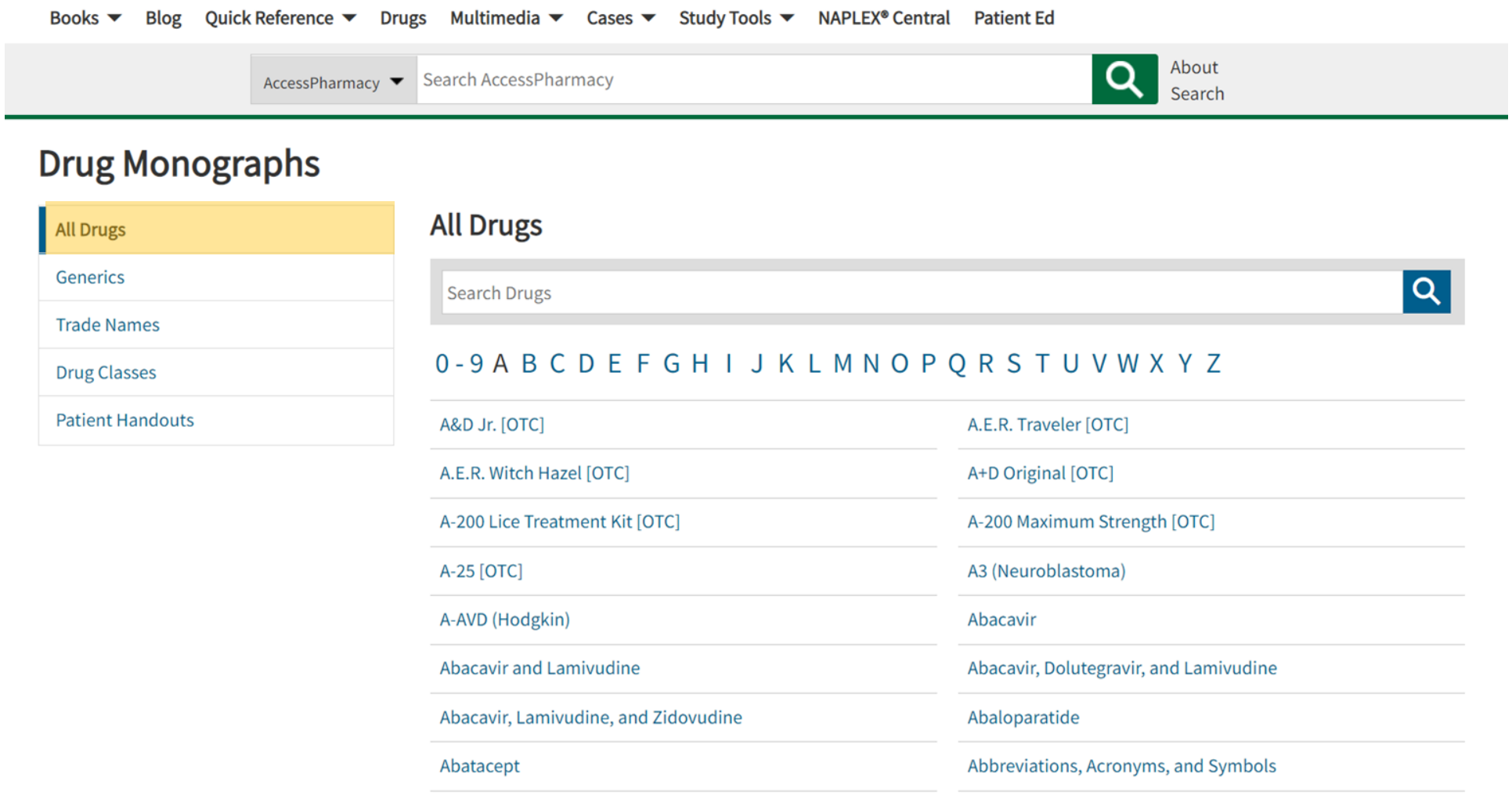
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A&D Jr. [OTC]
A.E.R. Traveler [OTC]
A.E.R. Witch Hazel [OTC]
A+D Original [OTC]
A-200 Lice Treatment Kit [OTC]
A-200 Maximum Strength [OTC]
A-25 [OTC]
A3 (Neuroblastoma)
A-AVD (Hodgkin)
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Abacavir and Lamivudine
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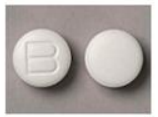 Formulation Details	Bufferin [GLAXO CONSUMER HEALTHCARE L.P.] 325 mg
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Pronunciation

(AS pir in)

Brand Names: US

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- Ascriptin Regular Strength [OTC]

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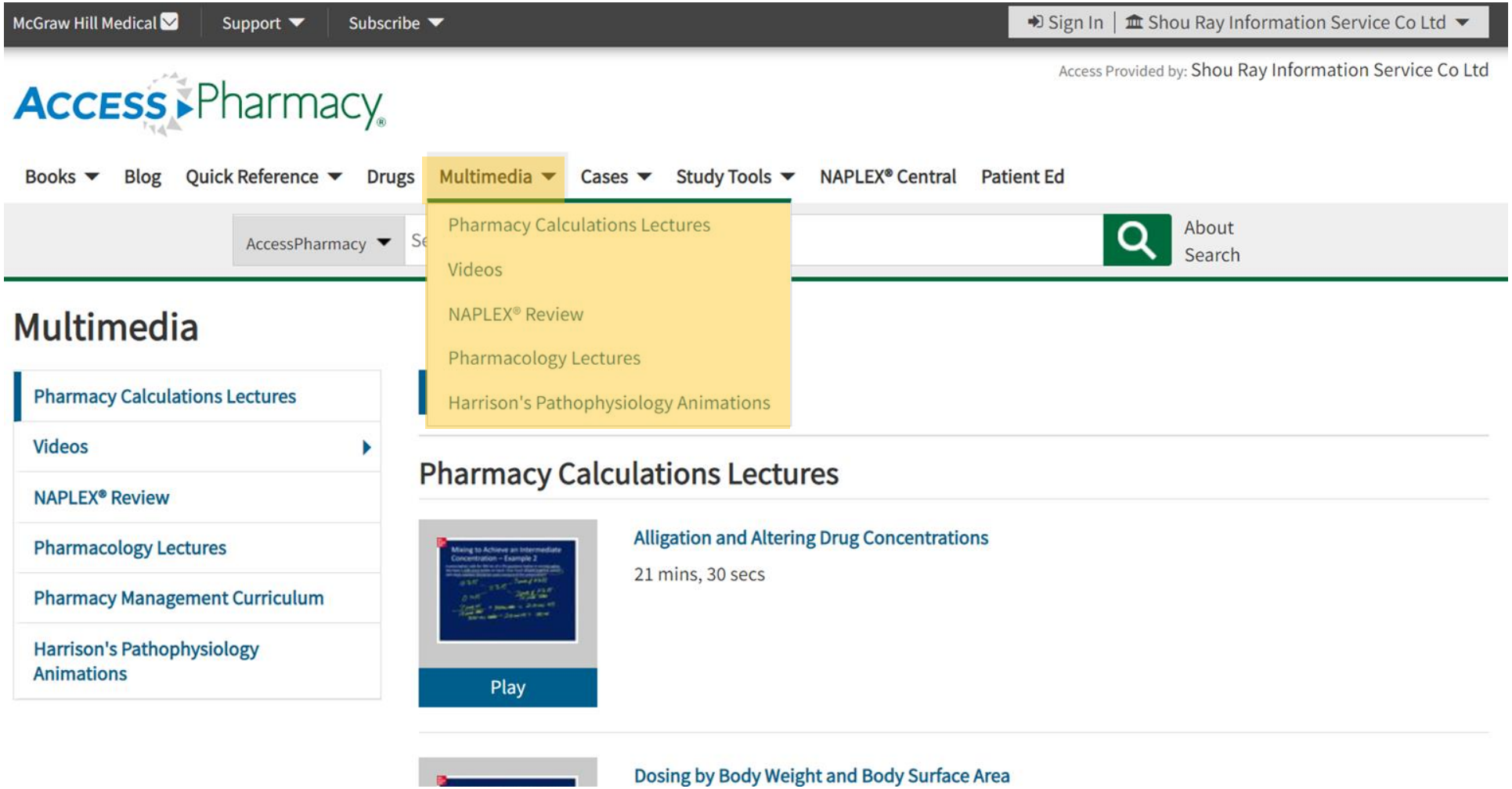
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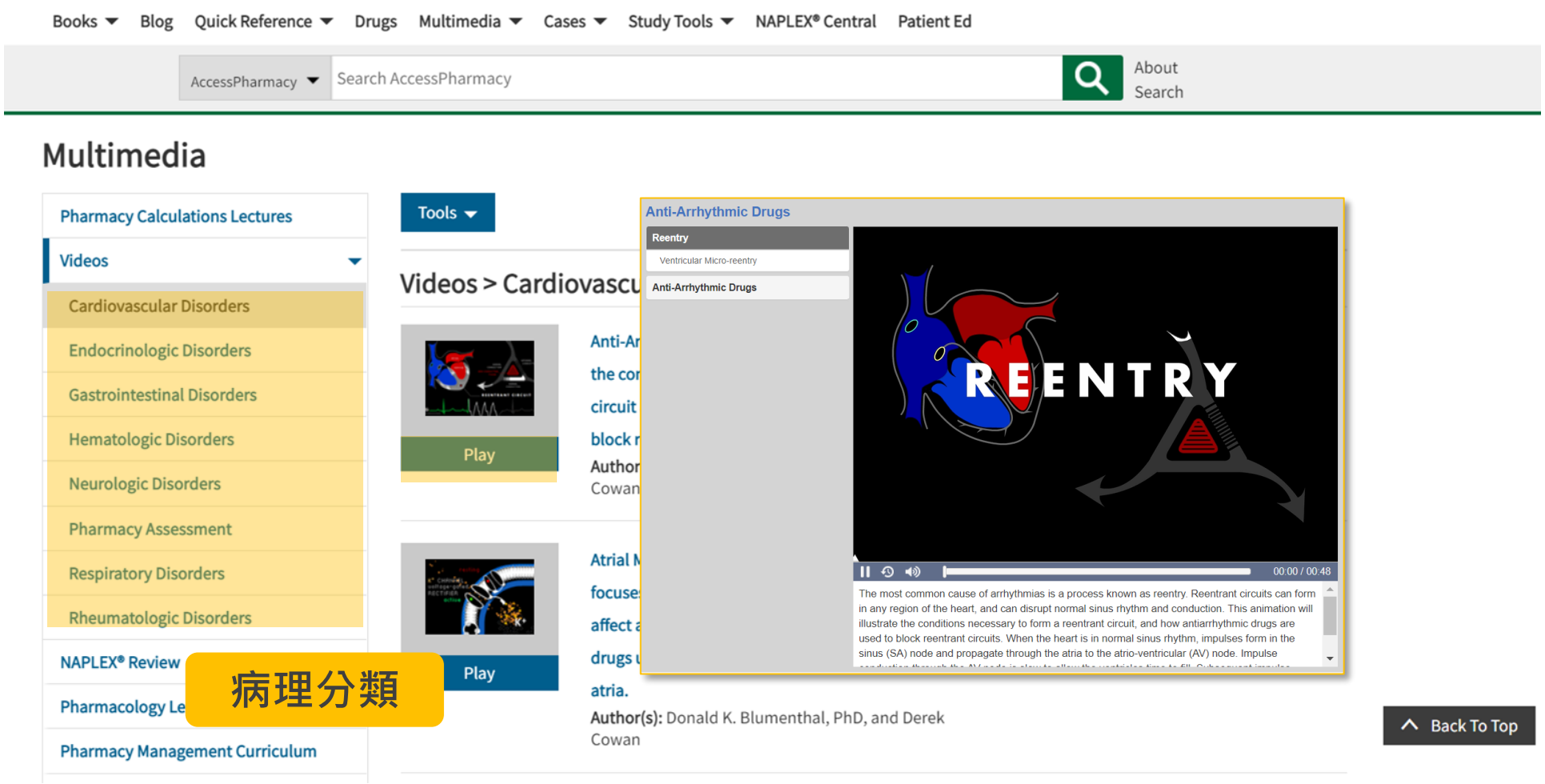

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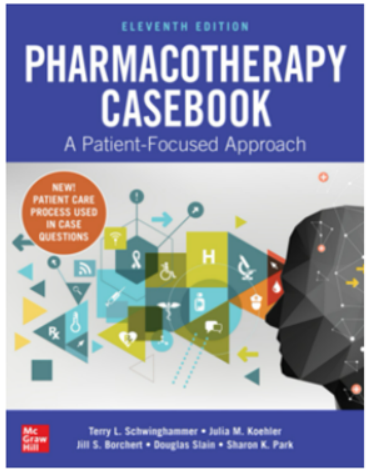

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Atrial Fibrillation

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Listen

After completing this case study, the reader should be able to:

- Describe the cornerstones of atrial fibrillation (AF) treatment.
- Determine therapeutic goals for managing AF in patients with heart failure.
- Recommend an optimal agent for anticoagulation in AF patients with heart failure.

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Next: Patient Presentation

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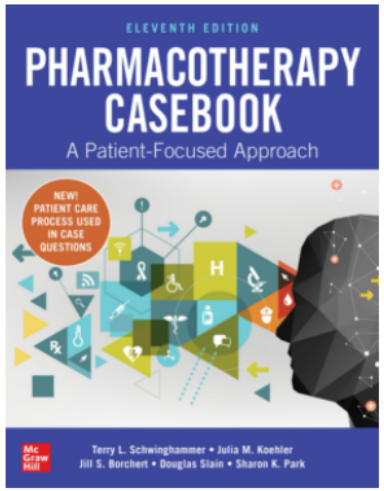

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Authors: Virginia H. Fle

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主訴

Chief Complaint

"Lately, I feel like my heart has been racing a bit. It really doesn't bother me that much, but I wanted to have it checked out to be sure."

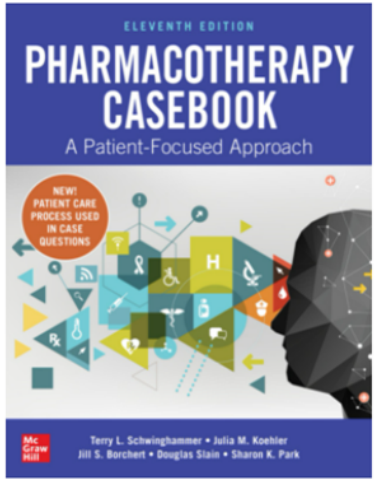
HPI 現在病史 (History of present Illness)

Cooper Riley is a 64-year-old man with heart failure and a history of persistent AF who presents to his primary care physician complaining of palpitations that he first noticed 7 days ago. He reports that he is aware of the palpitations but that he has remained relatively asymptomatic. There has not been a noticeable change in his level of fatigue or exercise capacity during his normal daily activities. Mr Riley was diagnosed with heart failure 6 years ago. For the past few years, his baseline exercise capacity would be described as slight limitation of physical activity with some symptoms during normal daily activities but asymptomatic at rest. He has a history of AF that was cardioverted to NSR, and he has been on amiodarone to maintain NSR for the past 8 months. In the office today, Mr Riley's ECG shows that he is in AF (Fig. 26-1).

FIGURE 26-1.

Rhythm recorded in Mr Riley's physician's office that depicts AF with a ventricular response rate of 110 bpm. AF is characterized by the absence of atrial "p" waves with varying distances between QRS complexes. AF is sometimes referred to as an irregularly irregular rhythm: irregular because it is not NSR; irregular because it produces an irregular ventricular response rate or peripheral pulse.

Cases 案例頁面



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練習題

Collect Information

1.a. What subjective and objective information indicates the presence of persistent atrial fibrillation with HF*r*EF?

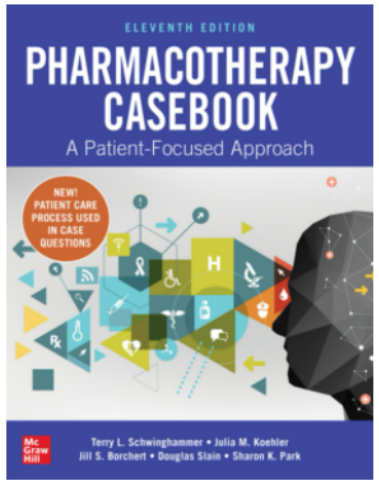
1.b. What additional information is needed to fully assess this patient?

Save Answers

Assess the Information

2.a. Assess the severity of Mr Riley's AF based on the subjective and objective information available. How would you evaluate the effectiveness of his current medication regimen?

Cases 案例頁面



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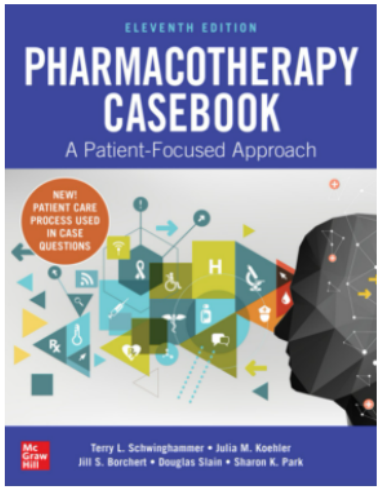
In treating AF with concomitant systolic heart failure, lenient ventricular rate control (<110 bpm) plus anticoagulation is a viable treatment option over maintaining NSR with antiarrhythmic therapy.

Next: References

Cases 案例頁面



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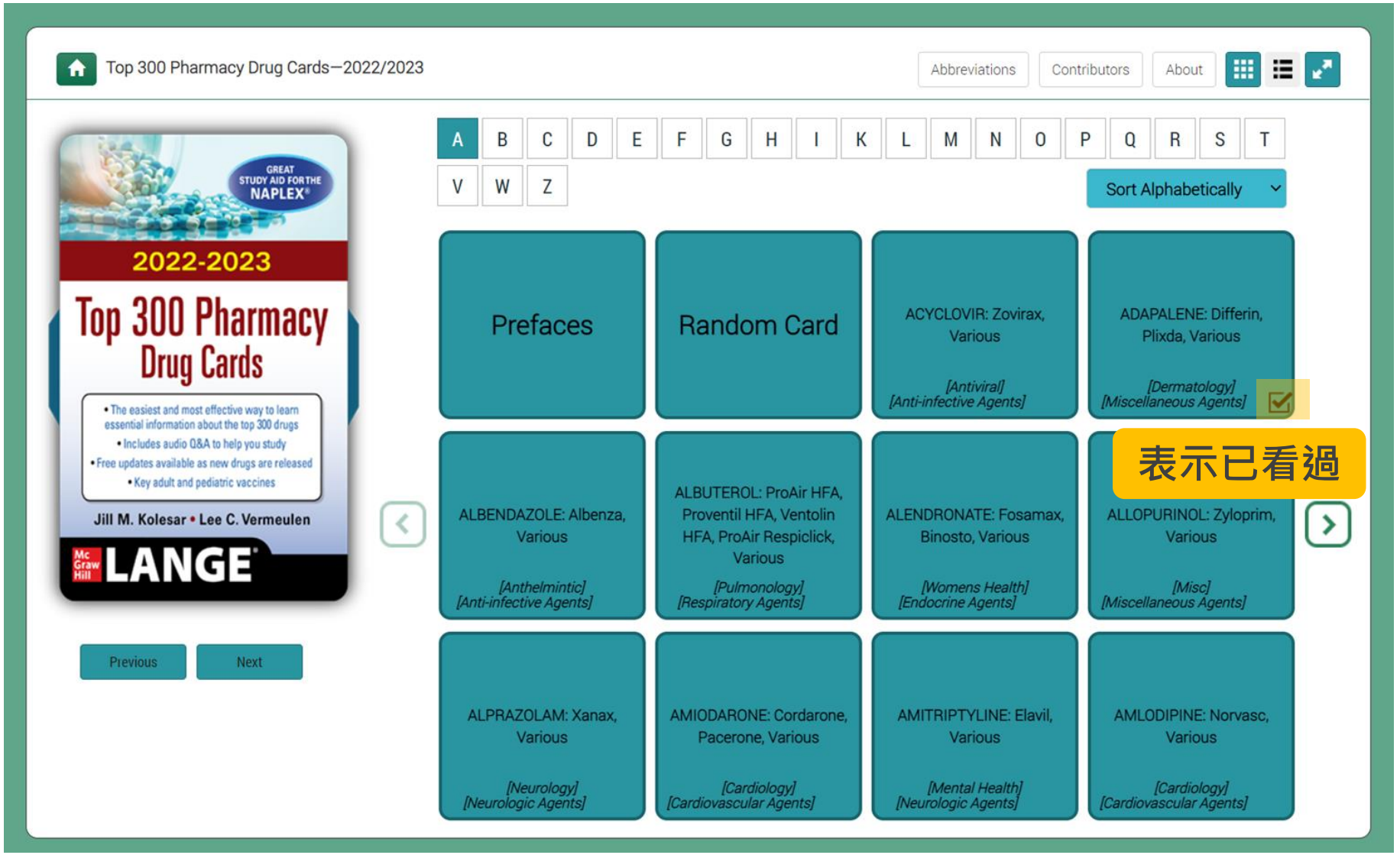

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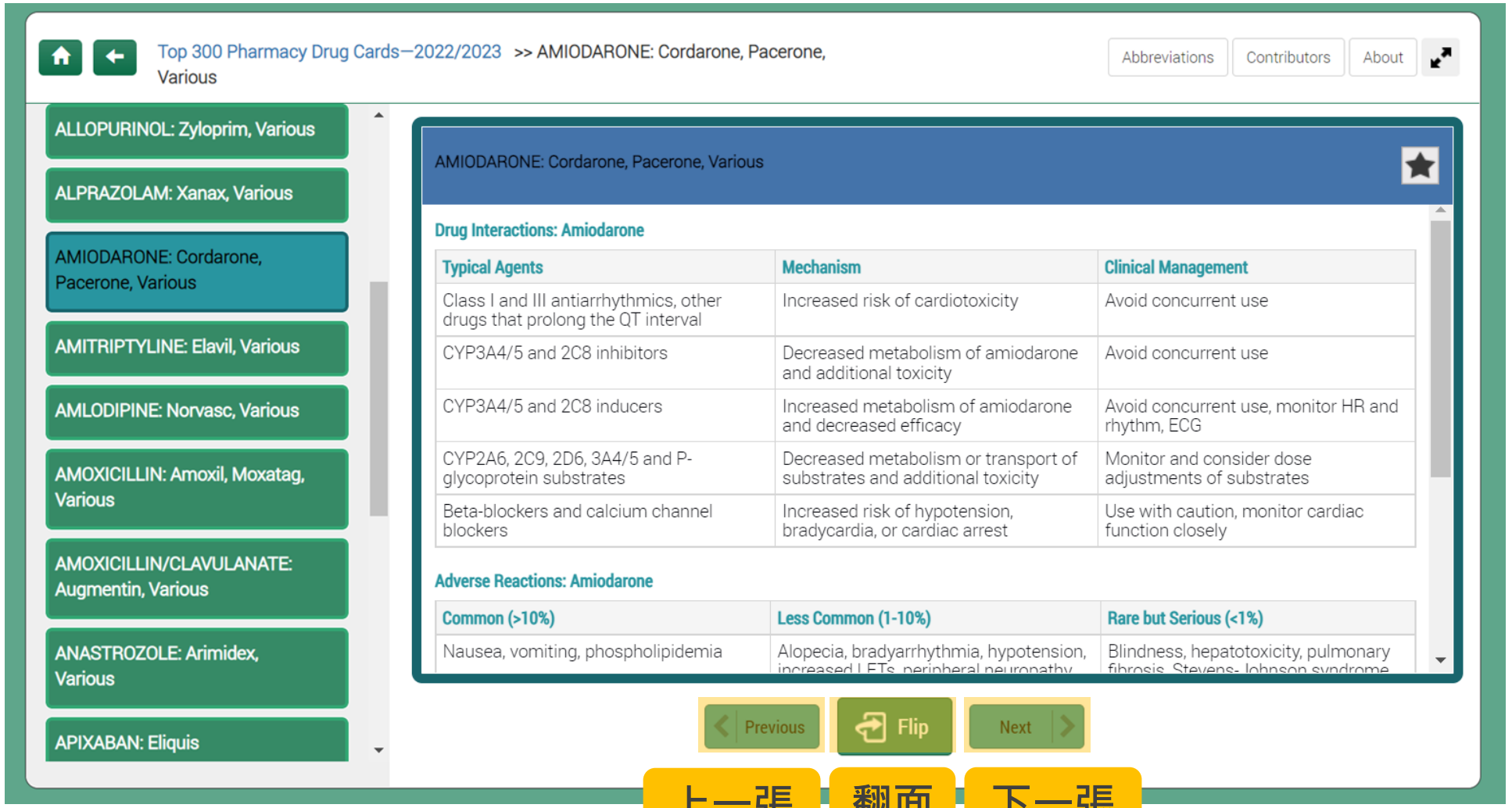

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Top 300 Prescription Drug Challenge

Test yourself on the top 300 drugs. Tiers correspond to PharmD 4-year programs. Each tier should test content appropriate to the corresponding year.

NOTE: Tier 1=Year 2; Tier 2=Year 3; Tier 3=Year 4

Anti-Infective Agents 選擇考題面向

Tier 1 (Brand/Generic and Classifications)
- Tier 1 (Brand/Generic and Classifications)
- Tier 2 (Available Dosage Forms/FDA Indications)
- Tier 3 (Dose and Patient Counseling and Adverse Drug Reactions)

Cardiology Module

Tier 1 (Brand/Generic and Classifications)

◉ Timed ○ Untimed 是否要限時

Start Test

Endocrinology Module

Edited by
Gina Carbonara Baugh, PharmD
Clinical Associate Professor
West Virginia University School of Pharmacy
Morgantown, West Virginia
Peer review completed by Ashleigh Barrickman PharmD, BCACP
Clinical Assistant Professor and Director of Skills Development
West Virginia University School of Pharmacy

Drug classification system based on Kolesar, Vermeulen: *Top 300 Pharmacy Drugs Flash Cards, 2nd ed.*
View the Top 300 Drug list

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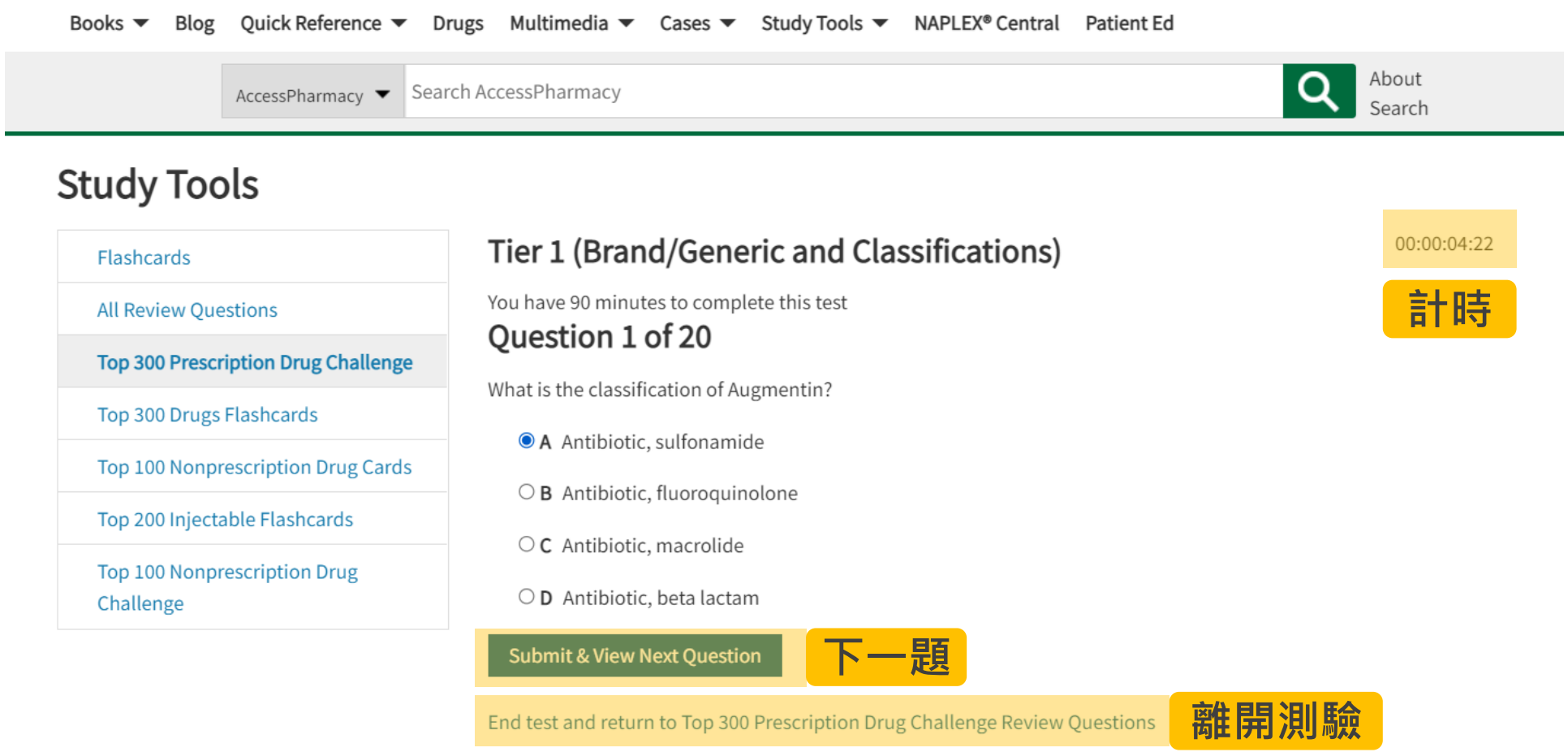

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Top 300 Prescription Drug Challenge >

Test Results

Your Score: 55 %

| Your Time: 00:00:42:47
You answered 3 of 20 questions correctly.

Question 1: Incorrect

Which of the following statements is *FALSE*?

A Truvada is an antiretroviral medication

B Zyclara is an immune response modifier

✓ C The generic of Cleocin T is azithromycin

✗ D The generic of Ceftin is cefuroxime

The correct answer is **C**. You answered **D**.

86% of users answered correctly.

答對率

Source: Top 300 Prescription Drug Challenge

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STUDY HINT

- First, complete all the 1800+ questions from the NAPLEX® Review book 4/e
- Then, go into the NAPLEX® Q&A and test yourself on these 1500 questions

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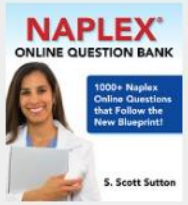
- The NAPLEX® Review Book 4/e and the NAPLEX Online Q-Bank have a total of 3300 UNIQUE questions combined.



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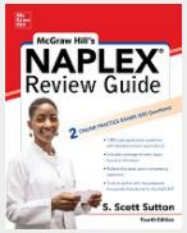

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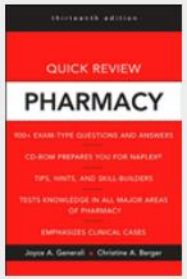

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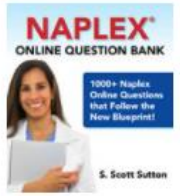

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Ensure Safe and Effective Pharmacotherapy and Health Outcomes

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Question 1 of 3

RC is a 35-year-old, 170 lb man. He reports a toothache for which he has been taking four 500-mg acetaminophen tablets every 3 to 4 hours for th right upper quadrant pain. He d does report consuming a 12 pac examination. Which of the follo

- A Acetaminophen level sl therapy is indicated.
- B Antidote therapy shoul
- C Acetaminophen level sl
- D Activated charcoal sho for antidote therapy.

Submit & View Answer

End quiz and return to NAPLEX®

Question 1 of 3

UV is a patient with partial epilepsy being treated with oxcarbazepine. UV has been stable (seizure free) for 18 months and tolerating the medication well. UV is presenting to a triage clinic with complaints of nausea, vomiting, headache, confusion, lack of energy, fatigue, restlessness, irritability, muscle cramps and recent seizure. The emergency room provider is evaluating possible causes of UV's symptoms. UV has a past medical history significant for hypertension, diabetes, dyslipidemia and partial epilepsy. Medications include metoprolol tartrate, metformin, pitavastatin and oxcarbazepine. Family and social history are non-contributory. The emergency room provider consults the pharmacy department to assist with drug and health information of drug-induced syndromes. Select the drug-induced syndrome that UV is most likely experiencing from oxcarbazepine.

- A Lactic acidosis
- ✗ B Rhabdomyolysis
- C Bradycardia
- ✓ D Hyponatremia

Next Question

You will be able to view all answers at the end of your quiz.

The correct answer is **D**. You answered **B**.

Explanation:
The correct answer is (D).

Oxcarbazepine can cause a clinically significant hyponatremia (sodium < 125 mmol/L).

Answer (A) is incorrect. Lactic acidosis is a potential side effect of metformin therapy.

Answer (B) is incorrect. Rhabdomyolysis is a potential side effect of pitavastatin.

Answer (C) is incorrect. Bradycardia is a potential side effect of metoprolol.

56% of users answered correctly.

End quiz and return to NAPLEX® Online Question Bank Review Questions

Patient Ed

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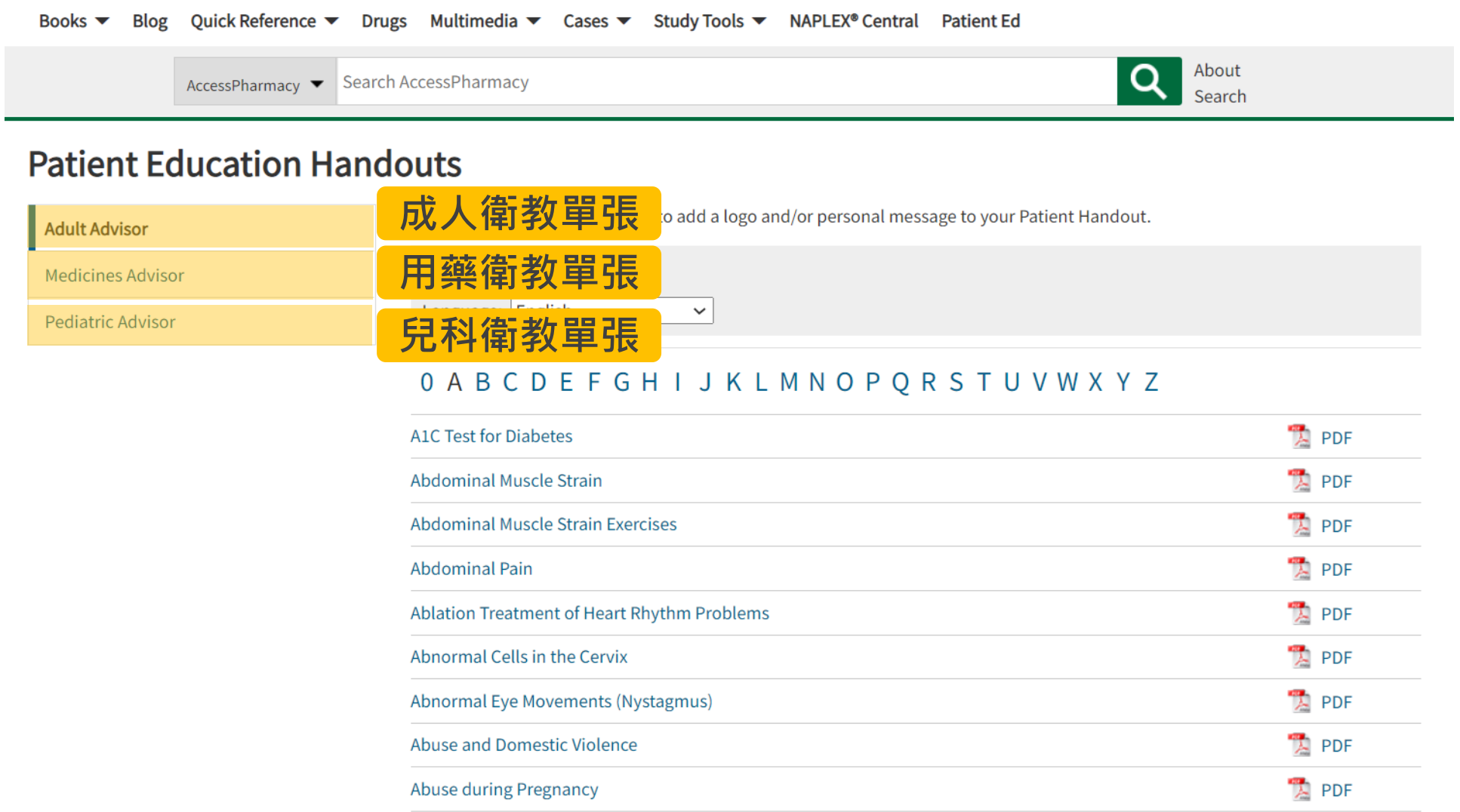

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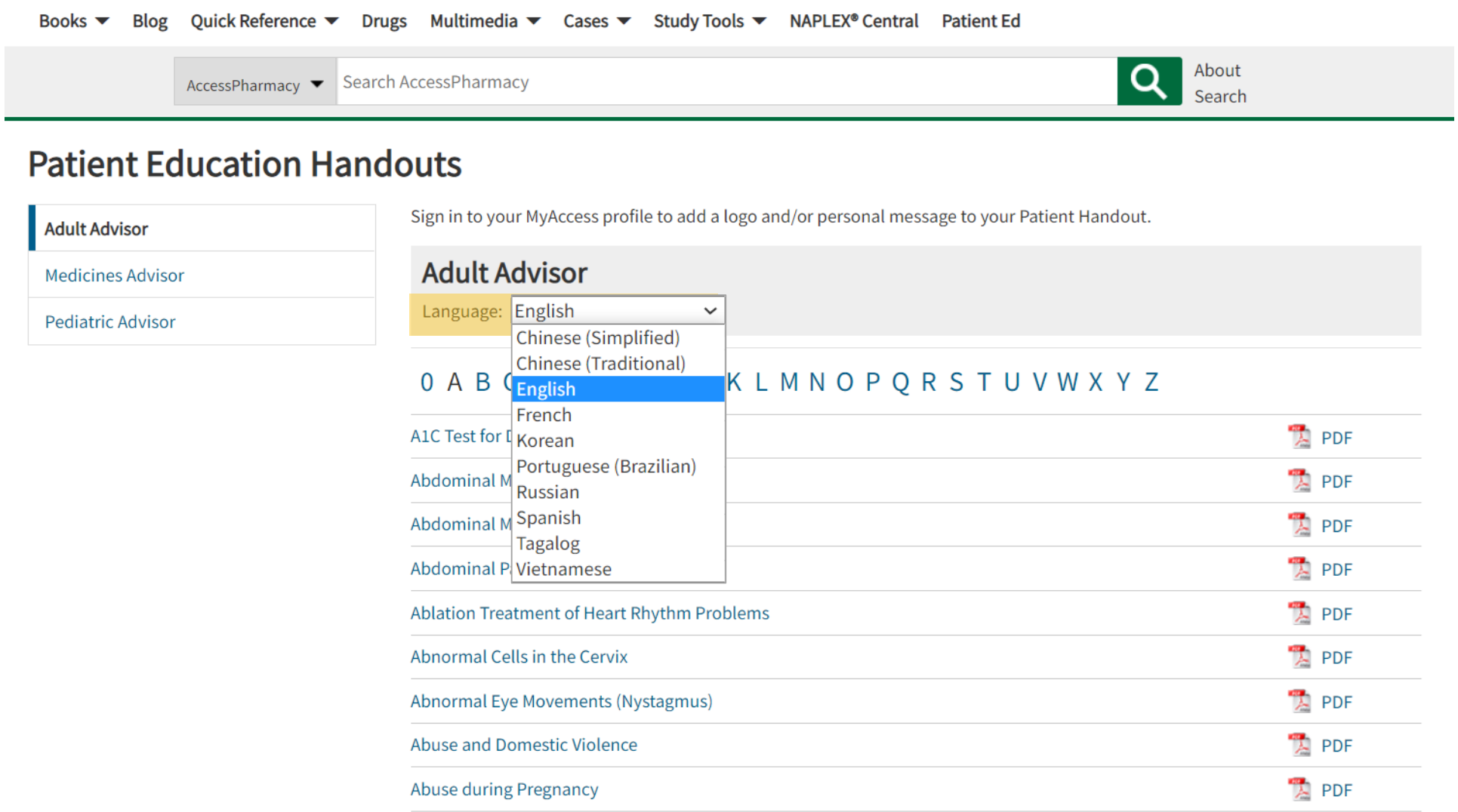

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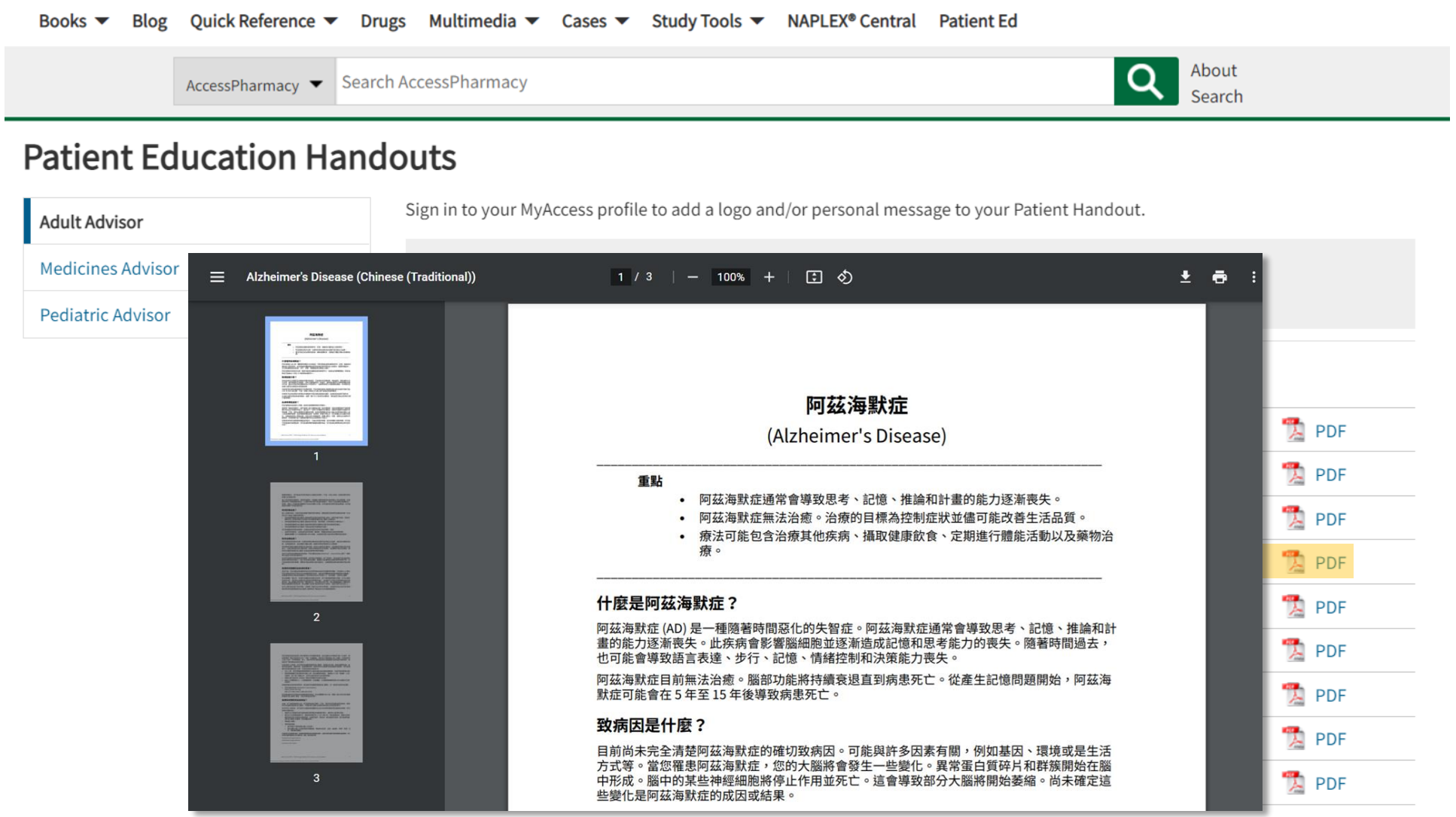

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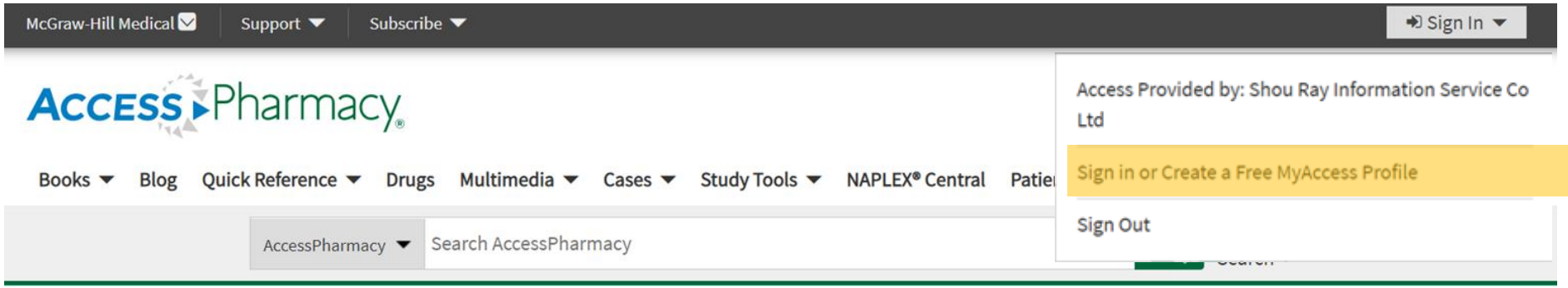

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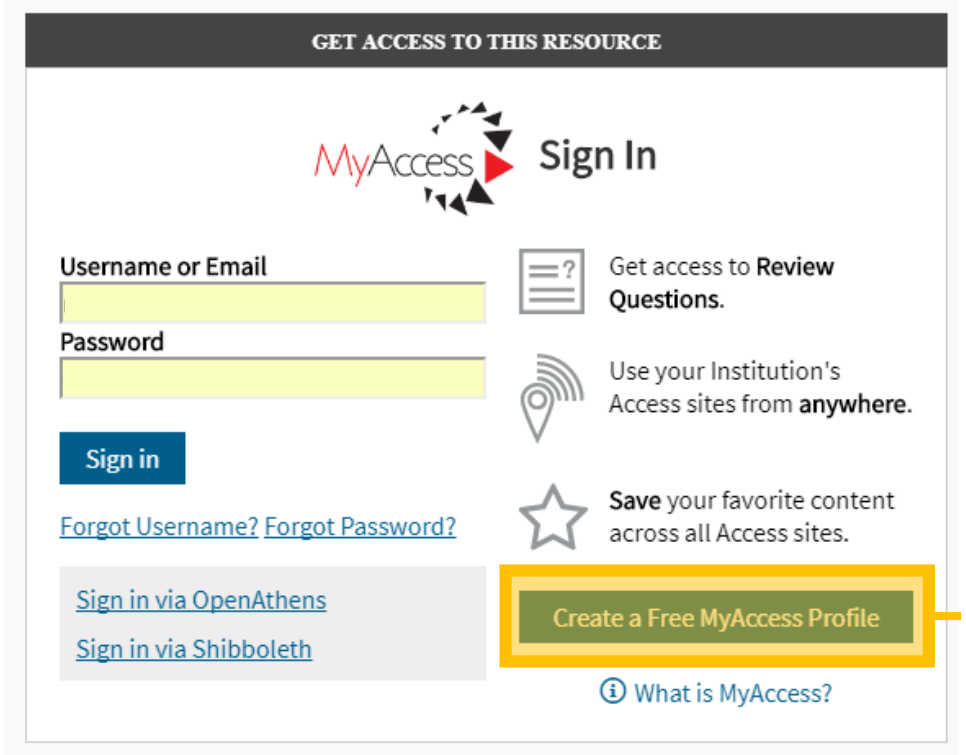
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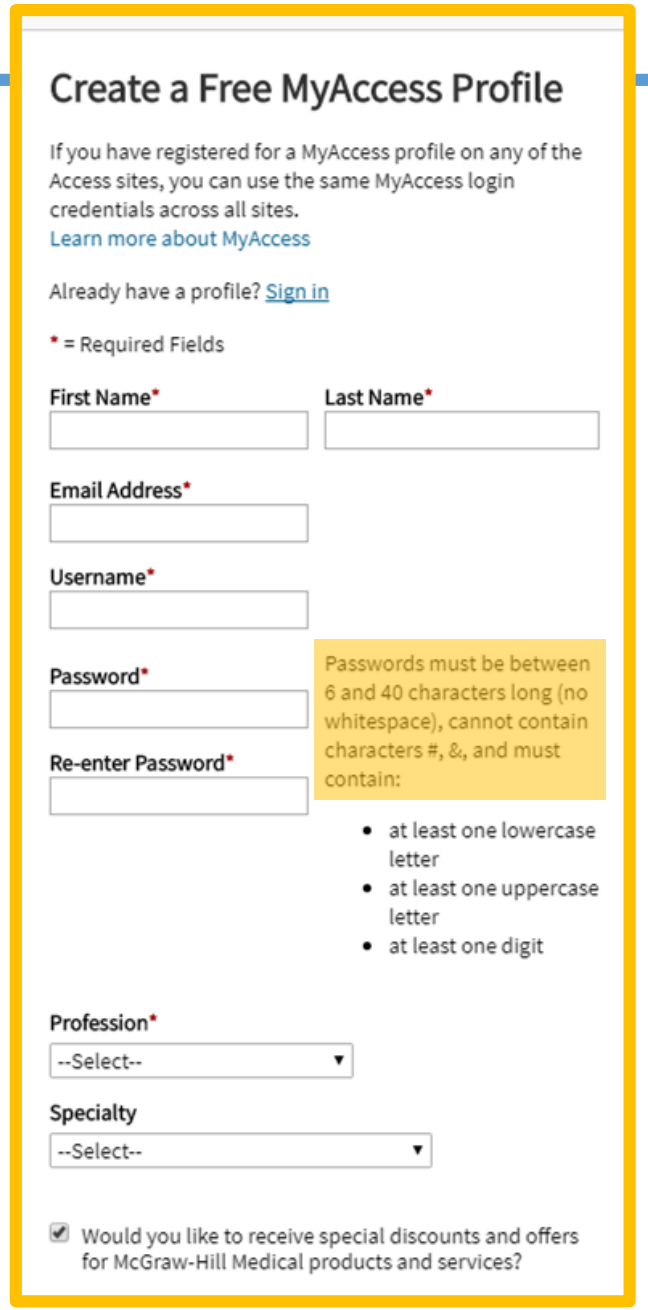
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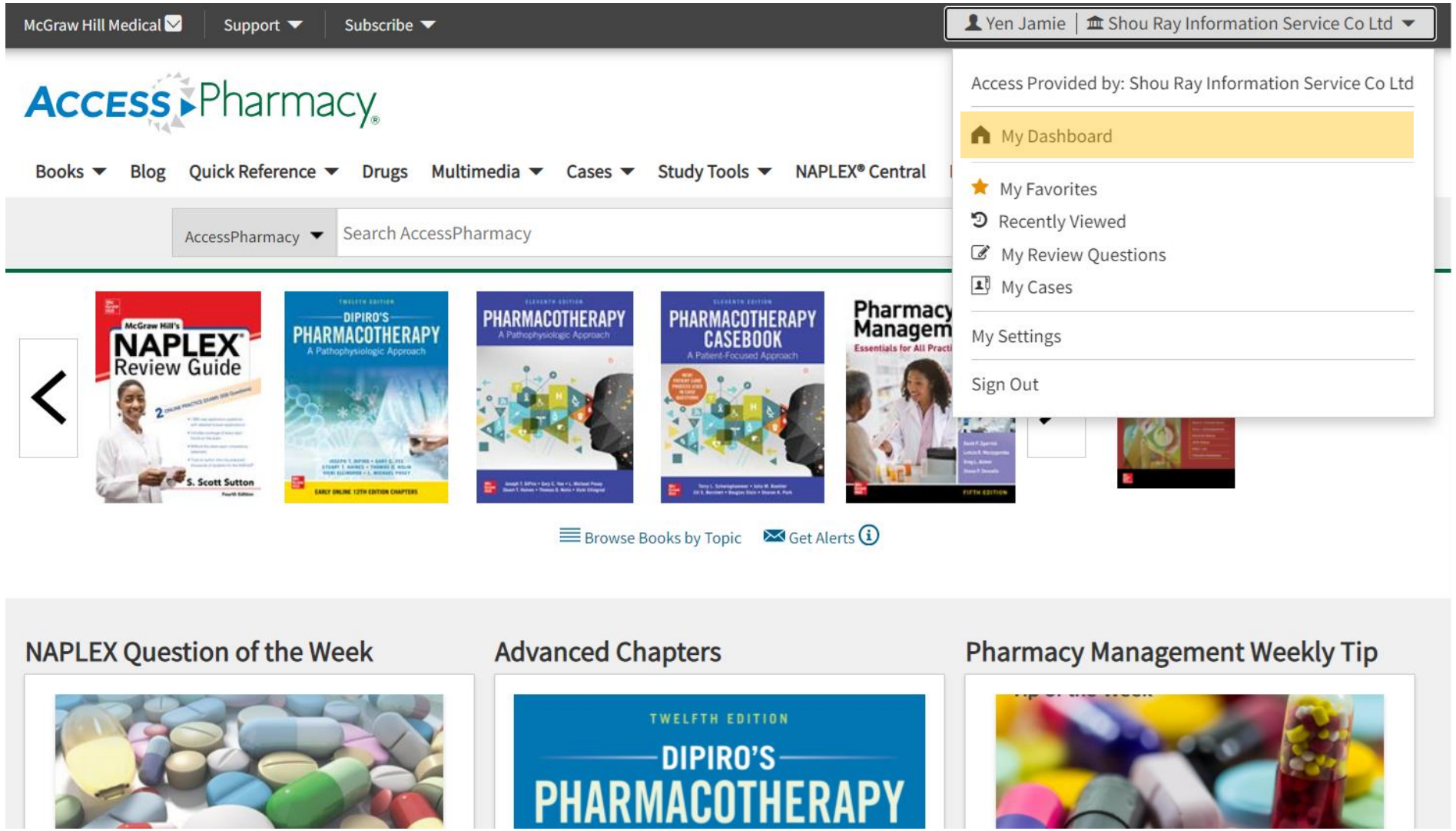
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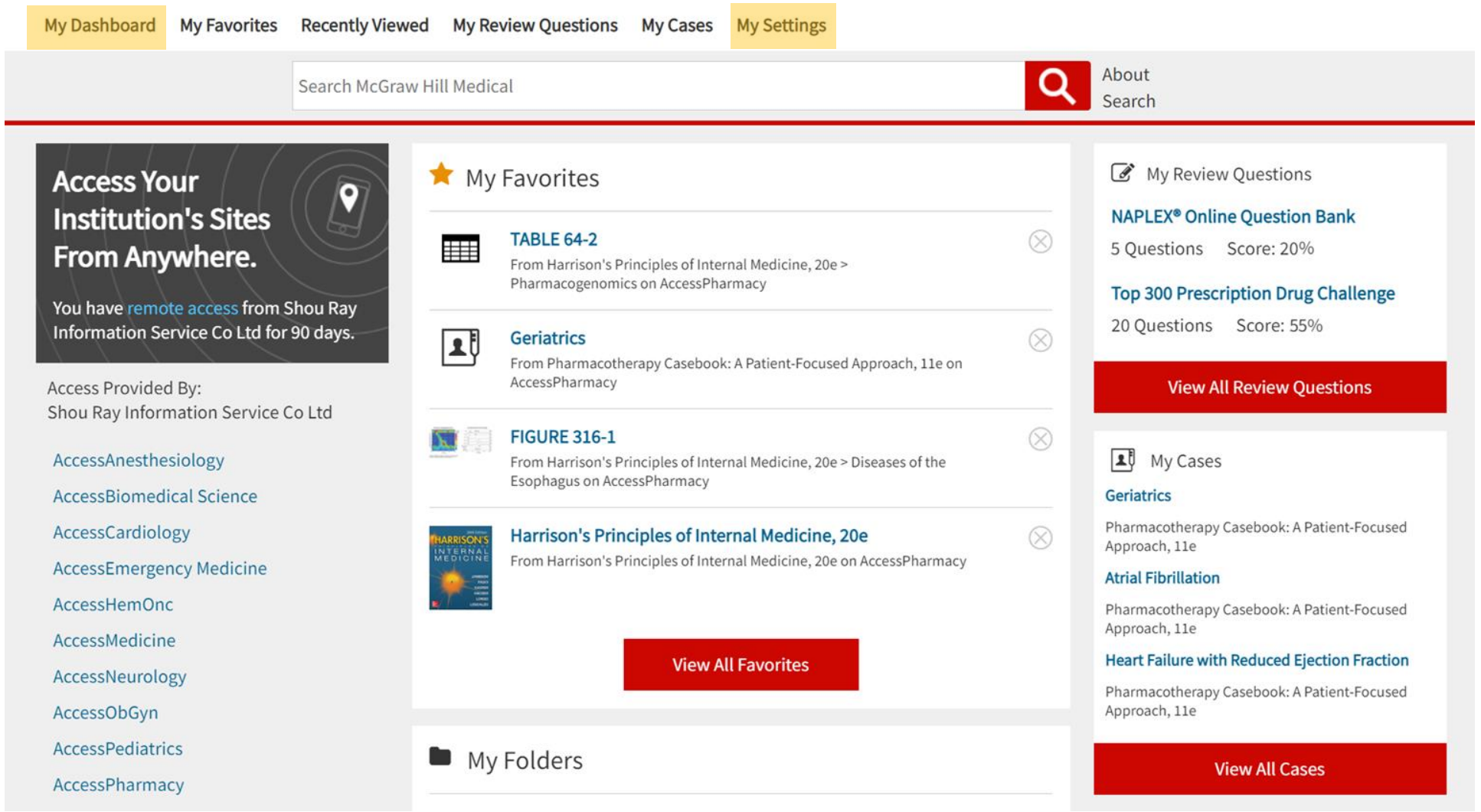

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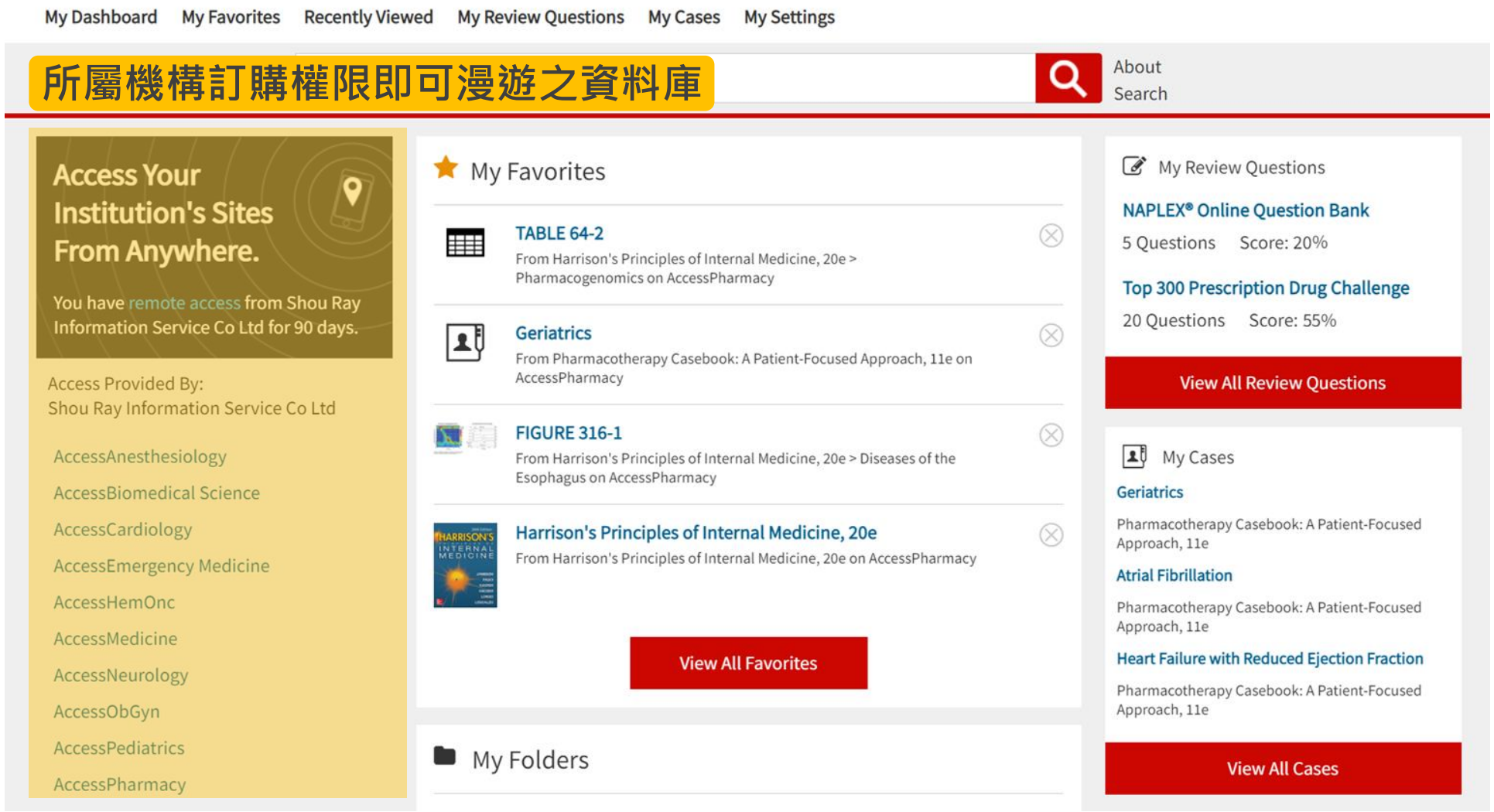
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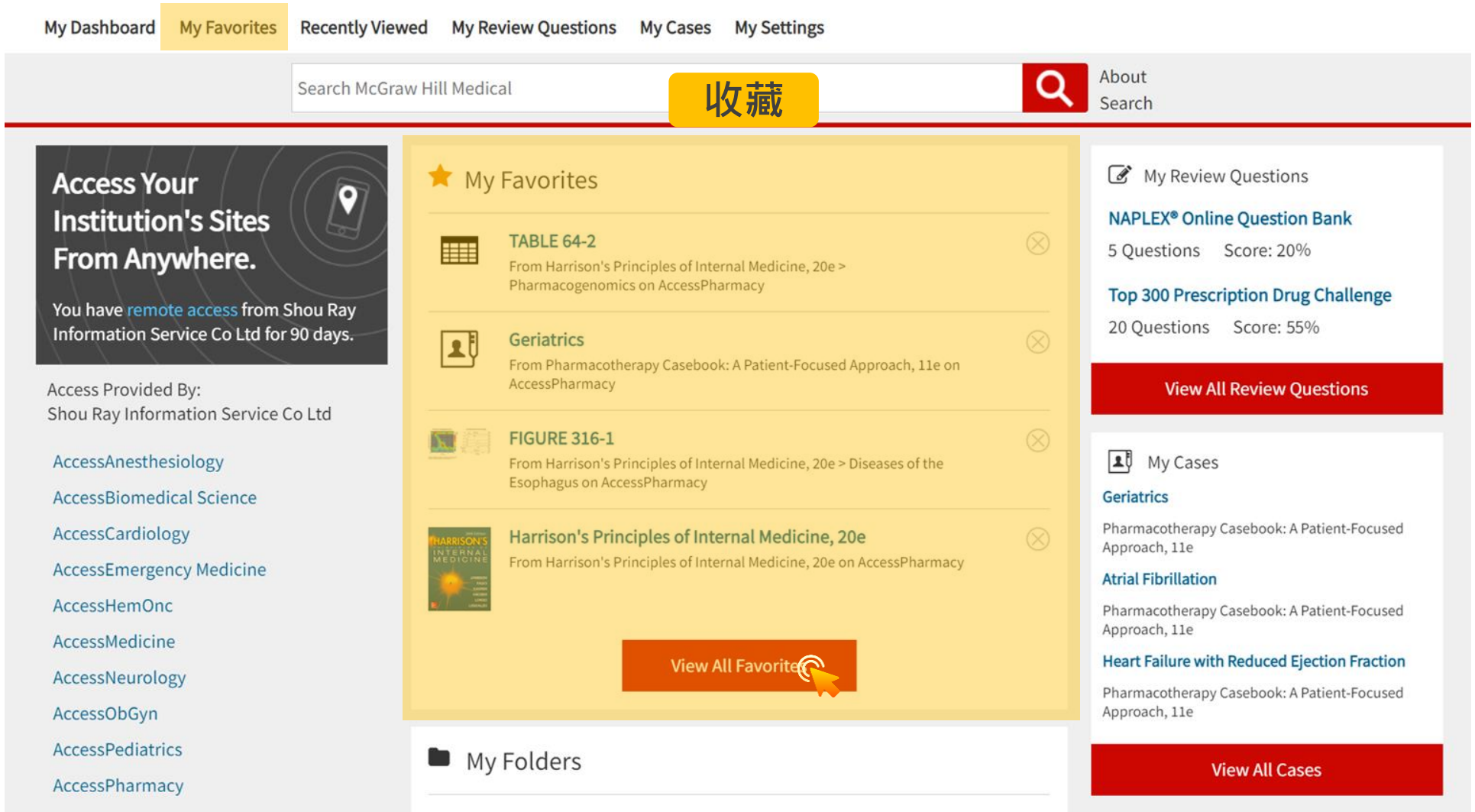
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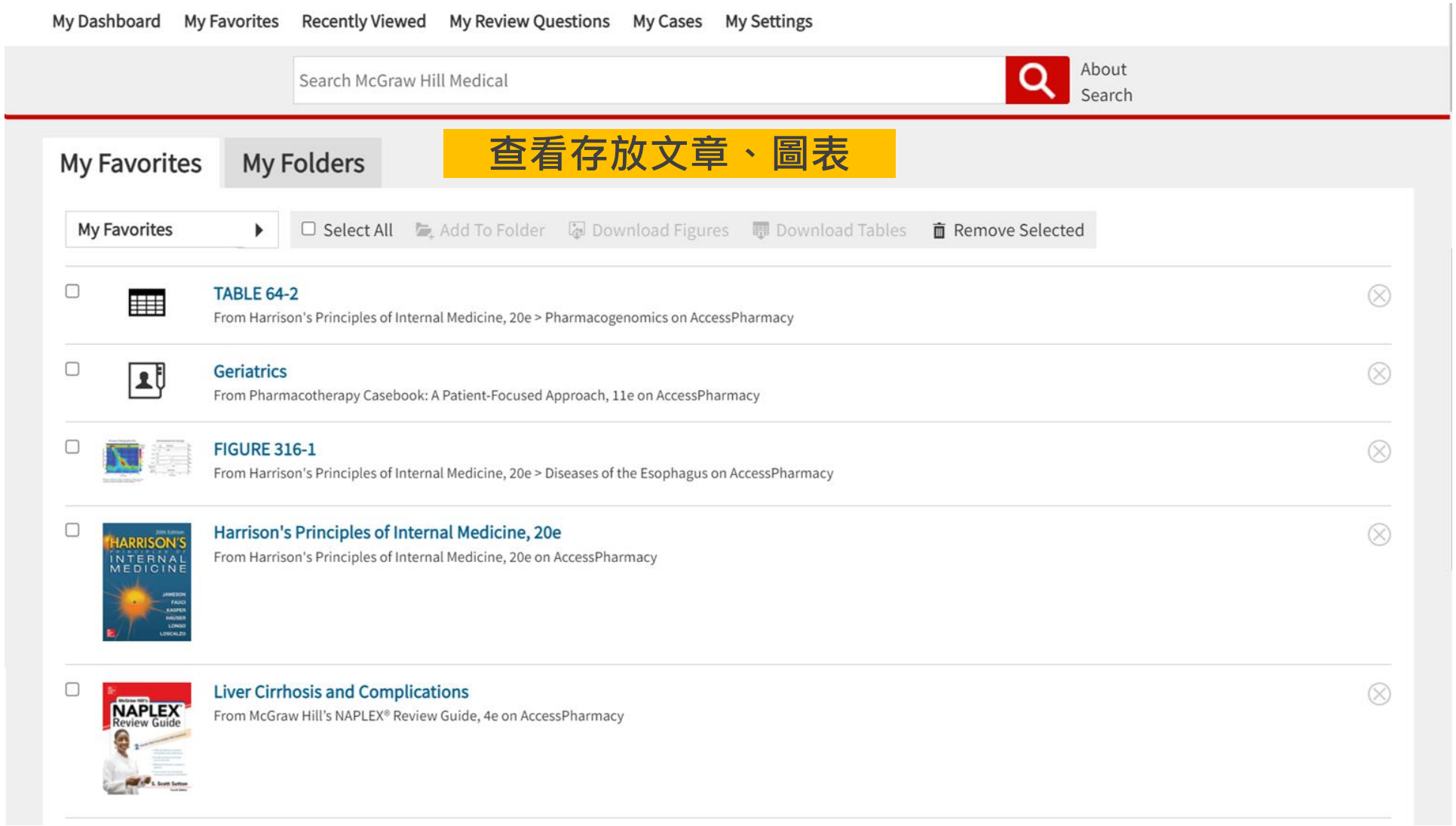

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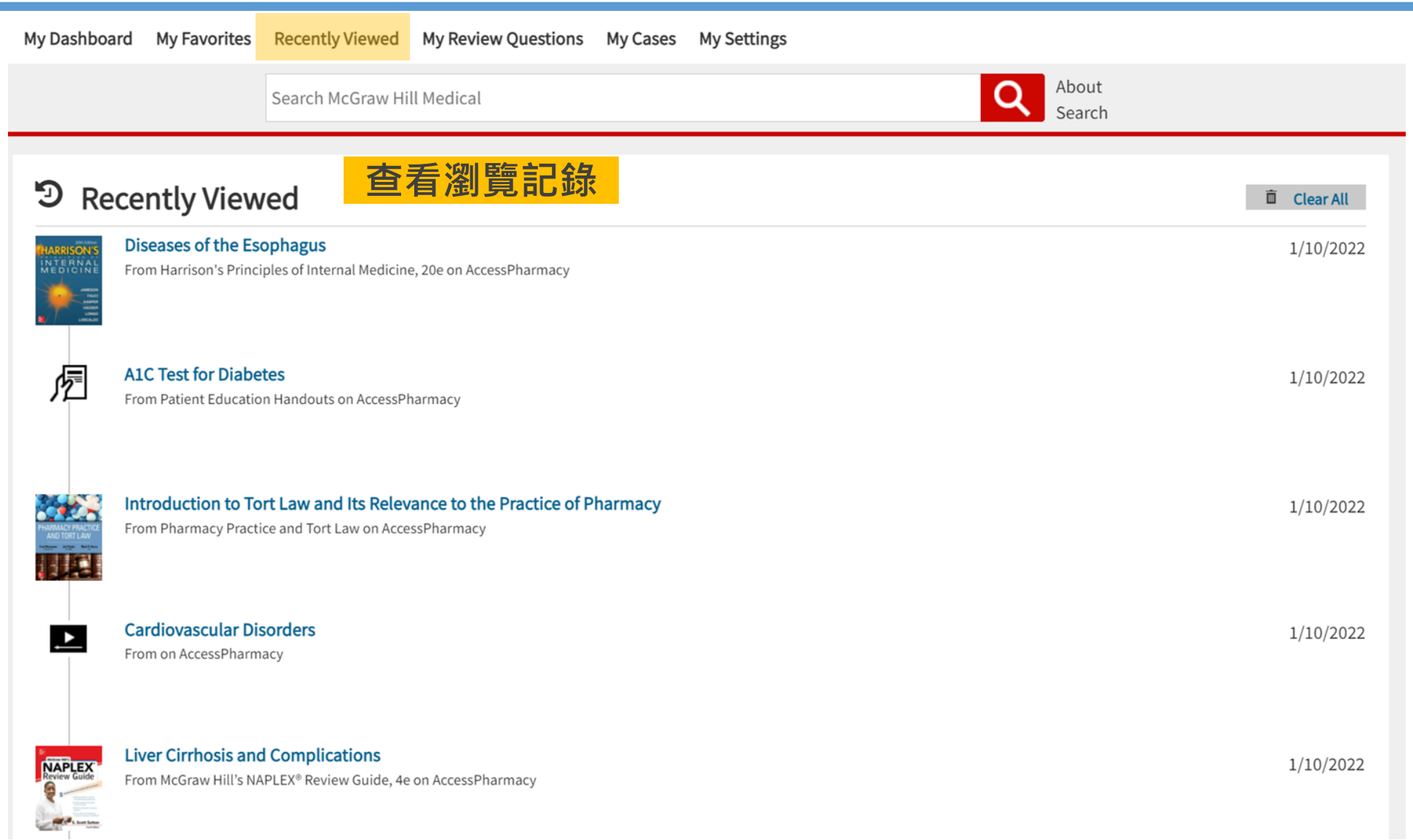

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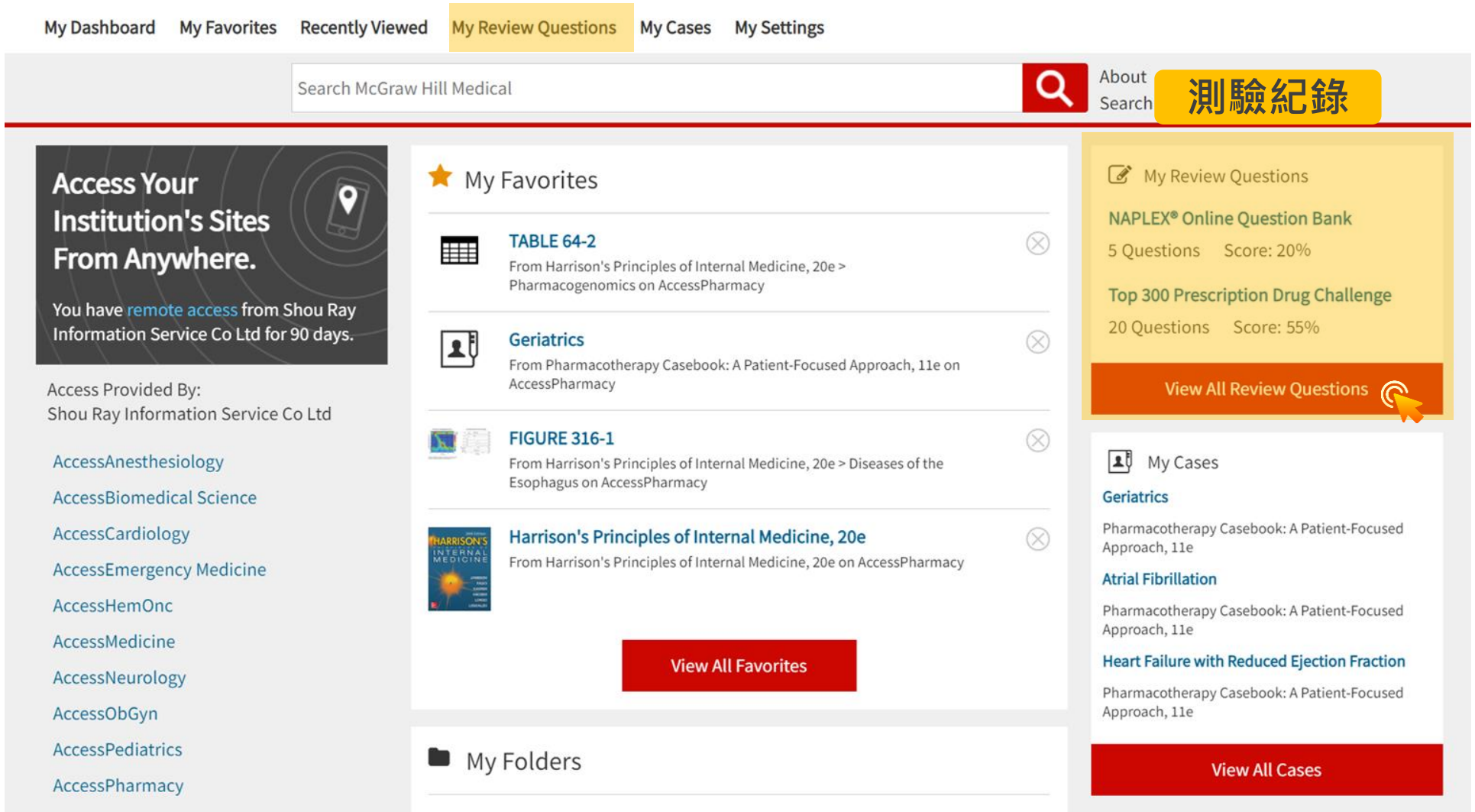
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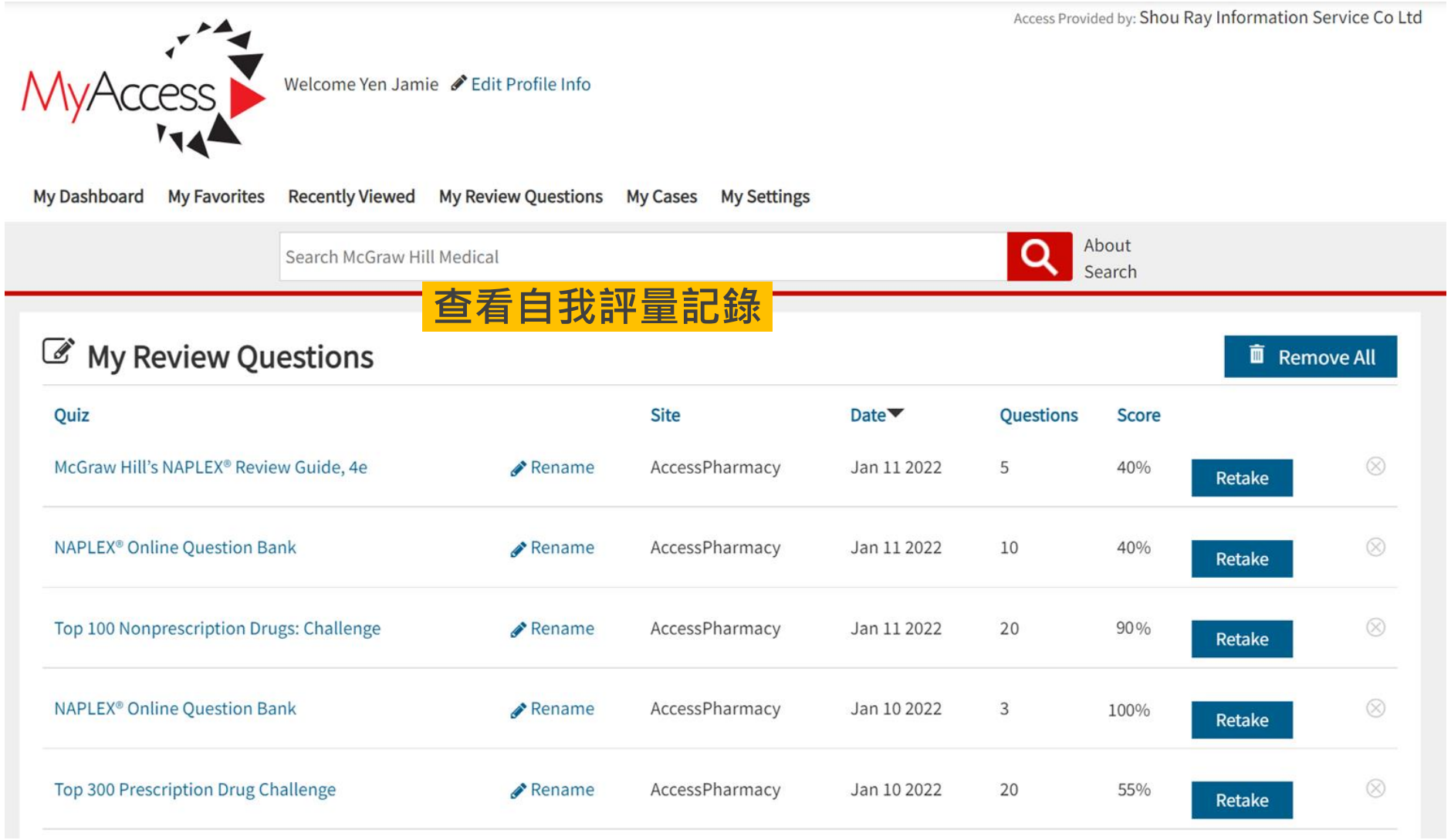

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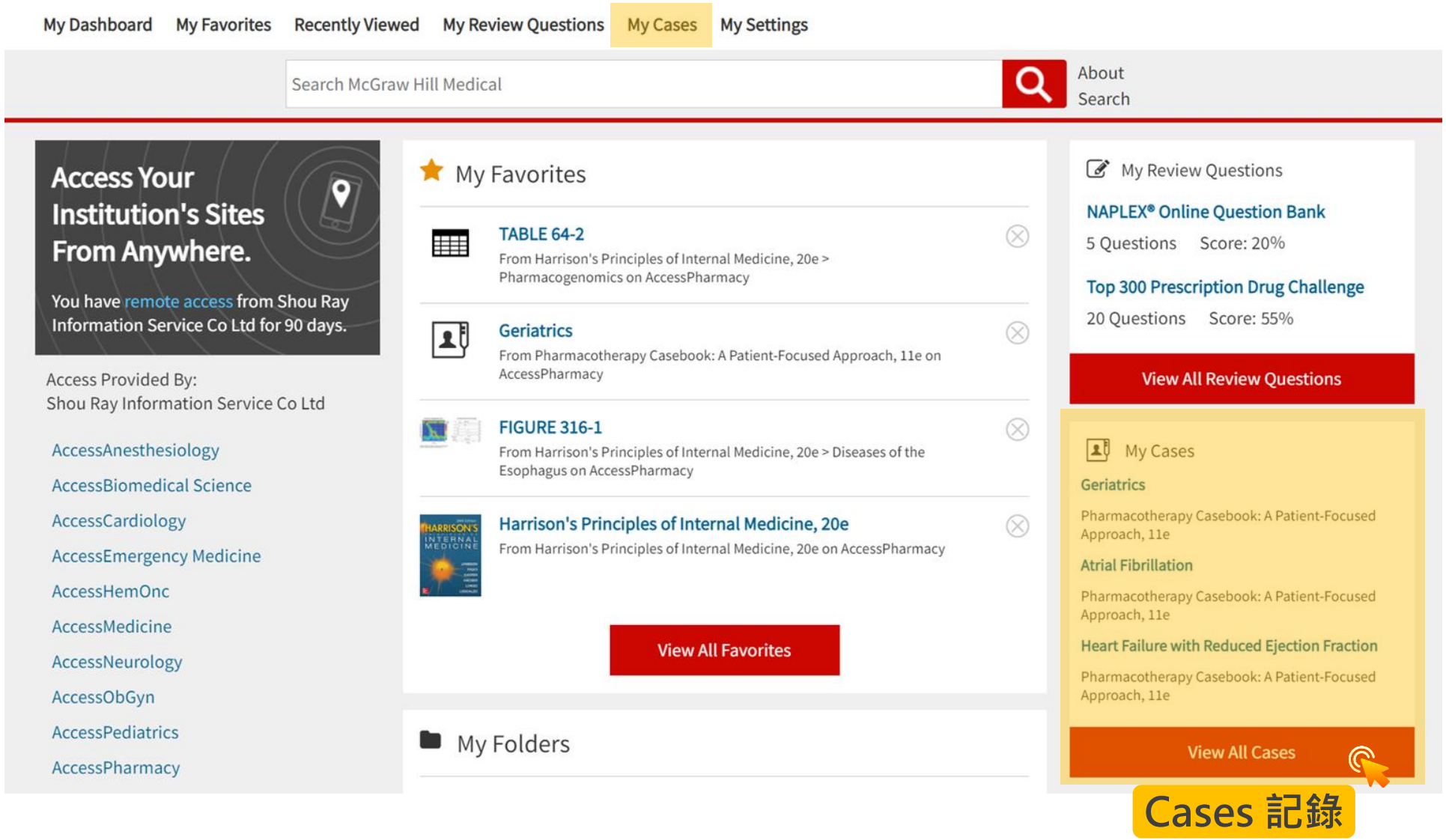
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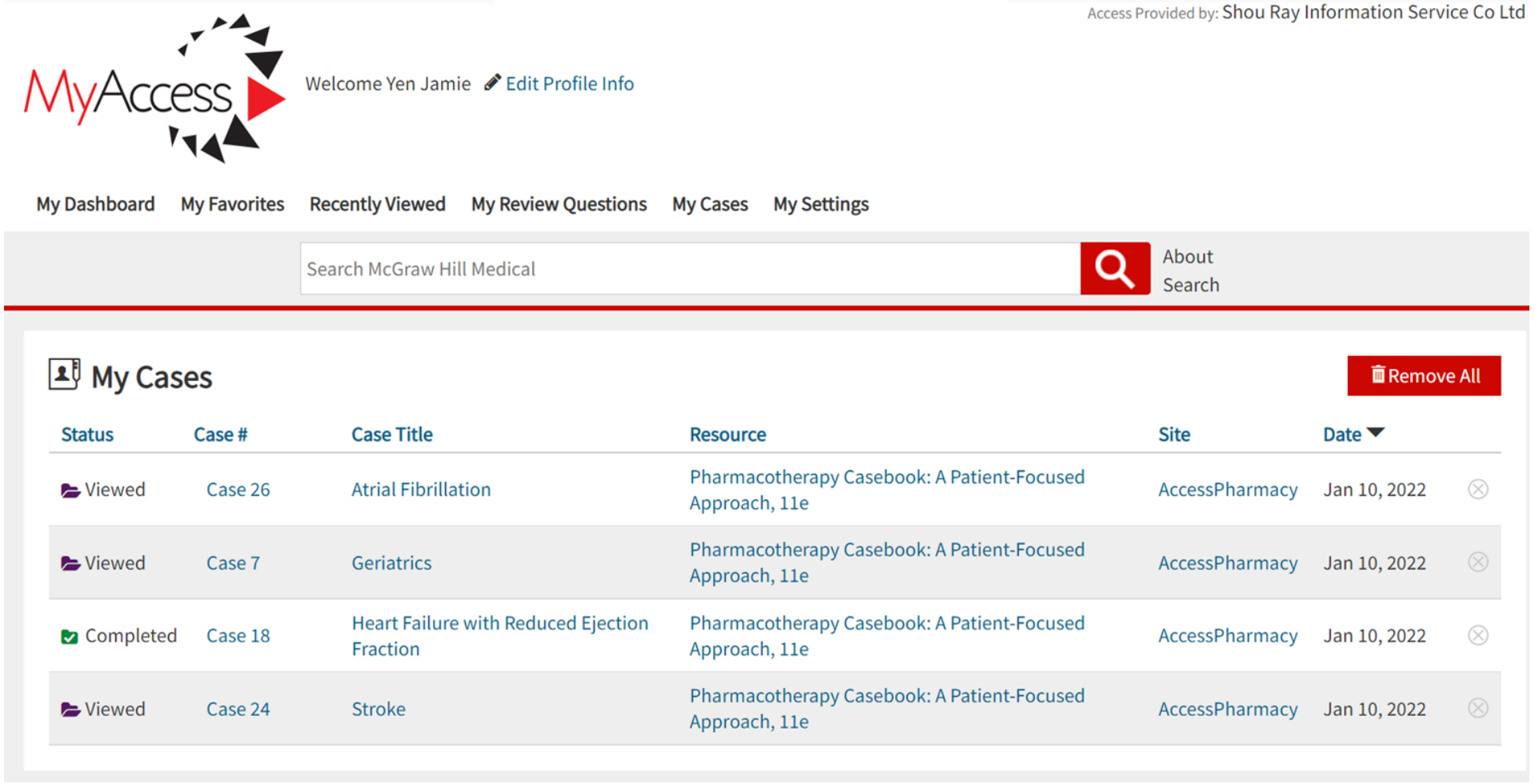
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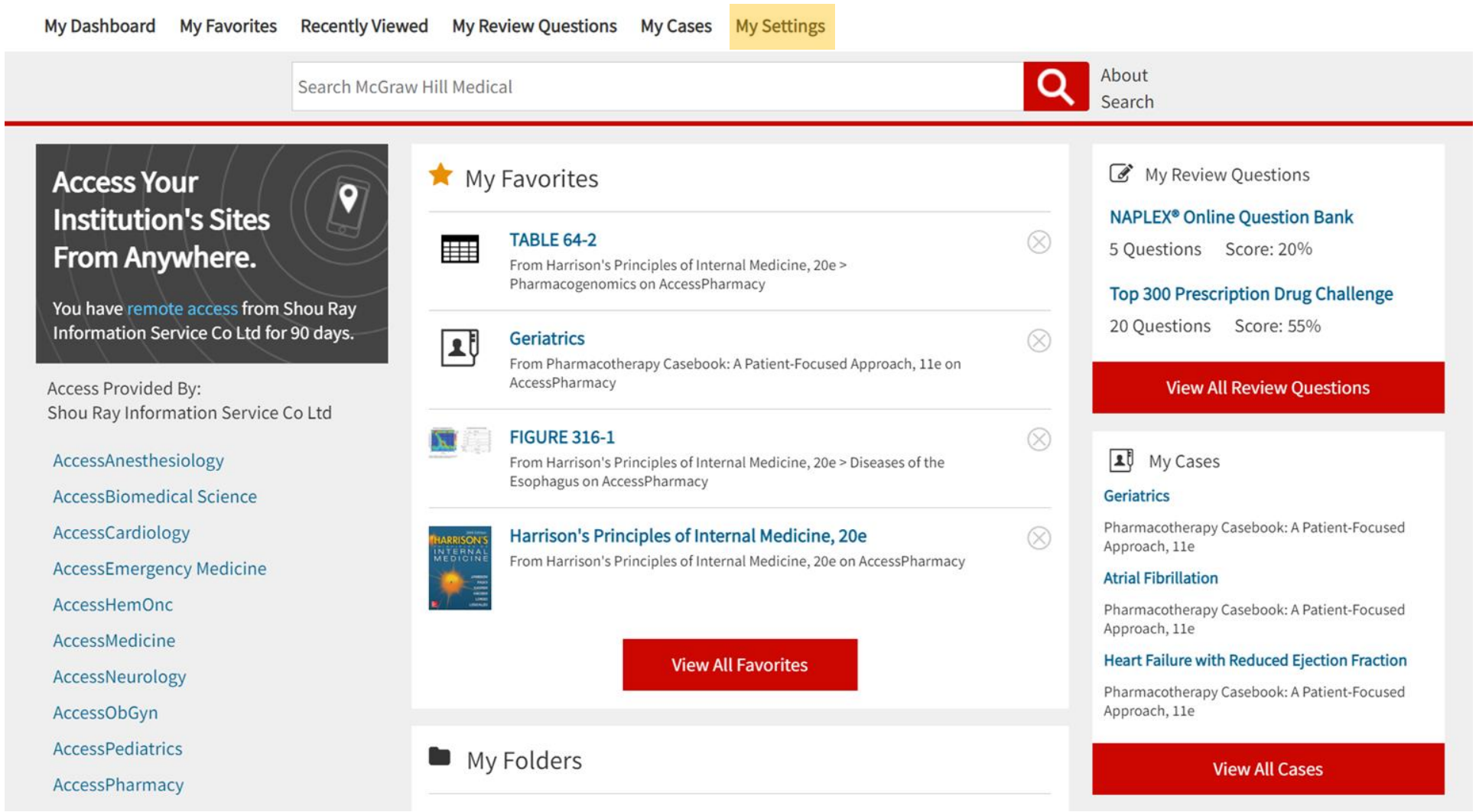

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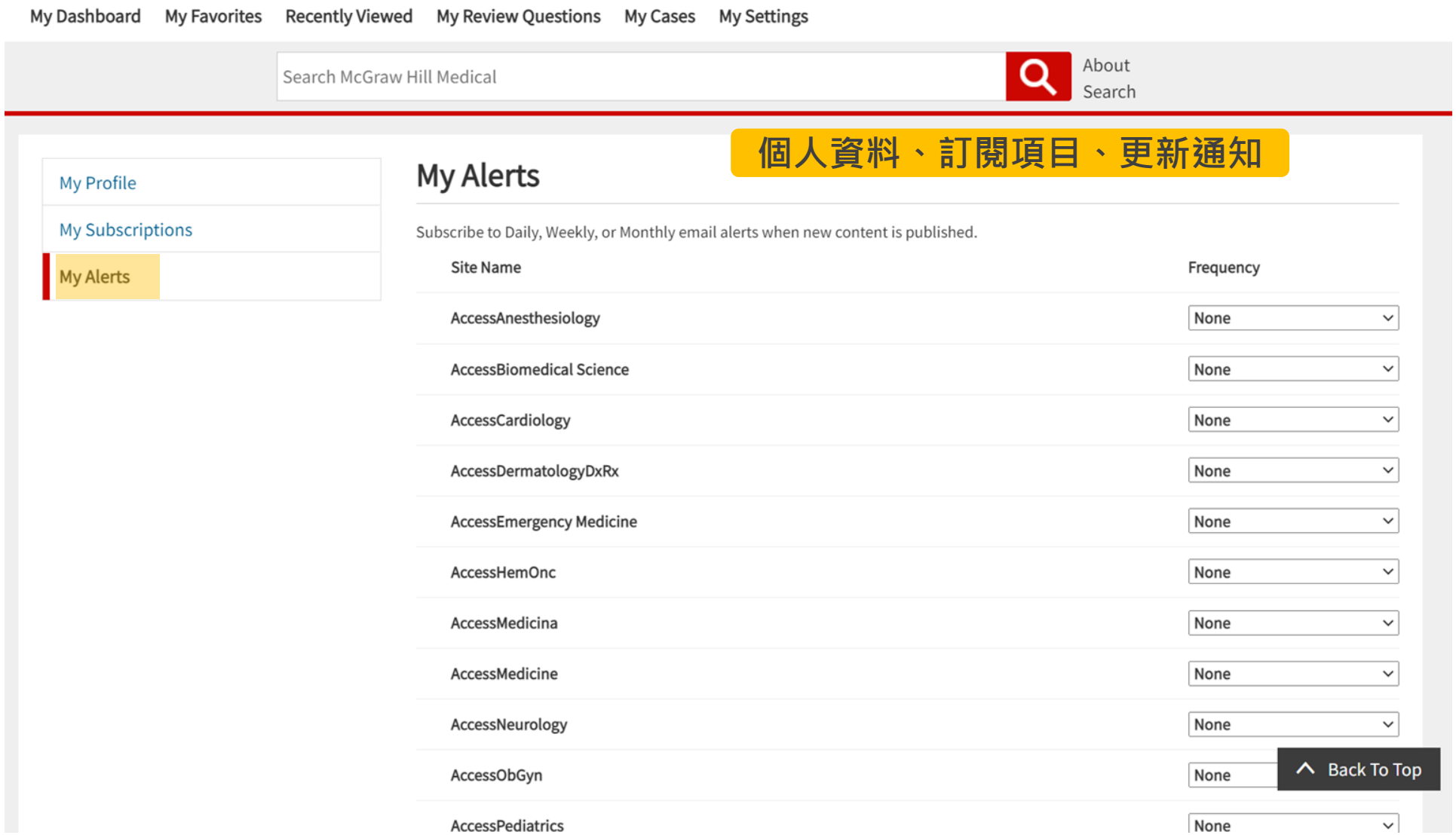
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Systemic Lupus Erythematosus

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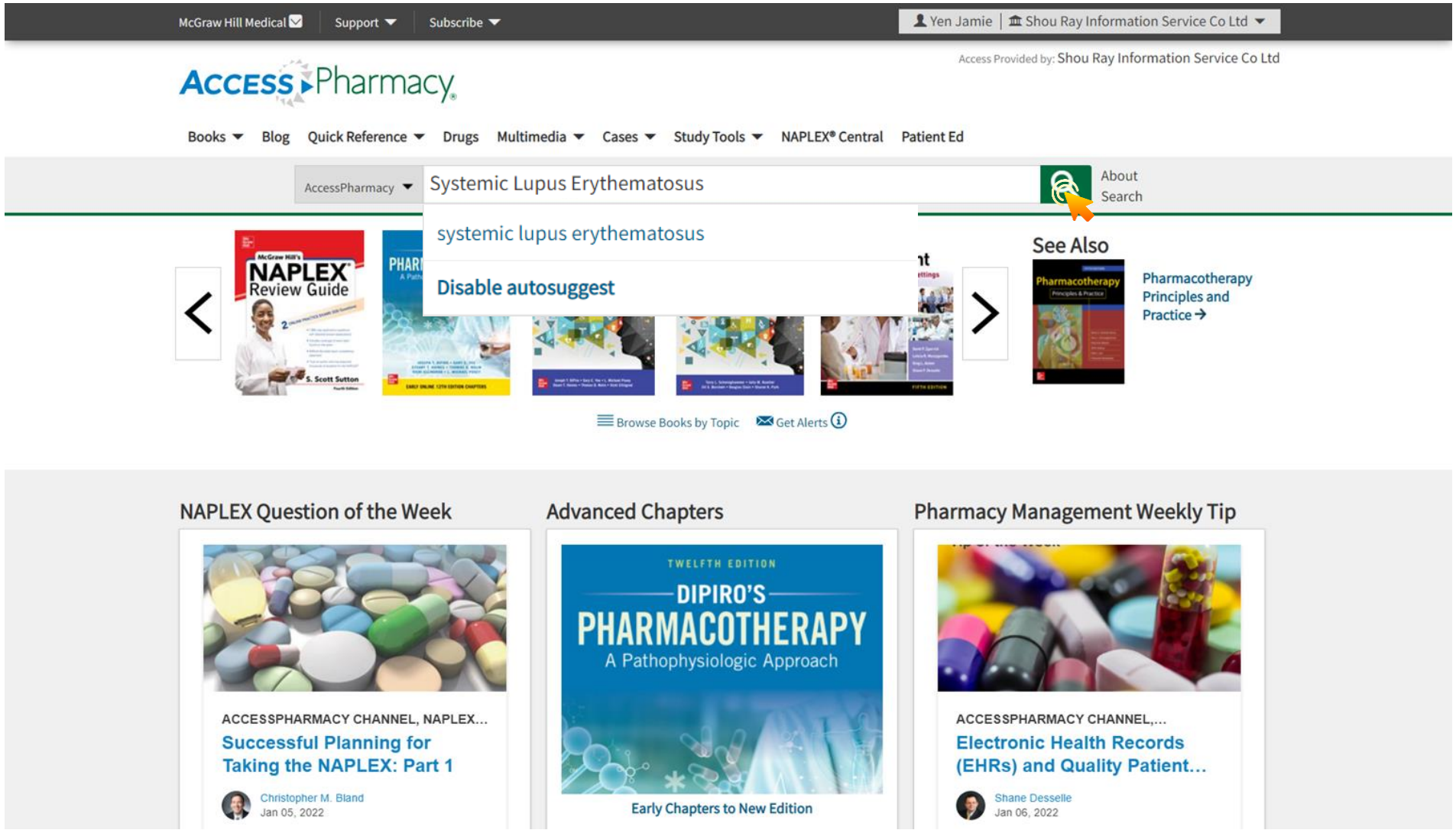
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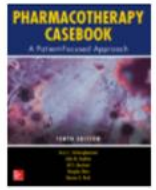

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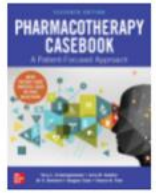

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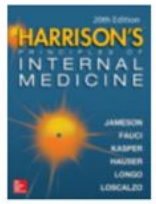
Book: Pharmacotherapy: A Pathophysiologic Approach, 11e

... tenderness. The study requires that participants fulfill the 2012 Systemic Lupus International Collaborating Clinics Classification Criteria for SLE. What additional finding would qualify this individual for the trial? Influenza 1. Which of the following individuals would be at the highest risk...



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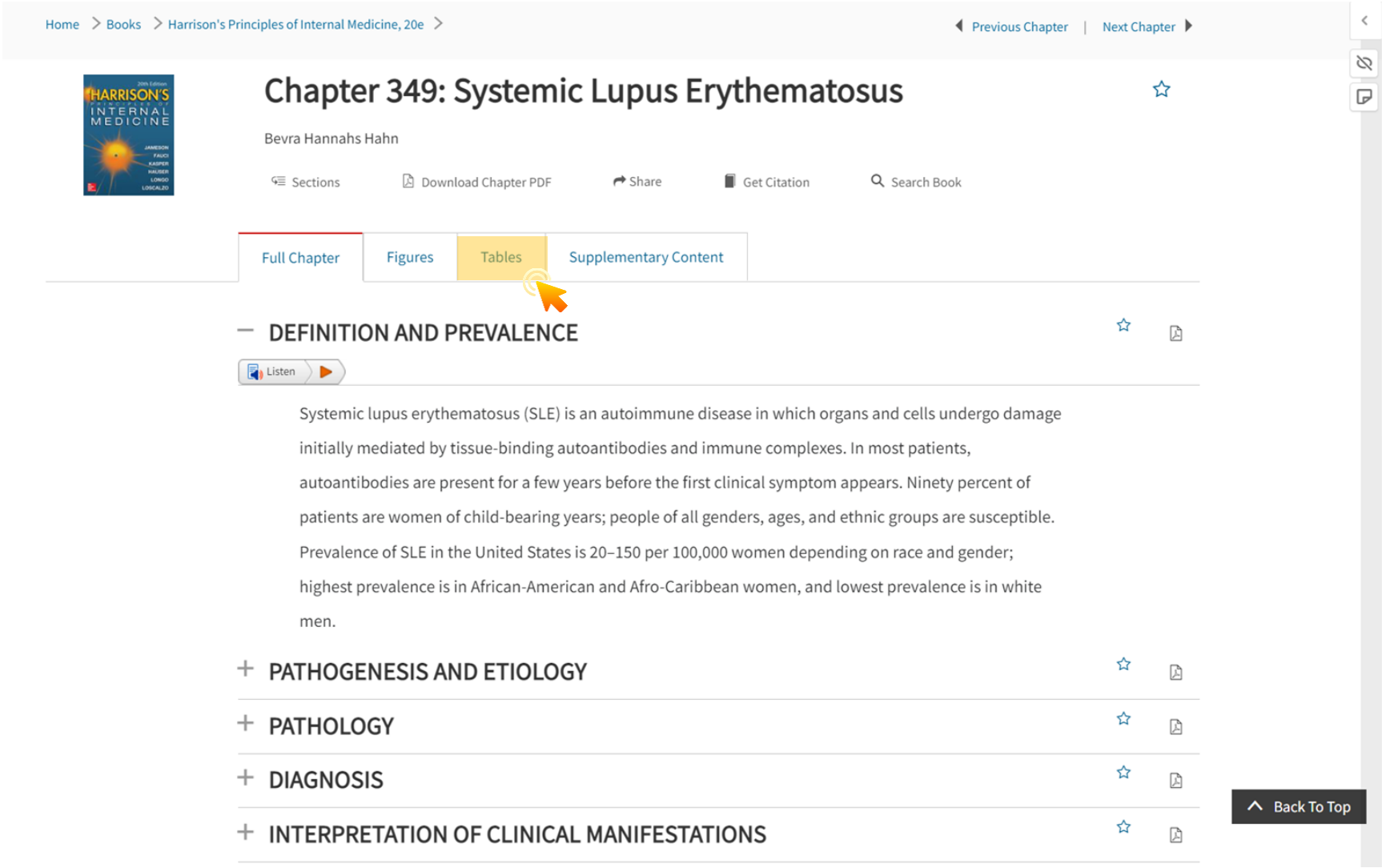
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Chapter 349: Systemic Lupus Erythematosus
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DEFINITION AND PREVALENCE
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Systemic lupus erythematosus (SLE) is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complexes. In most patients, autoantibodies are present for a few years before the first clinical symptom appears. Ninety percent of patients are women of child-bearing years; people of all genders, ages, and ethnic groups are susceptible. Prevalence of SLE in the United States is 20–150 per 100,000 women depending on race and gender; highest prevalence is in African-American and Afro-Caribbean women, and lowest prevalence is in white men.
PATHOGENESIS AND ETIOLOGY
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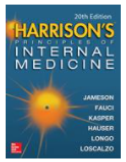

Chapter 349: Systemic Lupus Erythematosus

Bevra Hannahs Hahn

TABLE 349-1
Autoantibodies in Systemic Lupus Erythematosus (SLE)

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TABLE 349-2
Classification of Lupus Nephritis (International Society of Nephrology and Renal Pathology Society)

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TABLE 349-3
Systemic Lupus International Collaborating Clinic Criteria for Classification of Systemic Lupus Erythematosus

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國際合作臨床全身性紅斑性狼瘡分類標準

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J. Larry Jameson, Anthony S. Fauci, Dennis L. Kasper, Stephen L. Hauser, Dan L. Longo, Joseph Loscalzo

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TABLE 349-3

Systemic Lupus International Collaborating Clinic Criteria for Classification of Systemic Lupus Erythematosus

Clinical Manifestations	Immunologic Manifestations
Skin	ANA > reference negative value
Acute, subacute cutaneous LE (photosensitive, malar, maculopapular, bullous)	Anti-dsDNA >reference, if by ELISA 2x reference
Chronic cutaneous LE (discoid lupus, panniculitis, lichen planus-like, hypertrophic verrucous, chillblains)	Anti-Sm
Oral or nasal ulcers	Antiphospholipid (any of lupus anticoagulant, false-positive RPR, anti-cardiolipin, anti-β glycoprotein I
Nonscarring Alopecia	Low serum complement (C3, C4 or CH50)
Synovitis involving ≥2 joints	Positive direct Coombs test in absence of hemolytic anemia
Serositis (pleurisy, pericarditis)	
Renal	
Prot/Cr ≥0.5	
RBC casts	
Biopsy[a]	
Neurologic	
Seizures, psychosis, mononeuritis, myelitis, peripheral or cranial neuropathies, acute confusional state	
Hemolytic anemia	
Leukopenia (<4000/μL) or Lymphopenia (<1000/μL)	
Thrombocytopenia (<100,000/μL)	

[a] Renal biopsy read as systemic lupus qualifies for classification as SLE if any lupus autoantibodies are present, even if total criteria are fewer than 4.

Interpretation: Presence of any four criteria (must have at least 1 in each category) qualifies patient to be classified as having SLE with 93% specificity and 92% sensitivity. American College of Rheumatology is developing new criteria for SLE. For update see website *Rheumatology.org*.

Abbreviations: ANA, antinuclear antibody; Cr, creatinine; LE, lupus erythematosus; Prot, protein.

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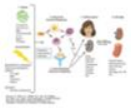

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IMMUNOLOGICALLY MEDIATED SKIN DISEASE

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(Figs. A4-58, A4-59, A4-60, A4-61, A4-62, A4-63, A4-64, A4-65, A4-66, A4-67, A4-68, A4-69) Immunologically mediated skin disease may be largely localized to skin and mucous membranes and manifest with blisters and erosions such as pemphigus, pemphigoid, and dermatitis herpetiformis. In diseases such as systemic lupus erythematosus, dermatomyositis, and vasculitis, skin manifestations are often only one element of a multisystem process.

FIGURE A4-58

Lupus erythematosus. ***A.*** Systemic lupus erythematosus, with prominent, scaly malar erythema. Involvement of other sun-exposed sites is also common. ***B.*** Acute lupus erythematosus on the upper chest, with brightly erythematous and slightly edematous coalescence of papules and plaques. *(B: Courtesy of Robert Swerlick, MD; with permission.)*

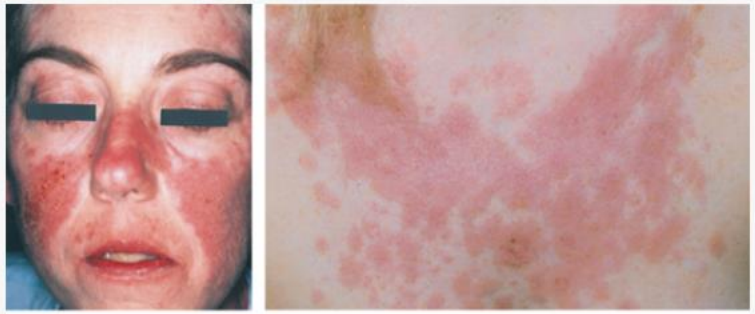

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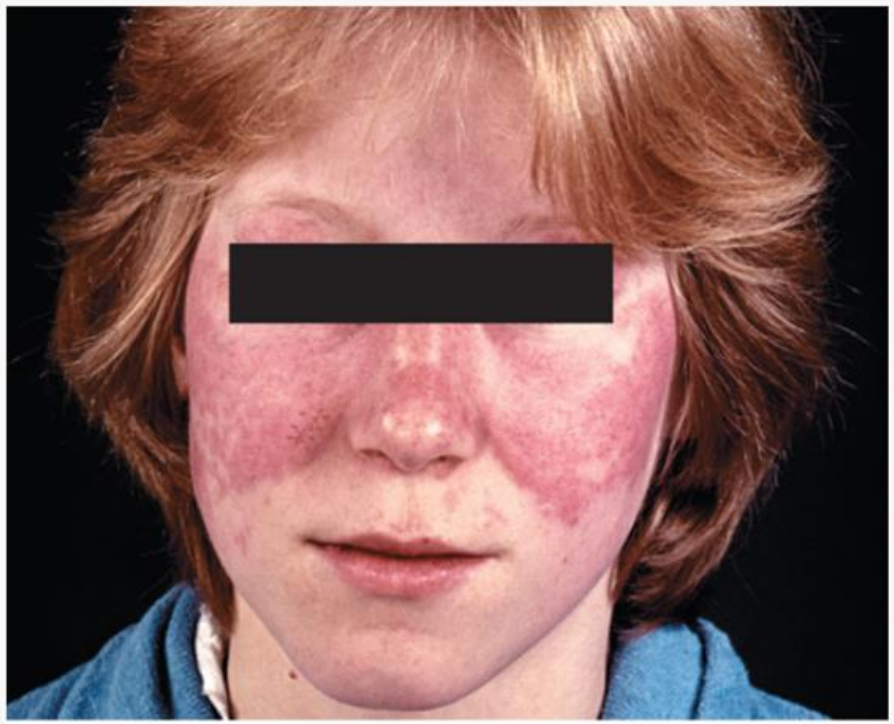
FIGURE A1-10

Systemic lupus erythematosus showing prominent malar erythema and minimal scaling. Involvement of other sun-exposed sites is also common. *(Reprinted from K Wolff, RA Johnson: Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology, 6th ed. New York, McGraw-Hill, 2009.)*



Chapter 87: Systemic Lupus Erythematosus

Beth H. Resman-Targoff

PATIENT CARE PROCESS

Listen

Patient Care Process for the Management of Systemic Lupus Erythematosus

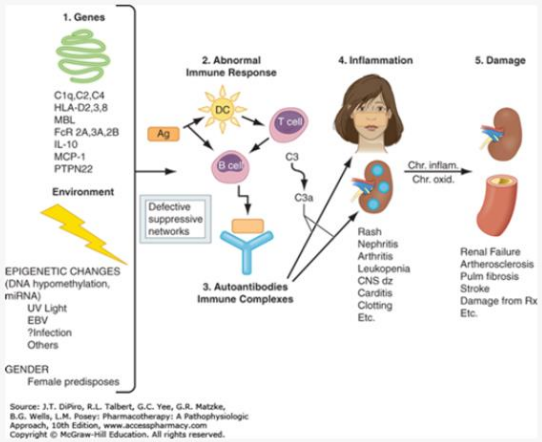
Collect

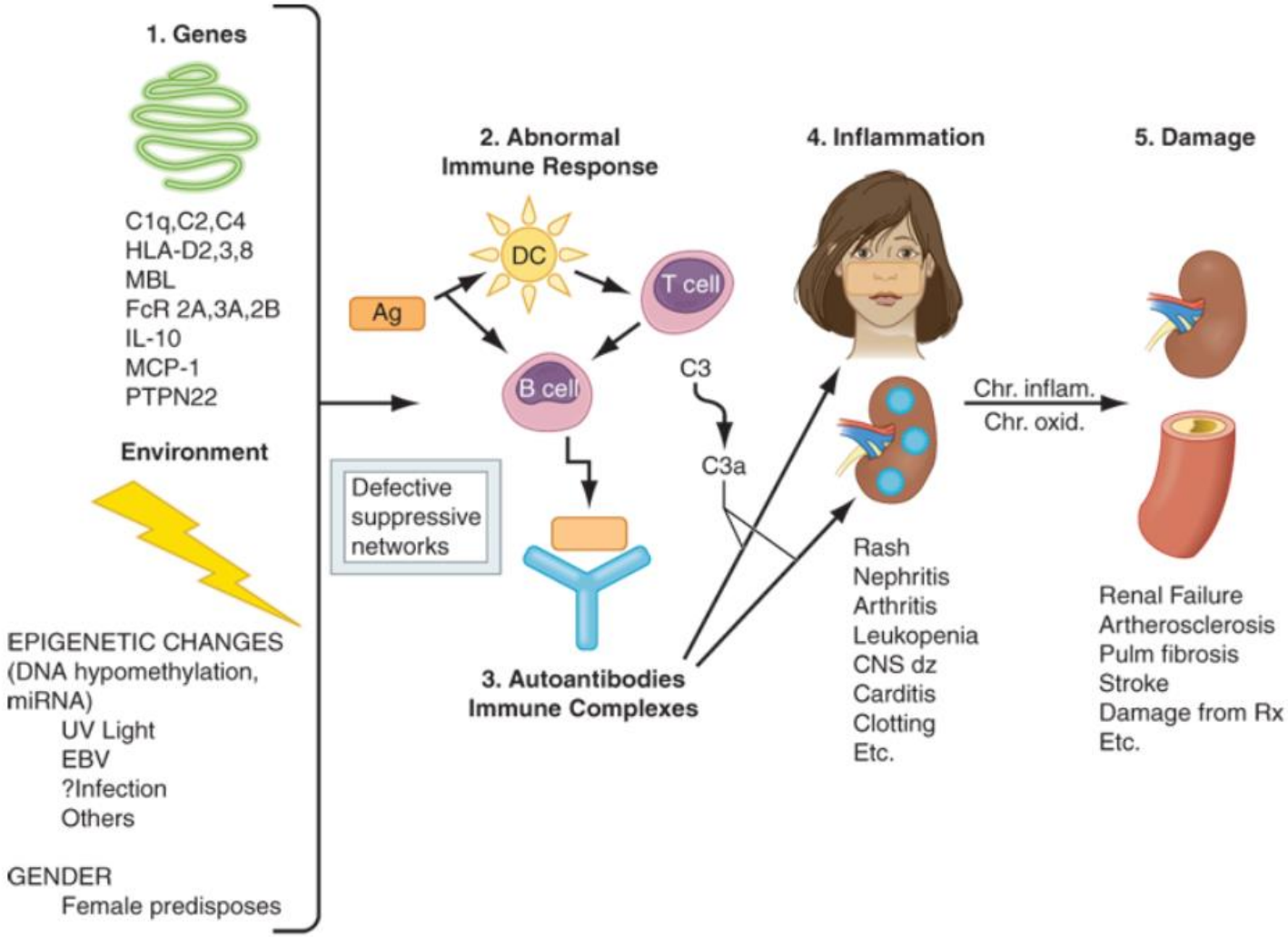
- Patient characteristics (e.g., age, race, sex, pregnancy status)
- Patient history (past medical, pregnancies and outcomes, symptoms, family, social—dietary habits, alcohol and tobacco use)
- Current medications and prior lupus medication use
- Immunization history
- Objective data (see Clinical Presentation and Table 87-1)
  - Blood pressure (BP), heart rate, height, weight, and BMI; other physical exam findings
  - Labs (metabolic panel, Scr, BUN, urinalysis, CBC, ANA, antiphospholipid antibodies, direct

PATHOPHYSIOLOGY

FIGURE 87-1

Pathogenesis of systemic lupus erythematosus (SLE). Genes confirmed in more than one genome-wide association analysis in northern European whites (several confirmed in Asians as well) as increasing susceptibility to SLE or lupus nephritis are listed (reviewed in SG Guerra et al. *Arthritis Res Ther* 2012;14:211). Gene-environment interactions (reviewed in KH Costenbader et al. *Autoimmune Rev* 2012;11:604) result in abnormal immune responses that generate pathogenic autoantibodies and immune complexes that deposit in tissue, activate complement, cause inflammation, and over time lead to irreversible organ damage (reviewed in GC Tsokos. *N Engl J Med* 2011;365:2110 and BH Hahn, in DJ Wallace, BH Hahn, eds. Dubois' Lupus Erythematosus and Related Syndromes, 8th ed. New York, Elsevier, 2013). Ag, antigen; C1q, complement system; C3, complement component; CNS, central nervous system; DC, dendritic cell; EBV, Epstein-Barr virus; HLA, human leukocyte antigen; FcR, immunoglobulin Fc-binding receptor; IL, interleukin; MCP, monocyte chemotactic protein; PTPN, phosphotyrosine phosphatase; UV, ultraviolet. *(Reproduced with permission from Hahn BH. Systemic lupus erythematosus. In: Kasper DL, Fauci AS, Hauser SL, et al., eds. Harrison's Principles of Internal Medicine. 19th ed. 2015.)*





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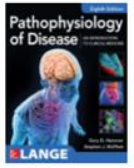

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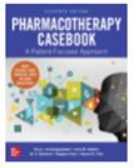

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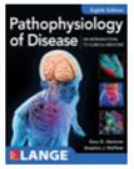

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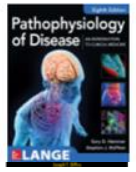

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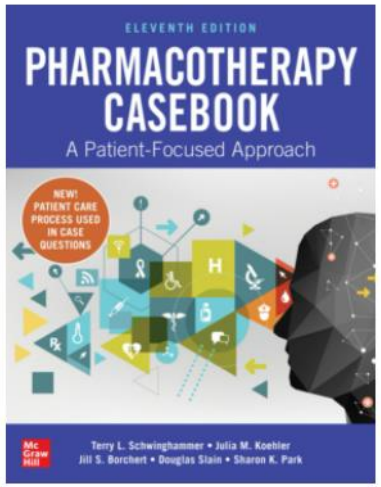

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After completing this case study, the reader should be able to:

- Discuss the clinical presentation of SLE, including its complications.
- Design appropriate therapy for the treatment of SLE and the complications of antiphospholipid syndrome (APS) and iron deficiency anemia.
- Construct a monitoring plan for SLE, including disease activity, drug efficacy, and drug toxicity.
- Recommend appropriate therapy for the treatment of SLE during pregnancy.

Next: Patient Presentation

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