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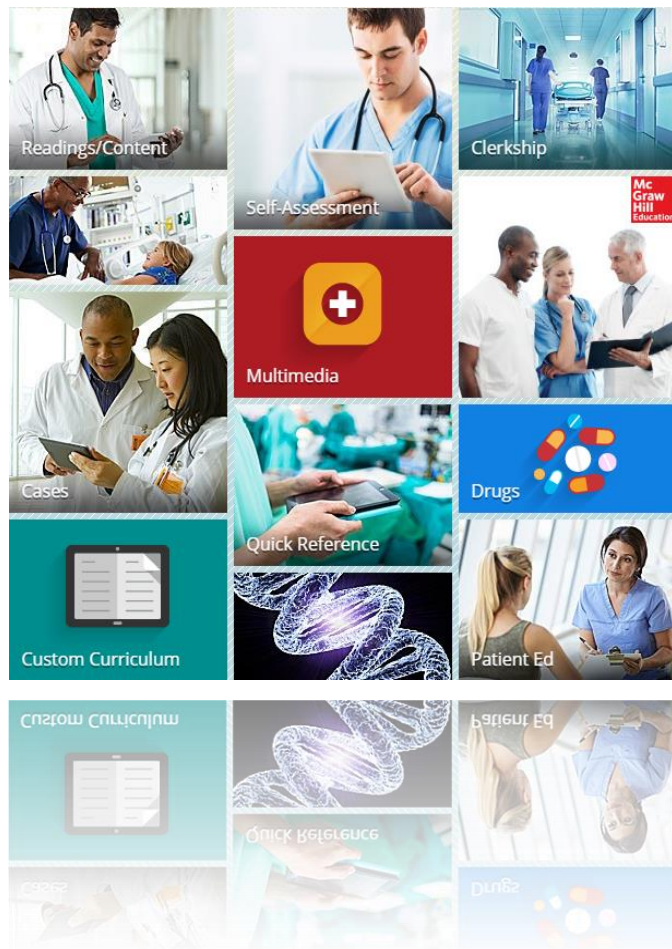
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- 為滿足藥學教育更新的需要而設計。
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 - 持續教育專業知識
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- 收錄優質的參考書：從基礎科學到臨床醫學、醫學法律等等，其中也涵蓋**國考用書**。
- 提供**多種測驗及自我評量**。
- 整合藥物資料庫提供臨床相關資訊。
- 支援追蹤技術，將案例研究、照護計畫、NAPLEX 評論整合提供關鍵資訊。

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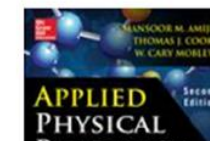
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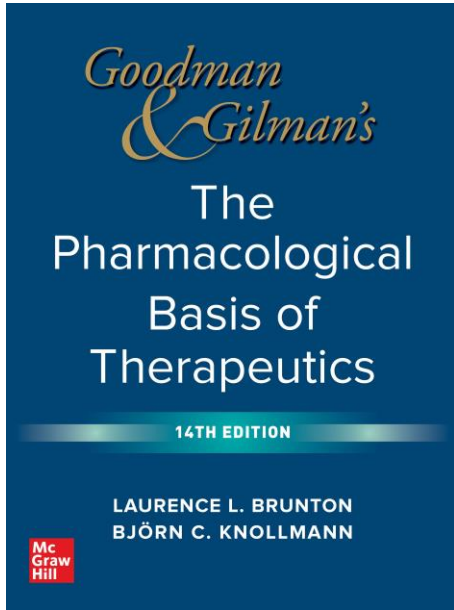


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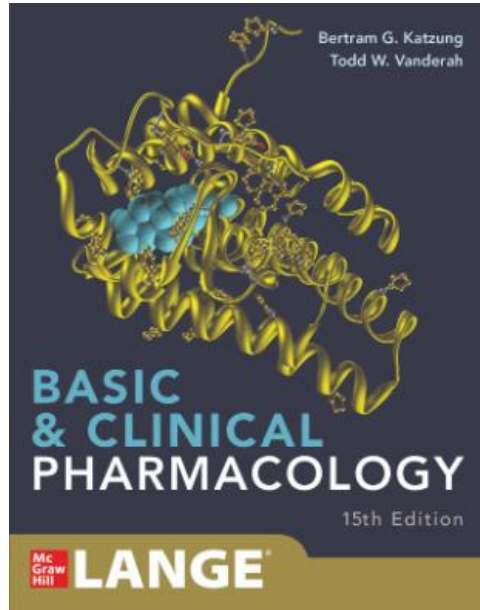
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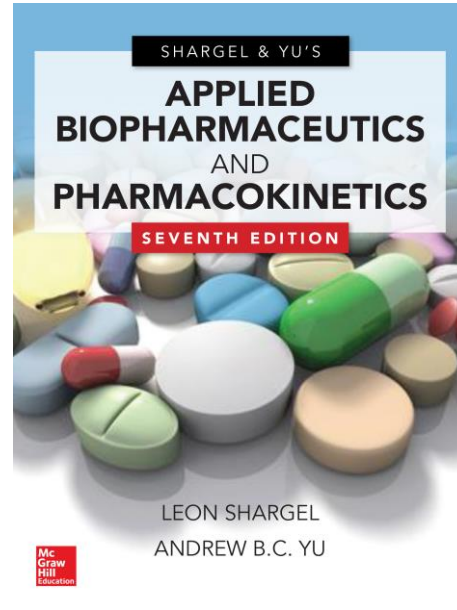
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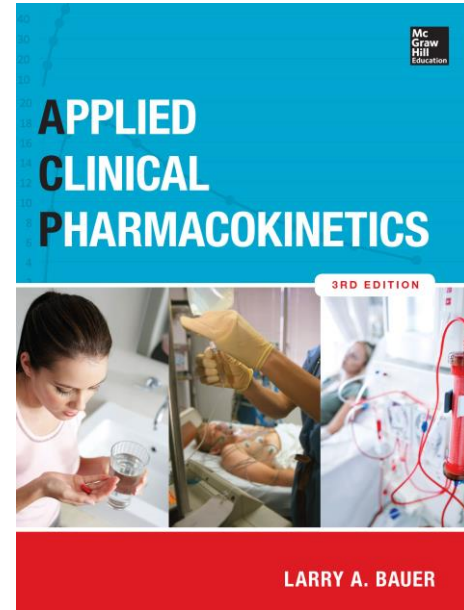
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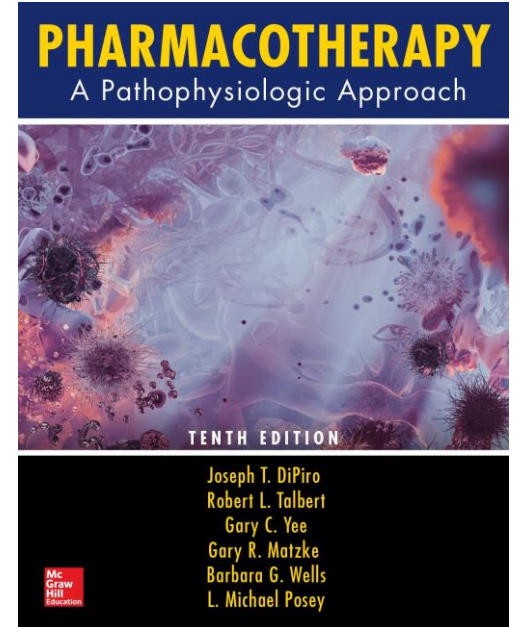
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Nicholas D. Franz, PharmD, Sarah Nordbeck, PharmD, BCNSP, Courtney Doellner, PharmD, BCPPS, Melissa R. Pleva, PharmD, BCPS, BCNSP, BCCCP



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Topics in Evidence-Based Pharmacy Practice is a continually updated collection of features and editorials that review, examine, and comment on the overload of information in the pharmacy field. Features are reviews of clinical trials, studies, and guidelines that examine the validity of approach, methodology, results, and recommendations, and make recommendations that can affect decision making. Editorials allow leading and emerging minds in the field express their opinion on the ever-changing world of pharmacy practice. [Read more...](#)

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Basic & Clinical Pharmacology, 15e

Bertram G. Katzung, Todd W. Vanderah

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Chapter 4: Drug Biotransformation

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Chapter 2: Drug Receptors & Pharmacodynamics

Mark von Zastrow

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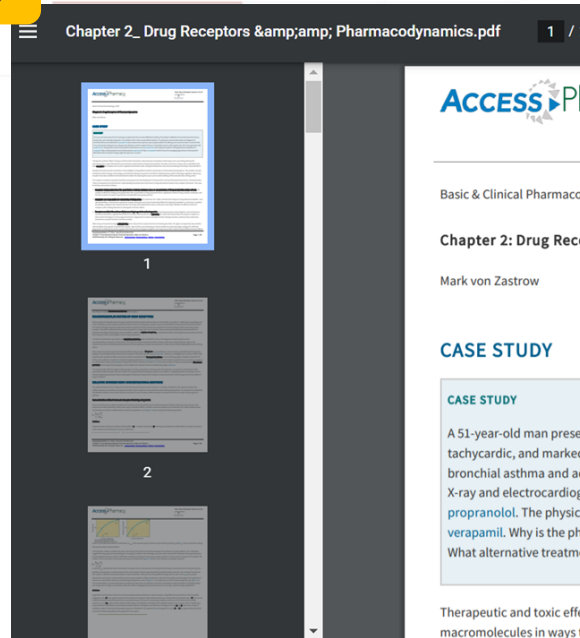
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CASE STUDY

A 51-year-old man presents to the emergency department due to acute difficulty breathing. The patient is afebrile and normotensive but anxious, tachycardic, and markedly tachypneic. Auscultation of the chest reveals diffuse wheezes. The physician provisionally makes the diagnosis of bronchial asthma and administers **epinephrine** by intramuscular injection, improving the patient's breathing over several minutes. A normal chest X-ray and electrocardiogram are subsequently obtained, and the medical history is remarkable only for mild hypertension that is being treated with **propranolol**. The physician instructs the patient to discontinue use of **propranolol**, and changes the patient's antihypertensive medication to **verapamil**. Why is the physician correct to discontinue **propranolol**? Why is **verapamil** a better choice for managing hypertension in this patient? What alternative treatment change might the physician consider?

Therapeutic and toxic effects of drugs result from their interactions with molecules in the patient. Most



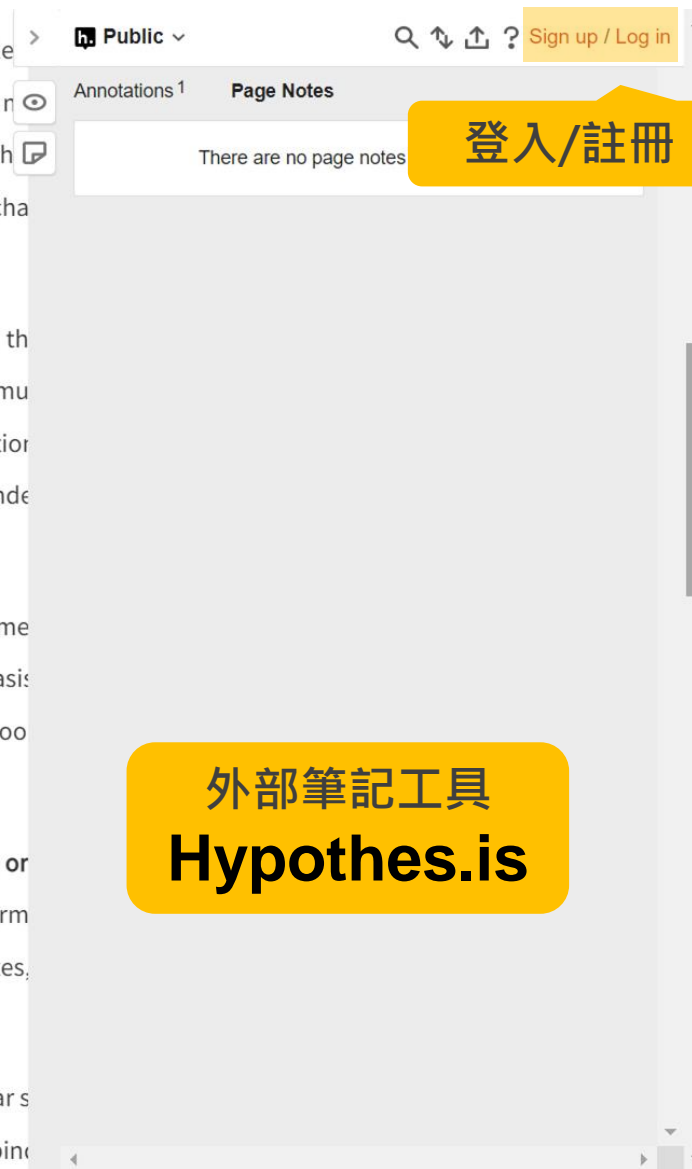
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Therapeutic and toxic effects of drugs result from their interactions with molecules. Drugs act by associating with specific macromolecules in ways that alter the normal or biophysical activities. This idea, more than a century old, is embodied in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

Receptors have become the central focus of investigation of drug effects and their pharmacodynamics. The receptor concept, extended to endocrinology, immunology, and cell biology, has proved essential for explaining many aspects of biologic regulation. Many receptors have been isolated and characterized in detail, thus opening the way to precise understanding of the basis of drug action.

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

1. **Receptors largely determine the quantitative relations between dose or concentration and pharmacologic effects.** The receptor's affinity for binding a drug determines the amount of drug required to form a significant number of drug-receptor complexes. The number of receptors may limit the maximal effect a drug may produce.
2. **Receptors are responsible for selectivity of drug action.** The molecular structure and charge of a drug determine whether—and with what affinity—it will bind to a receptor.





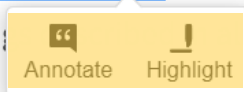
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better choice for managing hypertension in this patient? What alternative treatments should the physician consider?

Therapeutic and toxic effects of drugs result from their interactions with molecules. Most drugs act by associating with specific macromolecules in ways that alter the molecular or biophysical activities. This idea, more than a century old, is embodied in the concept of a component of a cell or organism that interacts with a drug and initiates the chain of events that leads to the drug's observed effects.

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

The receptor concept has important practical consequences for the development of new drugs and for the making of therapeutic decisions in clinical practice. These consequences form the basis for the actions and clinical uses of drugs. The following summarizes the consequences of the receptor concept as summarized as follows:



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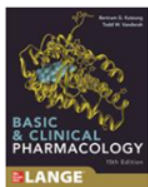
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Chapter 2: Drug Receptors & Pharmacodynamics



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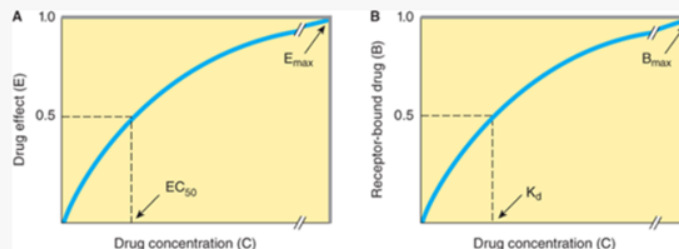
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FIGURE 2-1

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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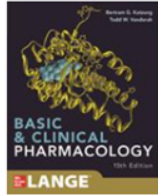
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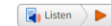


TABLE 2-1
G proteins and their receptors and effectors.

G Protein	Receptors for	Effector/Signaling Pathway
G _s	β-Adrenergic amines, histamine, serotonin, glucagon, and many other hormones	↑ Adenylyl cyclase → ↑ cAMP
G ₁₁ , G ₁₂ , G ₁₃	α ₂ -Adrenergic amines, acetylcholine (muscarinic), opioids, serotonin, and many others	Several, including: ↓ Adenylyl cyclase → ↓ cAMP Open cardiac K ⁺ channels → ↓ heart rate
G _{olf}	Odorants (olfactory epithelium)	↑ Adenylyl cyclase → ↑ cAMP
G _o	Neurotransmitters in brain (not yet specifically identified)	Not yet clear
G _q	Acetylcholine (muscarinic), bombesin, serotonin (5-HT ₂), and many others	↑ Phospholipase C → ↑ IP ₃ , diacylglycerol, cytoplasmic Ca ²⁺
G _{t1} , G _{t2}	Photons (rhodopsin and color opsins in retinal rod and cone cells)	↑ cGMP phosphodiesterase → ↓ cGMP (phototransduction)

cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; IP₃, inositol-1,4,5-trisphosphate.

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Alkalosis, Respiratory

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Clinically important safety information regarding human medical products

December 30, 2021 at 12:00 AM

[Taro Pharmaceuticals U.S.A. Issues Voluntary Nationwide Recall of Clobetasol Propionate Ointment USP, 0.05%, 60 g Tubes, Lot AC13786 Due to Microbial Contamination](#)

Taro Pharmaceuticals U.S.A., Inc. (“Taro” or the “Company”) is voluntarily recalling one (1) lot of Clobetasol Propionate Ointment USP, 0.05% packaged in 60 g tubes, to the consumer level. This recall ONLY applies to tubes labeled with “Lot AC13786” and “Exp Dec 2022”. No other lots of this product

December 28, 2021 at 12:00 AM

[Getinge/Datascope/Maquet Recalls Cardiosave Hybrid and Cardiosave Rescue Intra-Aortic Balloon Pump \(IABP\) Due to Reports of Fluid Leaks](#)

Getinge/Datascope/Maquet recalls the Cardiosave IABP after complaints of fluid leaks causing the system to shut down.

December 28, 2021 at 12:00 AM

[Viona Pharmaceuticals Inc., Issues Voluntary Nationwide Recall of Metformin HCl Extended-Release Tablets, USP 750 mg, Due to the Detection of N-Nitrosodimethylamine \(NDMA\) Impurity](#)

Cranford, New Jersey, Viona Pharmaceuticals Inc., is voluntarily recalling thirty three (33) lots of Metformin Hydrochloride Extended-Release Tablets, USP 750 mg to the retail level. Reason for the recall is an Out of specification result observed for the said product, Lot number M008132, “N-nitroso

December 27, 2021 at 12:00 AM

[Padagis Issues Voluntary Nationwide Recall](#)

December 27, 2021 – Padagis US LLC announced today it has issued a voluntary nationwide recall to the consumer/user level of the lots of Nitroglycerin Lingual Spray listed in the table below. Out of an abundance of caution, this product is being recalled from the market



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A-200 Lice Treatment Kit [OTC]	A-200 Maximum Strength [OTC]
A-25 [OTC]	A3 (Neuroblastoma)
A-AVD (Hodgkin)	Abacavir
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A3 (Neuroblastoma)

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- Ascriptin Regular Strength [OTC]

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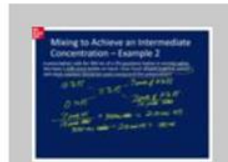
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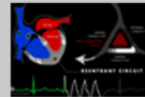
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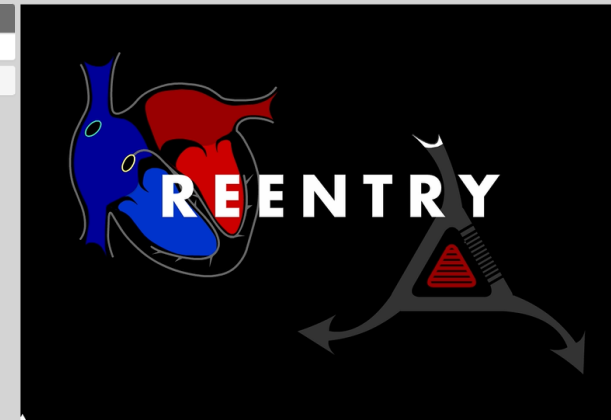
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Anti-Arrhythmic Drugs

Reentry

Ventricular Micro-reentry

Anti-Arrhythmic Drugs



00:00 / 00:48

The most common cause of arrhythmias is a process known as reentry. Reentrant circuits can form in any region of the heart, and can disrupt normal sinus rhythm and conduction. This animation will illustrate the conditions necessary to form a reentrant circuit, and how antiarrhythmic drugs are used to block reentrant circuits. When the heart is in normal sinus rhythm, impulses form in the sinus (SA) node and propagate through the atria to the atrio-ventricular (AV) node. Impulse conduction through the AV node is slow to allow the ventricles time to fill. Subsequent impulses

Anti-Arrhythmic Drugs
the con
circuit
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Author
Cowan

Atrial Micro-reentry
focuse
affect a
drugs u
atria.
Author(s): Donald K. Blumenthal, PhD, and Derek
Cowan

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Pharmacotherapy Casebook: A Patient-Focused Approach, 11e

Author(s): Terry L. Schwinghammer; Julia M. Koehler; Jill S. Borchert; Douglas Slain; Sharon K. Park

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- Acute Coronary Syndrome: St-Elevation Myocardial Infarction
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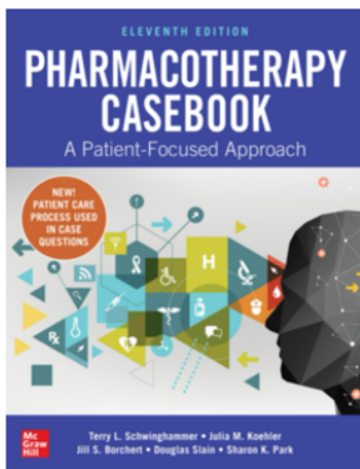
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書籍資訊

Atrial Fibrillation



Authors: Virginia H. Fleming

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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.

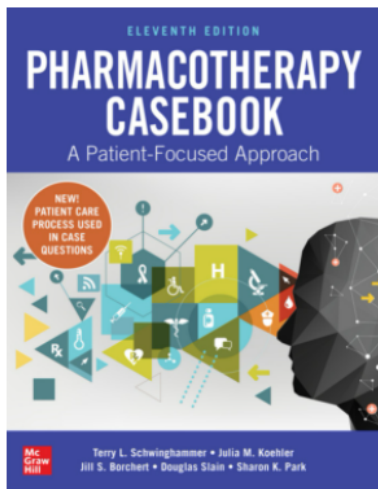
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主訴

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).

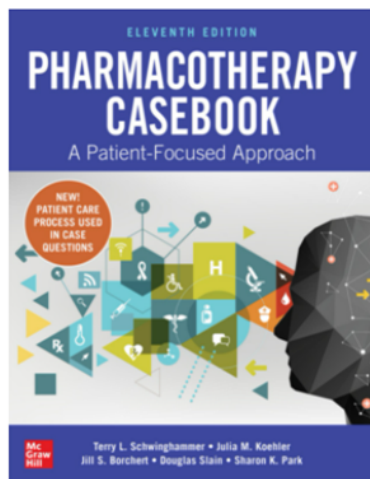
FIGURE 26-1.

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.



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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/rEF?

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

[Save Answers](#)

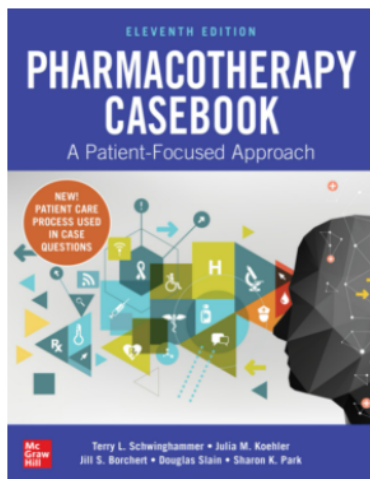
Assess the Information

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?



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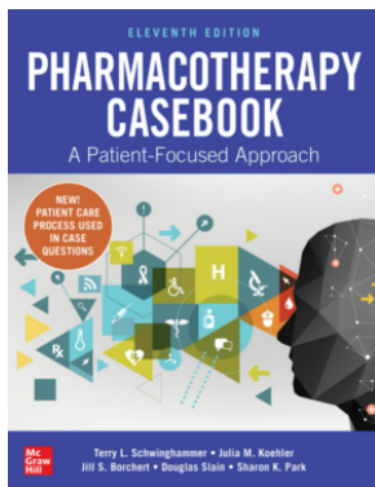
In treating AF with concomitant systolic heart failure, lenient ventricular rate control (<110 bpm) plus anticoagulation is a viable treatment option over maintaining NSR with antiarrhythmic therapy.

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ALBENDAZOLE: Albenza, Various <i>[Anthelmintic]</i> <i>[Anti-infective Agents]</i>	ALBUTEROL: ProAir HFA, Proventil HFA, Ventolin HFA, ProAir Respiclick, Various <i>[Pulmonology]</i> <i>[Respiratory Agents]</i>	ALENDRONATE: Fosamax, Binosto, Various <i>[Womens Health]</i> <i>[Endocrine Agents]</i>	ALLOPURINOL: Zyloprim, Various <i>[Misc]</i> <i>[Miscellaneous Agents]</i>
ALPRAZOLAM: Xanax, Various <i>[Neurology]</i> <i>[Neurologic Agents]</i>	AMIODARONE: Cordarone, Pacerone, Various <i>[Cardiology]</i> <i>[Cardiovascular Agents]</i>	AMITRIPTYLINE: Elavil, Various <i>[Mental Health]</i> <i>[Neurologic Agents]</i>	AMLODIPINE: Norvasc, Various <i>[Cardiology]</i> <i>[Cardiovascular Agents]</i>

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ALLOPURINOL: Zyloprim, Various

ALPRAZOLAM: Xanax, Various

AMIODARONE: Cordarone, Pacerone, Various

AMITRIPTYLINE: Elavil, Various

AMLODIPINE: Norvasc, Various

AMOXICILLIN: Amoxil, Moxatag, Various

AMOXICILLIN/CLAVULANATE: Augmentin, Various

ANASTROZOLE: Arimidex, Various

APIXABAN: Eliquis

AMIODARONE: Cordarone, Pacerone, Various

Drug Interactions: Amiodarone

Typical Agents	Mechanism	Clinical Management
Class I and III antiarrhythmics, other drugs that prolong the QT interval	Increased risk of cardiotoxicity	Avoid concurrent use
CYP3A4/5 and 2C8 inhibitors	Decreased metabolism of amiodarone and additional toxicity	Avoid concurrent use
CYP3A4/5 and 2C8 inducers	Increased metabolism of amiodarone and decreased efficacy	Avoid concurrent use, monitor HR and rhythm, ECG
CYP2A6, 2C9, 2D6, 3A4/5 and P-glycoprotein substrates	Decreased metabolism or transport of substrates and additional toxicity	Monitor and consider dose adjustments of substrates
Beta-blockers and calcium channel blockers	Increased risk of hypotension, bradycardia, or cardiac arrest	Use with caution, monitor cardiac function closely

Adverse Reactions: Amiodarone

Common (>10%)	Less Common (1-10%)	Rare but Serious (<1%)
Nausea, vomiting, phospholipidemia	Alopecia, bradyarrhythmia, hypotension, increased LFTs, peripheral neuropathy	Blindness, hepatotoxicity, pulmonary fibrosis, Stevens-Johnson syndrome

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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

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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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Challenge

Tier 1 (Brand/Generic and Classifications)

00:00:04:22

You have 90 minutes to complete this test

計時

Question 1 of 20

What is the classification of Augmentin?

- A Antibiotic, sulfonamide
- B Antibiotic, fluoroquinolone
- C Antibiotic, macrolide
- D Antibiotic, beta lactam

Submit & View Next Question

下一題

End test and return to Top 300 Prescription Drug Challenge Review Questions

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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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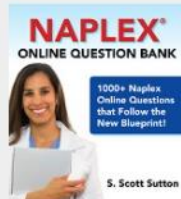
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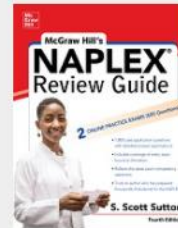
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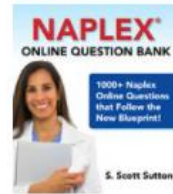


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Generate a custom quiz from the topics below.

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of 121 available 1.1.2: Obtain, interpret, assess, and/or evaluate patient medical records

of 49 available 1.1.3: Obtain, interpret, assess, and/or evaluate results from instruments and screening strategies used to assess patients

of 125 available 1.1.4: Obtain, interpret, assess, and/or evaluate laboratory and diagnostic findings



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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 the past 24 hours. He also reports right upper quadrant pain. He does not report consuming a 12 pack of alcohol in the past 24 hours. On examination, which of the following is the most likely cause of the patient's symptoms?

- A Acetaminophen level should be checked. Antidote therapy should be initiated.
- B Antidote therapy should be initiated.
- C Acetaminophen level should be checked.
- D Activated charcoal should be administered for antidote therapy.

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

Submit & View Answer

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.

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阿茲海默症 (Alzheimer's Disease)

重點

- 阿茲海默症通常會導致思考、記憶、推論和計畫的能力逐漸喪失。
- 阿茲海默症無法治癒。治療的目標為控制症狀並儘可能改善生活品質。
- 療法可能包含治療其他疾病、攝取健康飲食、定期進行體能活動以及藥物治療。

什麼是阿茲海默症？

阿茲海默症 (AD) 是一種隨著時間惡化的失智症。阿茲海默症通常會導致思考、記憶、推論和計畫的能力逐漸喪失。此疾病會影響腦細胞並逐漸造成記憶和思考能力的喪失。隨著時間過去，也可能會導致語言表達、步行、記憶、情緒控制和決策能力喪失。

阿茲海默症目前無法治癒。腦部功能將持續衰退直到病患死亡。從產生記憶問題開始，阿茲海默症可能會在 5 年至 15 年後導致病患死亡。

致病因是什麼？

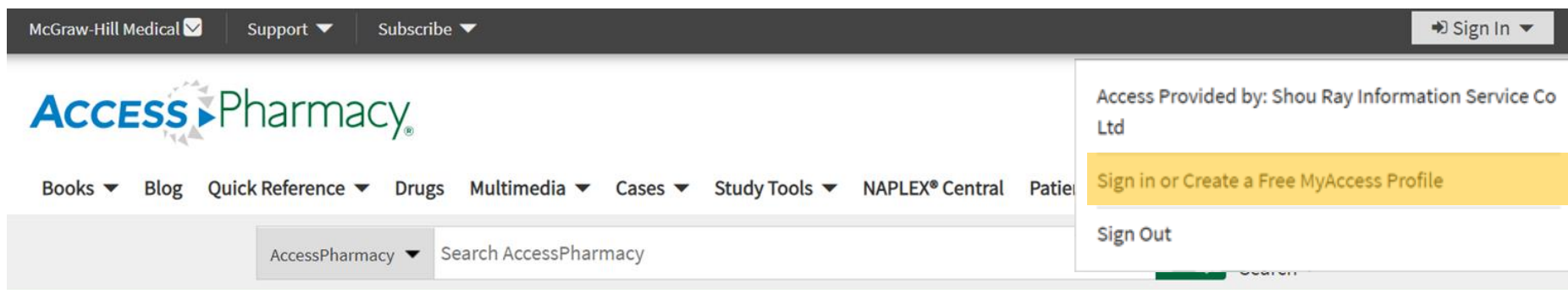
目前尚未完全清楚阿茲海默症的確切致病因。可能與許多因素有關，例如基因、環境或是生活方式等。當您罹患阿茲海默症，您的大腦將會發生一些變化。異常蛋白質碎片和群簇開始在腦中形成。腦中的某些神經細胞將停止作用並死亡。這會導致部分大腦將開始萎縮。尚未確定這些變化是阿茲海默症的成因或結果。

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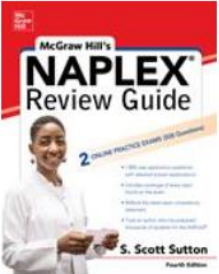
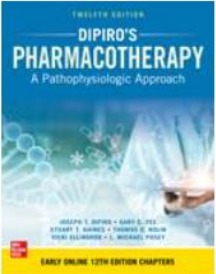
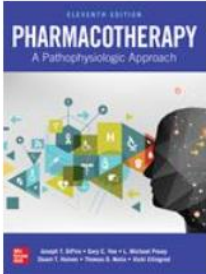
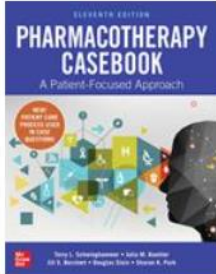

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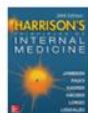
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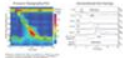
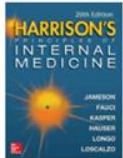


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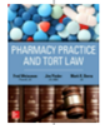
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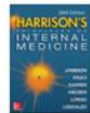
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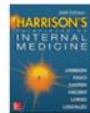
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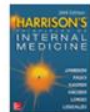
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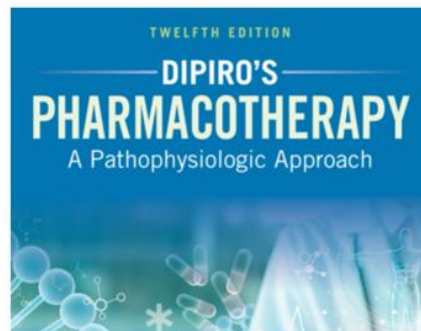
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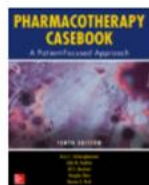
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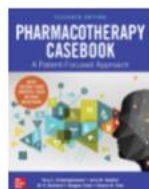
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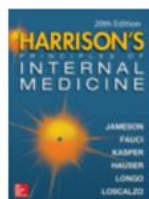


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... tenderness. The study requires that participants fulfill the 2012 **Systemic Lupus** International Collaborating Clinics Classification Criteria for SLE. What additional finding would qualify this individual for the trial? Influenza 1. Which of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



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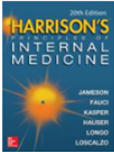
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Chapter 349: Systemic Lupus Erythematosus ☆

Bevra Hannahs Hahn


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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.

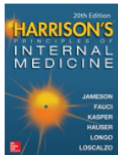
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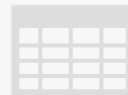
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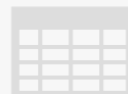
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TABLE 349-2

Classification of Lupus Nephritis (International Society of Nephrology and Renal Pathology Society)



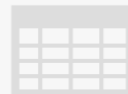
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TABLE 349-3

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



TABLE 349-3

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

Clinical Manifestations	Immunologic Manifestations
Skin Acute, subacute cutaneous LE (photosensitive, malar, maculopapular, bullous) Chronic cutaneous LE (discoid lupus, panniculitis, lichen planus-like, hypertrophic verrucous, chillblains) Oral or nasal ulcers Nonscarring Alopecia Synovitis involving ≥ 2 joints 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 ($<4000/\mu\text{L}$) or Lymphopenia ($<1000/\mu\text{L}$) Thrombocytopenia ($<100,000/\mu\text{L}$)	ANA $>$ reference negative value Anti-dsDNA $>$ reference, if by ELISA 2x reference Anti-Sm Antiphospholipid (any of lupus anticoagulant, false-positive RPR, anti-cardiolipin, anti- β glycoprotein I) Low serum complement (C3, C4 or CH50) Positive direct Coombs test in absence of hemolytic anemia

^aRenal 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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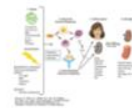
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(Figs. A4-58, A4-59, A4-60, A4-61, A4-62, A4-63, A4-64, A4-65, A4-66, A4-67, A4-68, A4-69) Immunologically mediated skin disease may be largely localized to skin and mucous membranes and manifest with blisters and erosions such as pemphigus, pemphigoid, 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.*)



A

B

Source: J.L. Jameson, A.S. Fauci, D.L. Kasper, S.L. Hauser, D.L. Longo, J. Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition
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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.*)



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Chapter 87: Systemic Lupus Erythematosus



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— PATIENT CARE PROCESS



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Patient Care Process for the Management of Systemic Lupus Erythematosus



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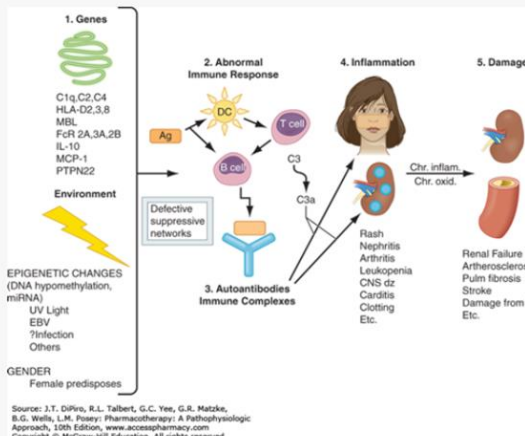
- Patient characteristics (e.g., age, race, sex, pregnancy status)
- Patient history (past medical, pregnancies and outcomes, symptoms, family, social—dietary habits, alcohol and tobacco use)
- Current medications and prior lupus medication use
- Immunization history
- Objective data (see [Clinical Presentation](#) 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, antinuclear 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 chemoattractant 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.)

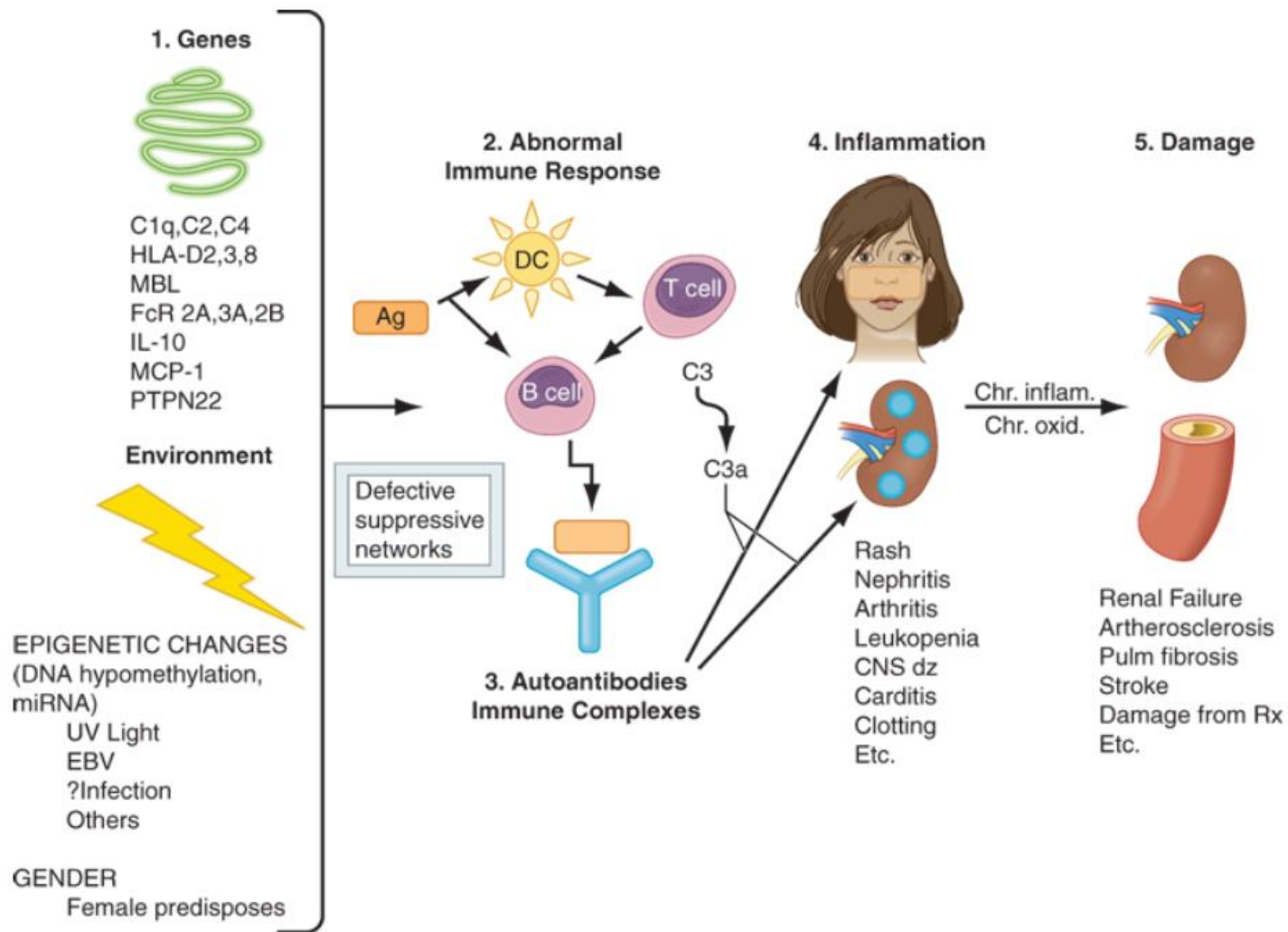


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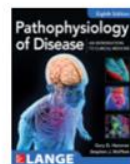
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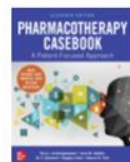
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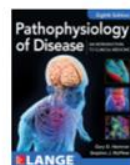
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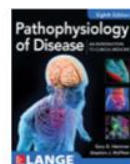
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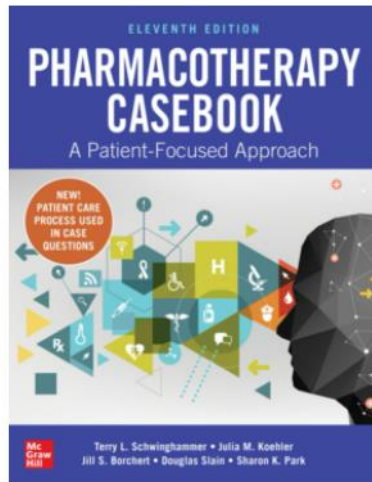
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Systemic Lupus Erythematosus



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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.

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