<<Braunwald心脏病学（套装上下>>

图书基本信息

书名：<<Braunwald心脏病学（套装上下册）>>

13位ISBN编号：9787543331792

10位ISBN编号：7543331799

出版时间：2013-1

出版时间：天津科技翻译出版公司

作者简介

作者：（美国）Eugene Braunwald 编者：（美国）罗伯特•O•波诺 （美国）道格拉斯•P•兹普 （美国）彼得•利贝 （美国）道格拉斯•L•曼 Eugene Braunwald，教授，美国哈佛大学医学院的著名心血管学专家，心脏病学泰斗，他主编的心脏病学和哈里森内科学是全世界心血管医生的圣经。

罗伯特•0•波诺教授，美国芝加哥西北大学Feinberg医学院心脏中心主任，医学部副主席，美国心脏学会主席。

道格拉斯•L•曼教授，美国圣 路易斯华盛顿大学Barnes jewish医院心脏科主任，细胞生物和生理医学教授。

道格拉斯•P•兹普教授，美国印第安纳大学医学院Krannert心脏研究所和心脏中心名誉主任。

彼得•利贝教授，美国布莱根妇女医院心血管科主任，哈佛医学院医学教授。

霍勇（专家推荐），北京大学第一医院，心内科及心脏中心主任，主任医师，教授，博士生导师，中华医学会心血管病学分会候选主任委员、中国医师协会心血管内科医师分会副会长，《中国介入心脏病学杂志》主编，中华医学会心血管介入治疗培训中心主任。

<<Braunwald心脏病学（套装上下>>

书籍目录

《Braunwald心脏病学：心血管内科学教科书（上册）》目录： PART Ⅰ FUNDAMENTALS OF CARDIOVASCULAF DISEASE CHAPTER1 Global Burden of Cardiovascular Disease 1 CHAPTER2 Heart Disease in Varied Populations 21 CHAPTER3 Ethics in Cardiovascular Medicine 30 CHAPTER4 Clinical Decision Making in Cardiology 35 CHAPTER5 Measurement and Improvement of Quality of Cardiovascular Care 42 CHAPTER6 Design and Conduct of Clinical Trials 49 PART Ⅱ MOLECULAR BIOLOGY AND GENETICS CHAPTER7 Principles of Cardiovascular Molecular Biology and Genetics 57 CHAPTER8 Inherited Causes of Cardiovascular Disease 70 CHAPTER 9 Genetics of Cardiac Arrhythmias 81 CHAPTER10 Principles of Drug Therapy 91 Dan M. Roden CHAPTER 11 Cardiovascular Regeneration and Tissue Engineering 99 PART Ⅲ EVALUATION OF THE PATIENT CHAPTER12 The History and Physical Examination: An Evidence-Based Approach 107 CHAPTER13 Electrocardiography 126 Guidelines: Electrocardiography 165 CHAPTER14 Exercise Stress Testing 168 Guidelines: Exercise StressTesting 192 CHAPTER15 Echocardiography 200 Appropriate Use Criteria: Echocardiography 270 CHAPTER16 The Chest Radiograph in Cardiovascular Disease 277 CHAPTER17 Nudear Cardiology 293 Appropriate Use Criteria: Nuclear Cardiology 336 CHAPTER18 Cardiovascular Magnetic Resonance Imaging 340 Appropriate Use Criteria: Cardiovascular Magnetic Resonance 359 CHAPTER19 Cardiac Computed Tomography 362 Appropriate Use Criteria: Cardiac Computed Tomography 379 CHAPTER20 Cardiac Catheterization 383 …… PART IV HEART FAILURE PART Ⅴ ARRHYTHMIAS, SUDDEN DEATH, AND SYNCOPE PART VI PREVENTIVE CARDIOLOGY PART Ⅶ ATHEROSCLERoT|C CARDlOVASCULAR DIASEASE PART Ⅷ DISEASES OF THE HEART, PERICARDIUM， AND PULMONARY VASCULATURE BED PART Ⅹ CARDIOVASCULAR DISEASE AND DISORDERS OF OTHER ORGANS …… 《Braunwald心脏病学：心血管内科学教科书（下册）》

章节摘录

版权页： 插图： Pharmacologic Rhythm Control The results of published studies on the efficacy of antiarrhythmic drugs for AF suggest that all of the available drugs except amiodarone have similar efficacy and are associated with a 50% to 60% reduction in the odds of recurrent AF during 1 year of treatment.29 The one drug that stands out as having higher efficacy than the others is amiodarone. In studies that directly compared amiodarone with sotalol or class I drugs, amiodarone was 60% to 70% more effective in suppressing AF. However, because of the risk of organ toxicity, amiodarone is not appropriate first-line drug therapy for most categories of patients with AF. Because their efficacy is in the same general range, the selection of an antiarrhythmic drug to prevent AF often is dictated by the issues of safety and side effects. Ventricular proarrhythmia from class IA agents (quinidine, procainamide, disopyramide) and class III agents (sotalol, dofetilide, drone-darone, amiodarone) is manifested as QT prolongation and polymorphic ventricular tachycardia (torsades de pointes). Risk factors for this type of proarrhythmia include female gender, left ventricular dysfunction, and hypokalemia.The risk of torsades de pointes appears to be much lower with dronedarone and amiodarone than with the other class Ill drugs.The ventricular proarrhythmia from class IC agents (flecainide and propafenone) is manifested as monomorphic ventricular tachycardia, sometimes associated with widening of the QRS complex during sinus rhythm but not QT prolongation. Published studies indicate that the drugs most likely to result in ventricular proarrhythmia are quinidine, fiecainide, sotalol, and dofetilide. In controlled studies, these agents increased the risk of ventricular tachycardia by a factor of 2 to 6. Adverse drug events resulting in discontinuation of drug therapy are fairly common with rhythm-control drugs.Withdrawal due to adverse effects was most common with quinidine, disopyramide, flecainide,sotalol, and amiodarone.29 A review of studies in which 32 treatment arms received an antiarrhythmic drug for AF found that 10.4% of patients discontinued drug therapy because of an adverse drug event,most commonly gastrointestinal side effects and neuropathy. The best options for drug therapy to suppress AF depend on the patient's comorbidities. In patients with lone AF or minimal heart disease (e.g., mild left ventricular hypertrophy), flecainide, propafenone, sotalol, and dronedarone are reasonable first-line drugs, and amiodarone and dofetilide can be considered if the first-line agents are ineffective or not tolerated. In patients with substantial left ventricular hypertrophy (left ventricutar wall thickness >13 mm), the hypertrophy may heighten the risk of ventricular proarrhythmia, and the safest choice for drug therapy is amiodarone. In patients with coronary artery disease, several of the class I drugs have been found toincrease the risk of death, and the safest first-line options are dofetilide, sotalol, and dronedarone, with amiodarone reserved for use asa second-line agent. In patients with heart failure, several antiarrhythmic drugs have been associated with increased mortality,and the onlytwo drugs known to have a neutral effect on survival are amiodaroroneand dofetilide (see Chap. 37).

编辑推荐

版权说明