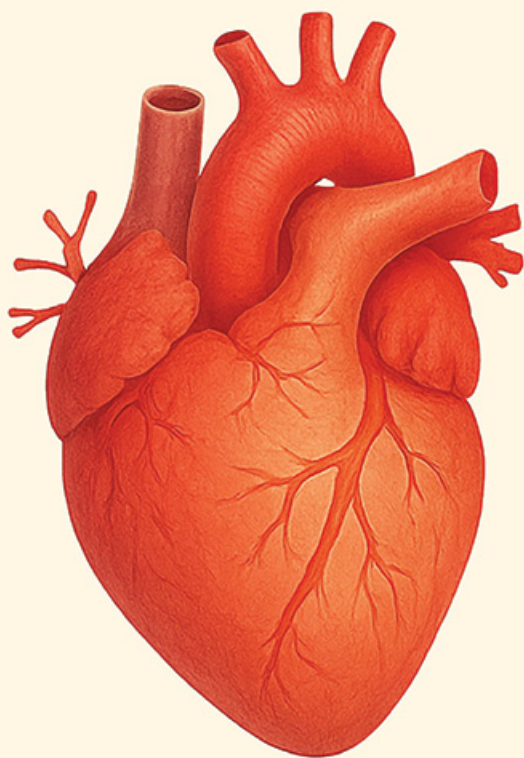




Manual of Cardiology

Kanu Chatterjee



JAYPEE

Contents

1. Cardiovascular Pharmacology	1
1.1 Diuretics	1
<i>Introduction</i>	1
<i>Classification of the diuretic compounds</i>	1
<i>Clinical pharmacology of the diuretic compounds</i>	2
<i>Adaptive responses to diuretic administration</i>	4
<i>Individual diuretic classes</i>	5
<i>Clinical use of diuretics in cardiovascular diseases</i>	11
<i>Adverse effects of diuretics</i>	15
1.2 Vasodilators and Neurohormone Modulators	18
<i>Introduction</i>	18
<i>Vasodilator drugs and low blood pressure</i>	19
<i>Arteriolar vasodilators</i>	19
<i>Renin–angiotensin–aldosterone system (RAAS) blockers</i>	22
<i>Mineralocorticoid (aldosterone) receptor blockers</i>	26
<i>Phosphodiesterase type 5 inhibitors</i>	28
<i>Intravenous vasodilators</i>	28
<i>Oral β-adrenergic blocking drugs</i>	29
1.3 Positive Inotropic Drugs	32
<i>Introduction</i>	32
<i>Intravenously administered, short-term positive inotropic therapy</i>	32
1.4 Antilipid Agents	41
<i>Introduction</i>	41
<i>Appropriate uses</i>	42
<i>Statins</i>	43
<i>Add-on to statin therapy</i>	50
<i>Bile acid sequestrants</i>	50
<i>Ezetimibe</i>	51
<i>Niacin</i>	51
<i>Triglyceride-lowering therapy</i>	52
<i>Fibrates</i>	52
<i>Omega-3 fatty acids</i>	53
<i>Drugs in development</i>	54
1.5 Antithrombotic and Antiplatelet Agents	54
<i>Introduction</i>	54
<i>Clotting, A primer</i>	55
<i>Antithrombotic agents</i>	55
<i>Vitamin K antagonists (VKA)</i>	58
<i>Direct factor Xa inhibitors</i>	59
<i>Direct thrombin inhibitors</i>	60
<i>Antiplatelet agents</i>	63

2. Diagnosis**70****2.1 History and Physical Examination 70***History 70**Physical examination 75***2.2 Plain Film Imaging of Adult Cardiovascular Disease 90***Introduction 90**Chest film technique 90**Cardiac anatomy on chest radiographs 91**Cardiac chamber enlargement 92**Radiographic manifestations of congestive heart failure 96**Cardiac calcifications 98**Acquired valvular heart disease 98**Pericardial disorders 100***2.3 Electrocardiogram 101***Introduction 101**Basis of electrocardiography 101**Component parts of the electrocardiogram 102**Lead systems used to record the electrocardiogram 102**A systematic way of looking at the interpretation of the electrocardiogram 103**Characterization of QRS complex 108**ST-T wave abnormalities 112**"U" wave 113**QT interval 113***2.4 ECG Exercise Testing 115***Introduction 115**Before the test 115**Methodology of exercise testing 117**During the test 119**After the test 126**Screening 128***2.5 Left Ventricle 132***Introduction 132**Systolic function 132**Contrast-enhanced echocardiography 134**Other echo-derived indices of left ventricular systolic function 134**Strain-derived indices 135**Recognizing the etiology of cardiac dysfunction 135**Visual qualitative indicators of**systolic dysfunction 135**Diastolic function 136***2.6 Transthoracic Echocardiography 139***Introduction 139**Chamber quantitation 141**Doppler ECHO 143**Diastolic function 143**Pulmonary hypertension 144**Pericardial disease 144*

<i>Valvular heart disease</i>	145
<i>Infective endocarditis</i>	148
<i>Intracardiac masses</i>	148
<i>Contrast echocardiography</i>	149
<i>Cardiac resynchronization therapy</i>	149

2.7 Stress Echocardiography 150

<i>Introduction</i>	150
<i>Using stress echocardiography in clinical decisions</i>	150
<i>Future of stress ECHO</i>	155

2.8 Transesophageal Echocardiography 156

<i>Introduction</i>	156
<i>Performance</i>	156
<i>Safety</i>	156
<i>Views</i>	156
<i>Major clinical applications</i>	158
<i>Structural valve assessment</i>	161
<i>Acute aortic dissection</i>	164
<i>Procedural adjunct or intraoperative transesophageal echocardiography</i>	165

2.9 Cardiovascular Nuclear Medicine— Nuclear Cardiology 167

<i>Introduction</i>	167
<i>Myocardial perfusion imaging</i>	167
<i>Positron emission tomography perfusion and metabolism</i>	169
<i>Imaging myocardial viability</i>	169
<i>Imaging perfusion</i>	171
<i>Radiation concerns</i>	172

2.10 Cardiac Computed Tomography 173

<i>Introduction</i>	173
<i>Coronary artery disease</i>	173
<i>Myocardium and chambers</i>	176
<i>Pulmonary veins</i>	176
<i>Cardiac veins</i>	177
<i>Valvular disease</i>	177
<i>Pericardium</i>	177
<i>Future</i>	177

2.11 Cardiovascular Magnetic Resonance Imaging 178

<i>Introduction</i>	178
<i>Diagnosis of epicardial coronary artery stenosis</i>	178
<i>Assessment of global and regional left ventricular function at rest and during inotropic stress</i>	179
<i>Myocardial perfusion imaging</i>	179
<i>Cardiovascular magnetic resonance coronary angiography</i>	180
<i>Dilated cardiomyopathy</i>	181
<i>Hypertrophic cardiomyopathy</i>	181
<i>Restrictive cardiomyopathy</i>	181
<i>Cardiovascular magnetic resonance-guided therapy</i>	182
<i>Valvular heart disease</i>	182
<i>Diseases with right ventricular predominance</i>	182
<i>Miscellaneous conditions</i>	183

- 2.12 Molecular Imaging of Vascular Disease 184**
 - Introduction 184*
 - Molecular imaging modalities 184*
 - Molecular imaging of vascular disease processes 186*
- 2.13 Cardiac Hemodynamics and Coronary Physiology 190**
 - Introduction 190*
 - Cardiac catheterization—the basics 190*
 - Cardiac cycle pressure waveforms 192*
 - Hemodynamics in valvular heart disease 193*
 - Hemodynamics in cardiomyopathy 197*
 - Hemodynamics in pericardial disease 198*
 - Coronary hemodynamics 200*
- 2.14 Cardiac Biopsy 201**
 - Introduction 201*
 - Techniques 201*
 - Safety and complications 202*
 - Analysis of EMB tissue 203*
 - Indications 204*
- 2.15 Coronary Angiography and Catheter-based Coronary Intervention 207**
 - Introduction 207*
 - Indications for coronary angiography 207*
 - Contraindications for coronary angiography 207*
 - Patient preparation 209*
 - Sites and techniques of vascular access 209*
 - Catheters for coronary angiography 211*
 - Arterial nomenclature and extent of disease 211*
 - Angiographic projections 212*
 - Congenital anomalies of the coronary circulation 213*
 - General principles for coronary and/or graft cannulation 214*
 - Fluoroscopic imaging system 214*
 - Characteristics of contrast media 214*
 - Access site hemostasis 215*
 - Complications of cardiac catheterization 216*
 - Percutaneous coronary intervention 217*
 - Pharmacotherapy for PCI 221*
 - Parenteral anticoagulant therapy 222*
 - Percutaneous transluminal coronary angioplasty 222*
 - Coronary stents 223*
 - Types of stents 223*
 - Procedural success and complications related to coronary intervention 223*

3. Electrophysiology

226

- 3.1 Arrhythmia Mechanisms 226**
 - Introduction 226*
 - Arrhythmia initiation 226*
- 3.2 Antiarrhythmic Drugs 234**
 - Introduction 234*

	<i>Arrhythmia mechanisms and antiarrhythmic drugs</i>	234
	<i>Indications for antiarrhythmic drug therapy</i>	234
	<i>Classification scheme</i>	235
	<i>Vaughan–Williams classification</i>	235
	<i>Antiarrhythmic drug selection in atrial fibrillation</i>	239
	<i>Outpatient versus in-hospital initiation for antiarrhythmic drug therapy</i>	240
	<i>Antiarrhythmic drugs in pregnancy and lactation</i>	241
	<i>Comparing antiarrhythmic drugs to implantable cardioverter defibrillators in patients at risk of arrhythmic death</i>	242
	<i>Antiarrhythmic drug–device interactions</i>	242
3.3	Syncope	243
	<i>Introduction</i>	243
	<i>Diagnostic tests</i>	245
	<i>Approach to the evaluation of syncope</i>	250
3.4	Atrial Fibrillation	253
	<i>Introduction</i>	253
	<i>Definition and Classification</i>	253
	<i>Etiology and Pathogenesis</i>	254
	<i>Diagnosis</i>	256
	<i>Management</i>	257
3.5	Supraventricular Tachycardia	261
	<i>Introduction</i>	261
	<i>Classification</i>	261
	<i>Diagnosis</i>	265
	<i>Treatments</i>	266
3.6	Clinical Spectrum of Ventricular Tachycardia	271
	<i>Introduction</i>	271
	<i>Monomorphic ventricular tachycardia</i>	272
3.7	Bradycardia and Heart Block	280
	<i>Introduction</i>	280
	<i>Bradycardia syndromes/diseases</i>	280
	<i>Clinical presentation</i>	281
	<i>Measurement/diagnosis</i>	281
	<i>Sinus node disease</i>	282
	<i>AV node disease</i>	282
	<i>Hemiblock</i>	283
	<i>Bundle branch block</i>	284
	<i>Treatment</i>	284
3.8	Arrhythmogenic Right Ventricular Dysplasia/ Cardiomyopathy	285
	<i>Introduction</i>	285
	<i>Molecular and genetic background</i>	285
	<i>Clinical presentation</i>	287
	<i>Clinical diagnosis</i>	288
	<i>Nonclassical ARVD/C subtypes</i>	289
	<i>Molecular genetic analysis</i>	289
	<i>Prognosis and therapy</i>	290

3.9 Long QT, Short QT, and Brugada Syndromes 291

Introduction 291
LQT syndrome 291
Brugada syndrome 294

3.10 Cardiac Resynchronization Therapy 296

Introduction 296
CRT: rationale for use 296
CRT in practice 296
Summary of CRT benefit 296
Prediction of response to CRT therapy 299
Role of dyssynchrony imaging 300
Dyssynchrony summary 300
CRT complications 300
Emerging CRT indications 300

3.11 Ambulatory Electrocardiographic Monitoring 305

Introduction 305
Holter monitoring 305
Event recorders 307
Mobile cardiac outpatient telemetry 308
Implantable loop recorders 308

3.12 Cardiac Arrest and Resuscitation 311

Introduction 311
Basic life support 312
Advanced cardiac life support 315
Risks 316
Cessation of resuscitation 318
Postresuscitation care 318

3.13 Risk Stratification for Sudden Cardiac Death 319

Introduction 319
Healthy athletes 319
Brugada syndrome 320
Long QT interval syndrome 320
Early repolarization 320
Short QT syndrome 320
Catecholamine polymorphic ventricular tachycardia 320
Wolff–Parkinson–White syndrome 321
Marfan's syndrome 321
Congenital heart disease 321
Nonischemic cardiomyopathy 322
Coronary artery disease 322

4. Coronary Heart Diseases

324

4.1 Coronary Heart Disease: Risk Factors 324

Introduction 324
CHD screening and prevention 325
Clustering and multiplicative effects of risk factors 325
CHD risk estimation 325
Traditional CHD risk factors 328
Emerging risk factors 332
Subclinical atherosclerosis 332

Translating risk factor screening into event reduction 332

4.2 Acute Coronary Syndrome (Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction): Diagnosis and Early Treatment 333

Introduction 333

Unstable angina and non-ST-segment 333

Clinical features 333

Risk stratification—putting it all together to determine the optimal treatment strategy 336

Early medical therapy 336

Early invasive or initial conservative strategy 340

Revascularization 341

4.3 Acute Coronary Syndrome II (ST-Elevation, Myocardial Infarction, and Post-Myocardial Infarction): Complications and Care 343

Introduction 343

Clinical presentation 343

Reperfusion 345

Early medical therapy 347

Postmyocardial infarction care 348

Complications 350

Special considerations 351

Continued medical therapy for patients with a myocardial infarction 351

4.4 Management of Patients with Coronary Artery Disease and Stable Angina 354

Introduction 354

Current therapeutic approaches for stable angina 354

Antianginal drug therapy 354

Newer antianginal drugs 356

Combination therapy 357

Other drugs in patients with stable angina and chronic CAD 357

Role of myocardial revascularization 357

Comparison of revascularization with pharmacological antianginal therapy 358

4.5 Cardiogenic Shock in Acute Coronary Syndromes 360

Introduction 360

Pathophysiology 360

Other cardiac causes of cardiogenic shock 361

Diagnostic evaluation 363

Medical management 363

Mechanical support 363

Revascularization 363

4.6 Acute Right Ventricular Infarction 364

Introduction 364

Patterns of coronary compromise resulting in RVI 364

Effects of ischemia on RV systolic and diastolic function 365

Effects of reperfusion on ischemic RV dysfunction 365

Rhythm disorders and reflexes associated with RVI 366

Clinical Presentations and Evaluation 366
Noninvasive and hemodynamic evaluation 367
Differential diagnosis of RVI 367
Therapy 367

5. Valvular Heart Diseases

369

5.1 Aortic Valve Disease 369

Aortic stenosis 369
Aortic regurgitation 374

5.2 Mitral Valve Disease 380

Introduction 380
Rheumatic heart disease 380
Mitral stenosis 381
Mitral regurgitation 388

5.3 Tricuspid Valve Disease: Evaluation and Management 397

Introduction 397
Etiology of tricuspid valve disease 397
Clinical presentation 398
Laboratory diagnosis 399
Treatment 400

5.4 Congenital Pulmonic Stenosis 403

Introduction 403
Valvar pulmonic stenosis 403
Isolated infundibular stenosis 408
Supravalvar stenosis 409

5.5 Catheter-based Treatment of Valvular Heart Disease 410

Introduction 410
Catheter-based treatment of mitral valve disease 410
Catheter-based treatment of pulmonary valve disease 413
Catheter-based therapies for aortic stenosis 414

5.6 Infective Endocarditis 417

Introduction 417
Pathogenesis 418
Presentation and diagnosis 422
Management 425

5.7 Prosthetic Heart Valves 433

Risk of valve replacement 433
Types of prosthetic valves 434
Selecting the optimal prosthesis 434
Prosthesis-patient mismatch 437
Long-term management 441
Long-term complications 444

5.8 Antithrombotic Therapy in Valvular Heart Disease 451

Introduction 451
Prophylactic antithrombotic therapy 452
Rheumatic valvular heart disease 453
Mitral valve prolapse 453
Calcified or degenerative valvular disease 453

Prosthetic valves 454
Bioprosthetic valves 454
Valvuloplasty and valve repair 455
Management issues 455

6. Vascular Diseases

458

6.1 Evaluation and Management of the Patient with Essential Hypertension 458

Evaluation of the patient with hypertension 458
Antihypertensive therapy 462
Hemodynamic concepts 463
Clinical pharmacologic concepts 463
Treatment algorithms advocated over the years 468

6.2 Peripheral Vascular and Cerebrovascular Disease 469

Introduction 469
Peripheral arterial disease 470
Carotid artery disease 477
Renal artery stenosis 479
Subclavian artery stenosis 480
Mesenteric ischemia 481

6.3 Aortic Dissection and Aneurysm 483

Aortic Dissection 483
Introduction 483
Classification 483
Clinical examination 484
Diagnosis 485
Treatment 487
Aortic Aneurysm 489
Introduction 489
Treatment 489
Anatomic substrate for endovascular aneurysm repair 490
Adjunctive devices and techniques 490
Endoleak 490
Late-occurring complications of endovascular aneurysm repair 491
Follow-up imaging 492

6.4 Autonomic Dysfunction and the Cardiovascular System 493

Introduction 493
Autonomic regulation of the cardiovascular system 493
Autonomic testing 495
Primary chronic autonomic failure 497
Secondary and congenital autonomic failure 498
Chronic orthostatic intolerance 498

7. Heart Failure

500

7.1 Diagnosis 500

Analysis of symptoms 500
Physical examination 501

Electrocardiogram 503
Chest radiograph 505
Echocardiography 505
Cardiac magnetic resonance and cardiac tomography 506
Biomarkers 507
Exercise tests and six-minute walk test 510
Myocardial ischemia 511
Genetic studies 512

7.2 Systolic Heart Failure (Heart Failure with Reduced Ejection Fraction) 513

Introduction 513
Ventricular remodeling 514
Functional derangements and hemodynamic consequences 519
Initial treatment of systolic heart failure 520
Symptomatic systolic heart failure 521
Nonpharmacologic treatments 524
Follow-up evaluation 525

7.3 Diastolic Heart Failure (Heart Failure with Preserved Ejection Fraction) 526

Definition 526
Epidemiology and risk factors 526
Pathophysiology 526
Future directions 532

7.4 Cardiorenal Syndrome: The Interplay between Cardiac and Renal Function in Patients with Congestive Heart Failure 534

Definition of the cardiorenal syndrome 534
*Role of evidence-based therapies in patients with
 heart failure and the cardiorenal syndrome* 536
*Role of ultrafiltration on diuretic resistance and the cardiorenal
 syndrome* 541
*Treatment of the cardiorenal syndrome:
 an approach to the individual patient* 541

7.5 Acute Heart Failure Syndromes 545

Introduction 545
Pathophysiology 546
Acute heart failure syndromes management 548
Clinical trials in acute heart failure syndromes 556

7.6 Cardiopulmonary Exercise Testing and Training in Heart Failure 558

Introduction 558
Exercise response in heart failure 559
Cardiopulmonary exercise testing 560
Indications for CPX testing in heart failure 565
Exercise training in heart failure 566

7.7 Advanced Cardiac Therapies for End-stage Heart Failure: Cardiac Transplantation and Mechanical Circulatory Support 568

Introduction 568

<i>Identifying candidates for advanced cardiac therapies</i>	569
<i>Heart transplantation</i>	571
<i>Mechanical circulatory support</i>	581
<i>Future directions</i>	587

8. Myocardial and Pericardial Diseases

591

8.1 Hypertrophic Cardiomyopathy 591

<i>Introduction</i>	591
<i>Pathology</i>	592
<i>Clinical presentation</i>	594
<i>Diagnosis</i>	595
<i>Natural history</i>	598
<i>Management</i>	599
<i>Dual-chamber pacemaker</i>	602

8.2 Dilated Cardiomyopathy 604

<i>Introduction</i>	604
<i>Definition</i>	604
<i>Pathology</i>	604
<i>Etiology</i>	605
<i>Prognosis</i>	608

8.3 Restrictive and Obliterative Cardiomyopathies 610

<i>Introduction</i>	610
<i>Restrictive cardiomyopathies</i>	610
<i>Tropical endomyocardial fibrosis (Davie's disease)</i>	612
<i>Right ventricular endomyocardial fibrosis</i>	612
<i>Left ventricular endomyocardial fibrosis</i>	613
<i>Loeffler's endocarditis</i>	614
<i>Idiopathic restrictive cardiomyopathy</i>	615

8.4 Specific Cardiomyopathies 616

<i>Amyloid Heart Disease</i>	616
<i>Introduction</i>	616
<i>Overview of cardiac amyloidosis</i>	616
<i>Classification of amyloidosis</i>	617
<i>Cardiac amyloidosis</i>	617
<i>Treatment</i>	621
<i>Peripartum Cardiomyopathy</i>	623
<i>Introduction</i>	623
<i>Diagnosis</i>	623
<i>Prognosis</i>	623
<i>Treatment</i>	624
<i>Labor and delivery</i>	624
<i>Chemotherapy-induced Cardiomyopathy</i>	625
<i>Introduction</i>	625
<i>Pathophysiology of anthracycline-induced cardiomyopathy</i>	625
<i>Mechanism of chemotherapy-induced cardiac dysfunction</i>	626
<i>Diagnosis</i>	627
<i>Monitoring</i>	628
<i>Preventive strategies</i>	629
<i>Treatment</i>	629

- 8.5 Pericardial Disease 631**
Introduction 631
Acute pericarditis 631
Chronic relapsing pericarditis 633
Pericardial effusion and pericardial tamponade 634
Constrictive pericarditis 635
- 8.6 Radiation-induced Heart Disease 641**
Introduction 641
Radiation-induced pericardial disease 641
Radiation-induced myocardial disease 642
Radiation-induced coronary artery disease 643
Radiation-induced valvular heart disease 643
Conduction system disease 644
Prevention 644

9. Pulmonary Vascular Disease and Adult Congenital Heart Disease 646

- 9.1 Pulmonary Arterial Hypertension 646**
Definitions and classifications 646
Pathophysiology of pulmonary arterial hypertension 649
Diagnostic evaluation 651
Therapeutic options for the treatment of pulmonary arterial hypertension 657
Treatment algorithm and evaluating response to therapy 661
Therapy of decompensated right heart failure in pulmonary arterial hypertension 662
- 9.2 Congenital Heart Disease in the Adult Patient 665**
Acyanotic Heart Disease 665
Congenital valvar aortic stenosis 665
Coarctation of the aorta 667
Valvar pulmonic stenosis 670
Atrial septal defects 672
Ventricular septal defects 674
Patent ductus arteriosus 677
Other Acyanotic Lesions 680
Ebstein's Anomaly 680
Cyanotic Congenital Heart Disease 682
Palliative shunts 683
Tetralogy of Fallot 683
Truncus arteriosus 690
Total anomalous pulmonary venous return 691

10. Secondary Disorders of the Heart 695

- 10.1 Alcohol and Arrhythmia 695**
Direct effects of ethanol exposure on heart cells and tissues 695
Ethanol ingestion and the normal cardiac conduction system 695

- Binge drinking and transient clinical arrhythmias—
holiday heart* 695
- Alcohol consumption, chronic atrial fibrillation, and
atrial flutter* 696
- Alcohol consumption and sudden cardiac death* 696
- Summary and clinical guidelines* 697

10.2 Insulin Resistance and Cardiomyopathy 698

- Introduction* 698
- Diastolic heart failure and insulin resistance* 698
- Myocardial energy metabolism* 699
- Metabolic effects of insulin resistance* 700
- Structural effects of insulin resistance* 701
- Treatment* 702

10.3 Cardiac Complications of Substance Abuse 705

- Introduction* 705
- Substances of abuse* 705
- Marijuana, tetrahydrocannabinol, hashish* 710
- Club drugs: MDMA, GHB, ketamine, rohypnol* 710
- Body image drugs* 712
- Narcotics* 712

10.4 HIV/AIDS and Cardiovascular Disease 713

- Introduction* 713
- HIV and coronary heart disease* 714
- Surrogate measures of atherosclerosis* 716
- HIV-related left ventricular dysfunction and myocarditis* 717
- Cerebrovascular disease* 718
- Miscellaneous* 718

10.5 Systemic Autoimmune Diseases and the Heart 719

- Rheumatoid arthritis* 719
- Spondyloarthropathies* 721
- Polymyositis—dermatomyositis* 723
- Systemic lupus erythematosus* 724

10.6 Neurogenic and Stress Cardiomyopathy 726

- Introduction* 726
- Neurogenic cardiomyopathy* 726
- Stress cardiomyopathy* 729

10.7 Kidney and the Heart 732

- Introduction* 732
- Pathophysiology* 732
- Cardiovascular risk factors in chronic kidney disease* 733
- Spectrum of cardiovascular disease in
chronic kidney disease* 734
- Diagnostic tests* 736
- Principles of treatment of cardiovascular disease* 738
- Kidney transplant recipients* 738

10.8 Endocrine Heart Disease 740

- Introduction* 740
- Diabetes mellitus* 740
- Thyroid disease* 742

Pituitary disorders 744
Adrenal disorders 745
Parathyroid disorders 747
Carcinoid syndrome 747

10.9 Venous Thromboembolism and Cor Pulmonale 749

Venous thromboembolism 749
Cor pulmonale 758

11. Relevant Issues in Clinical Cardiology

761

11.1 Noncardiac Surgery in Cardiac Patients 761

Introduction 761
Preoperative cardiac risk assessment 761
Preoperative diagnostic testing 764
Preoperative risk mitigation strategies 766
Intraoperative management 768
*Management of patients with implanted
 electronic devices* 768
Postoperative management 768

11.2 Gender and Cardiovascular Disease 770

Introduction 770
Prevalence of IHD in women 770
Identification of IHD risk factors in women 770
*Assessment of symptoms and myocardial ischemia in
 women symptom assessment* 771
Management of IHD in women 774
Heart failure in women 777
Gender and cardiac arrhythmias 777

11.3 Overview of the Athlete's Heart 779

Introduction 779
Exercise physiology and the athlete's heart: overview 779
Issues relevant to the cardiovascular care of athletes 780

11.4 Cardiovascular Aging 783

Introduction 783
Age-related changes 784
Clinical syndromes 785
Special issues 788

12. Preventive Strategies for Other Cardiovascular Diseases

790

12.1 Prevention of Heart Failure 790

Introduction 790
Staging of heart failure 790
Future perspectives 794

12.2 Stroke: Prevention and Treatment 795

Introduction 795
Definitions 795
Stroke as a symptom 796
Prevention 804

General acute treatment 804
Treatment of acute ischemic stroke 805
Treatment of acute hemorrhagic stroke 807
General in-hospital care and rehabilitation 808

12.3 Rheumatic Fever 810

Introduction 810
Pathogenesis 810
Diagnosis of rheumatic fever 811
Clinical features 812
Treatment 814

13. Evolving Concepts

818

13.1 Preventing Errors in Cardiovascular Medicine 818

Introduction 818
Modern approach to patient safety 818
How to improve patient safety? 819
Communication and culture 820
Learning from mistakes 821
Creating a safe workforce 821
Preventing diagnostic errors 821
What can patients do to keep themselves safe? 822
Changing policy context for patient safety 822

13.2 Integrative Cardiology: The Use of Complementary Therapies and Beyond 823

Nonconventional therapies and cardiology 823
What is integrative medicine? 823
What is integrative cardiology? 823
Dyslipidemia 824
Hypertension 826
Coronary artery disease 828
Heart failure 830

Index

835

Chapter 2

Diagnosis

2.1 History and Physical Examination

The history and physical examination are essential, not only for the diagnosis of cardiovascular disorders but also to assess severity, to establish a plan of management and to assess the prognosis. Appropriate history and physical examination are also essential to decide what tests are necessary for a patient, as presently a plethora of tests is available for the diagnosis and management of the same cardiac disorder. It should also be appreciated that “history and physical examination” are cost-effective. During examination of the patient, the physician can also gain the confidence of the patient and of the family and can establish a good rapport that is necessary for the appropriate management of the problem of the patient.

□ HISTORY

General Approach

During history taking, it is desirable to allow the patient to present the symptoms without interruption. Frequent interruptions give the impression that the physician is in hurry and impatient and disinterested. After the patient describes the symptoms, it is appropriate to discuss with the patient and the family to ascertain the chronology of symptoms and their severity. It is pertinent to enquire about each symptom. The major symptoms associated with cardiovascular disorders are chest pain or discomfort, dyspnea, palpitations, dizziness, and syncope.

Analysis of Symptoms

Chest Pain or Discomfort

Chest pain is one of the very common presenting symptoms that patients present to the cardiologists. The chest pain or discomfort can be caused by various cardiac and noncardiac causes, which are summarized in Tables 1 to 4.

Angina pectoris is a symptom of both of chronic coronary artery disease and of acute coronary syndromes. For the diagnosis of angina pectoris, it is imperative to enquire about the character, location, site of radiation, duration, and precipitating and relieving factors of the chest discomfort. While elucidating the history, the following thing should be noted:

Table 1: Cardiac chest pain

- | | |
|----------------------------|------------------------------|
| • Coronary artery disease | • Noncoronary artery disease |
| • Acute coronary syndromes | • Aortic dissection |
| • Stable angina | • Acute pulmonary embolism |
| • Ischemic cardiomyopathy | |

Table 2: Noncardiac chest pain

- | | |
|---------------------|-----------------|
| • Pulmonary | ▪ Pneumothorax |
| ▪ Pleuritis | ▪ Mediastinitis |
| ▪ Pneumonia | ▪ Tumor |
| ▪ Tracheobronchitis | |

Table 3: Cardiac causes of chest discomfort

- | | |
|-------------------------------|------------------------------|
| • Aortic stenosis | • Restrictive cardiomyopathy |
| • Aortic regurgitations | • Pulmonary hypertension |
| • Hypertrophic cardiomyopathy | |

Table 4: Noncardiac chest pain

- | | |
|-----------------------------|-----------------------|
| • Musculoskeletal | • Other causes |
| ▪ Costochondritis | ▪ Herpes zoster |
| ▪ Intercostal muscle cramps | ▪ Emotional |
| ▪ Cervical disk disease | ▪ Chest wall tumor |
| | ▪ Disorders of breast |

Character: Frequently, it is described as “heaviness”, “pressure”, “tightness”, “squeezing” or “band across the chest”. The character of angina pectoris is usually “dull and deep” and not “sharp and superficial”.

Location: The location of the chest discomfort can be retrosternal, epigastric or left pectoral. When chest pain is much localized and can be indicated by one or two finger tips, it is unlikely to be angina pectoris.

Radiation: The radiation of angina pain may be to one or both shoulders, one or both arms and hands, one or both sides of the neck, lower jaw and interscapular area. The radiation can also occur to armpits, epigastrium and subcostal areas. The pain radiates from the center to the periphery (centripetal) and rarely centrifugal.

Intensity: The intensity of angina increases slowly and reaches its peak in minutes, not instantaneously. Similarly, it is relieved gradually, not abruptly. Analysis of the duration of chest discomfort is also helpful to decide whether it is ischemic or nonischemic pain.

Gestures: Patient’s gestures during describing the chest discomfort have been thought to be useful in the diagnosis of its etiology.

Precipitating and relieving factors: The precipitating and relieving factors of the chest discomfort should be analyzed to determine its etiology. Classic angina (Heberden’s angina) is precipitated by exercise or by emotional stress and is relieved when the exercise is discontinued. It tends to occur often after meals.

Exposure to cold weather precipitates angina more easily in patients with classic angina. Similarly, carrying heavy objects and heavy meals are also frequent precipitating factors. The character, location, and radiation of chest discomfort are similar in the different clinical subsets of angina. However, some distinctive features can be recognized in various subsets.

The atypical presentations frequently called “angina equivalents” are dyspnea, indigestion and belching, and dizziness and syncope without chest pain. The atypical presentations are more common in diabetics, women, and the elderly. A few clinical features of angina are summarized in Tables 5 and 6.

Table 5: Clinical features of stable angina

- Location
 - Usually retrosternal, can be epigastric, interscapular
- Localization
 - Usually diffuse, difficult to localize
 - When is very localized (point sign)—unlikely to be angina
- Quality
 - Pressure, heaviness, squeezing, indigestion
- Radiation
 - One or both arms, upper back, neck, epigastrium, shoulder
 - Lower jaw (upper jaw, head, lower back, lower abdomen, or lower extremities radiation is not feature of angina)
- Duration
 - Usually 1–10 minutes (not a few seconds or hours)
- Precipitating factors
 - Physical activity, emotional stress, sexual intercourse
- Aggravating factors
 - Cold weather, heavy meals
- Relieving factors
 - Cessation of activity, nitroglycerin (if relief is instantaneous it is unlikely to be angina)
- Associated symptoms
 - Usually none, occasionally dyspnea

Table 6: Clinical features of chest pain in acute coronary syndromes

- Location
 - Same as stable angina
- Quality
 - Same as stable angina
- Duration
 - Usually longer than stable angina
- Relieving factors
 - Usually unrelieved by rest or nitroglycerin
- Associated symptoms
 - Dyspnea, sweating, weakness, nausea, vomiting presyncope, or syncope

Chest pain resulting from acute aortic dissection is usually severe. The location can be anterior chest. Radiation to the back is common. The downward radiation along the spine is very suggestive of aortic dissection. The onset of pain is frequently instantaneous and the maximal severity may occur at the onset. A few clinical features of pain of acute pericarditis, pulmonary embolism and acute aortic dissection are summarized in Table 7.

The severity of angina is assessed by the Canadian Cardiovascular Society (CCS) functional classification (Table 8) or specific activity scale (Table 9). CCS is most frequently used to assess the severity of angina.

In suspected patients inquiries should be made about the risk factors. The modifiable and nonmodifiable risk factors for atherosclerotic coronary artery diseases are summarized in Table 10.

Dyspnea

Dyspnea is an uncomfortable sensation of breathing. It is also defined as “labored” breathing. Dyspnea can occur during exertion (exertional), during recumbency

Table 7: Clinical features of chest pain due to acute pericarditis, acute pulmonary embolism, and acute aortic dissection

Acute pericarditis	Acute pulmonary embolism	Acute aortic dissection
• Location ▪ Anterior chest, superficial • Character ▪ Sharp, can be pleuritic • Radiation ▪ Supraspinatus areas, shoulders, back • Relieving factors ▪ Worse in supine position, less severe in sitting and leaning forward position, relieved by analgesics, nonsteroidals and steroids	• Location ▪ Usually retrosternal • Quality ▪ Deep, may be similar to acute coronary syndromes • Associated symptoms ▪ Tachypnea and dyspnea	• Location ▪ Chest, back • Quality ▪ Shearing, tearing • Onset ▪ Instantaneous • Radiation ▪ Downwards along the spine

Table 8: Canadian Cardiovascular Society (CCS) functional classification**Class I**

- Ordinary physical activity, such as walking and climbing stairs, does not cause angina
- Angina with strenuous or rapid or prolonged exertion at work or recreation

Class II

- Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind or when under emotional stress, or only during the few hours after awakening
- Walking more than two blocks on the level, and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions

Class III

- Marked limitation of ordinary physical activity
- Walking 1–2 blocks on the level and climbing more than one flight in normal conditions

Class IV

- Inability to carry on any physical activity without discomfort—anginal syndrome may be present at rest

Table 9: Specific activity scale**Class I**

- Patients can perform to completion any activity requiring ≤ 7 metabolic equivalents [e.g. can carry 24 lbs up eight steps; carry objects that weigh 80 lbs, do outdoor work (shovel snow, spade soil); do recreational activities (skiing, basketball, squash, handball, jog/walk 5 mph)]

Class II

- Patients can perform to completion any activity requiring ≤ 5 metabolic equivalents (e.g. have sexual intercourse without stopping, garden, rake, weed, roller skate, dance fox trot, walk 4 mph on level ground), but cannot and do not perform to completion activities requiring ≥ 7 metabolic equivalents

Class III

- Patients can perform to completion any activity requiring ≥ 2 metabolic equivalents (e.g. shower without stopping, strip and make bed, clean windows, walk 2.5 mph, bowl, play golf, dress without stopping), but cannot and do not perform to completion any activities requiring ≥ 5 metabolic equivalents

Class IV

- Patients cannot or do not perform to completion activities requiring ≥ 2 metabolic equivalents. Cannot carry out activities listed above (Specific Activity Scale Class III)

Table 10: Cardiac and pulmonary causes of dyspnea**Differential diagnosis of dyspnea***Cardiac*

- CHF
- CAD
- Cardiomyopathy
- Valvular dysfunction
- LVH
- Pericardial diseases
- Arrhythmias
- Congenital HD

Pulmonary

- COPD
- Asthma
- Restrictive lung diseases
- Hereditary lung diseases
- Pneumothorax

(Abbreviations: CHF: Congestive heart disease; HD: Heart disease; CAD: Coronary heart disease; LVH: Left ventricular hypertrophy)

Table 11: Cardiac cause of dyspnea**Dyspnea***Cardiac or noncardiac dyspnea*

Physical examination:

- Signs of heart failure—diagnostic of cardiac cause, e.g. S3, elevated JVP, positive HJR

Presence of cardiac pathology:

- Very suggestive of cardiac cause

Chest X-ray:

- Very helpful when findings of pulmonary venous congestion or pulmonary hypertension are present

ECG:

- Normal electrocardiogram
- A negative predictive value has over 90%

(Abbreviations: JVP: Jugular venous pressure; HJR: Hepatojugular reflux)

(orthopnea) or even with standing (platypnea). There are both cardiac and noncardiac (Table 10) causes of dyspnea. Many patients have both cardiac and pulmonary disease. To distinguish between cardiac and noncardiac dyspnea, the measurements of serum B-type natriuretic peptide (BNP) or N-terminal ProBNP (NTBNP) is helpful. In noncardiac dyspnea, the natriuretic peptide levels are normal, and in patients with heart failure, they are substantially elevated.

Exertional dyspnea can be caused by both cardiac and noncardiac causes. Exertional dyspnea is an important symptom of chronic heart failure. Orthopnea is defined when patients develop dyspnea lying flat and feel better when the upper part of the torso is elevated.

There are many cardiac conditions, which can be associated with episodic severe dyspnea. In between the episodes of dyspnea, these patients are relatively asymptomatic and may have good exercise tolerance. Careful cardiovascular examination may also reveal the etiology of dyspnea. For example, evidence of valvular and myocardial heart diseases suggests cardiac cause of dyspnea (Table 11).

Palpitation

Palpitation is perceived as an uncomfortable sensation in the chest associated with heartbeats. The most frequent cause of palpitation is premature atrial or ventricular contractions. The premature beat itself is not felt; the normal beat following the

compensatory pause is felt as a strong beat. The patients usually describe this uncomfortable sensation as “a thump”, “skipped beat”, “the heart is coming out of chest”, “heart stops” and “heart stops beating”. The frequent premature beats may also be associated with other symptoms, such as dizziness, sinking feeling, shortness of breath and chest pain.

Syncope

Syncope is defined as transient loss of consciousness. Patients with presyncope complain of dizziness and near fainting, although they do not lose consciousness completely. The mechanism of cardiac syncope is reduced cerebral perfusion resulting from decreased cardiac output and hypotension.

A careful history is helpful for the diagnosis of the cause of syncope. Enquiry should be made about the circumstances in which syncope occurred, whether it was accompanied or preceded by palpitation, whether it occurs during exertion or it can also occur at rest, and whether it occurs only during upright position or it is unrelated to body position.

Edema

Patients with edema present with the symptom of “swelling”, usually of the lower extremities. Both cardiac and noncardiac conditions may be associated with edema. Enquiries should be made regarding the initial location and progression of edema. Right heart failure with systemic venous hypertension causes dependent edema, such as in the ankles, feet, and legs. In bedridden patients, edema can be predominantly in the back. Generalized edema is uncommon in heart failure and usually suggests permeability edema, such as in patients with hypoalbuminemia.

Cough

Paroxysms of cough may be the presenting symptoms in cardiac and noncardiac patients. Patients with left heart failure may complain of nocturnal cough with or without dyspnea. Paroxysms of nonproductive cough are bothersome complications of angiotensin-converting enzyme inhibitor therapy.

Hemoptysis

Hemoptysis is an uncommon presenting symptom of cardiac patients. Patients with hemodynamic pulmonary edema may present with history of frothy pink, blood-tinged sputum. These patients also have dyspnea.

Recurrent hemoptysis may be a presenting symptom in patients with precapillary pulmonary arterial hypertension and Eisenmenger's syndrome. Hemoptysis associated with pleuritic chest pain should raise the suspicion of pulmonary embolism. Patients on anticoagulation therapy may present with hemoptysis.

It should be appreciated, however, that frank hemoptysis is an uncommon presenting symptom of cardiac patients, and primary bronchopulmonary disease, such as malignancy, should always be suspected.

□ PHYSICAL EXAMINATION

Physical examination like history taking, is an integral part of evaluation of a patient with suspected or established cardiovascular disorders.

General Appearance

The physical examination starts with the inspection. During inspection, the physician has the opportunity to observe the patient's expression, skin color, posture, and general health status. During inspection, the nutritional status of the patient can be determined. Obesity or cachexia can easily be recognized. Obesity is a risk factor for metabolic syndrome, coronary artery disease, and heart failure.

Examination of the Skin

Examination of the color of the skin can reveal presence of cyanosis, jaundice and slaty, and bronze discoloration. Cyanosis is characterized by bluish discoloration of the skin and mucous membrane. Most frequently cyanosis is due to presence of abnormal amount of reduced hemoglobin. Cyanosis can be central, peripheral or mixed. Central cyanosis is due to intracardiac or intrapulmonary right-to-left shunt. The amount of desaturated hemoglobin is increased in central cyanosis and best recognized inspecting the buccal mucous membrane, tongue, and oropharyngeal mucous membrane.

Jaundice due to hyperbilirubinemia is occasionally seen in patients with severe right heart failure associated with congestive hepatopathy. Patients with portopulmonary hypertension may also have jaundice. A few abnormalities of skin, which can be associated with cardiovascular disorders are summarized in Table 12.

Examination of the Musculoskeletal System

The majority of congenital heart disease with musculoskeletal abnormalities is encountered in the pediatric population. In adult cardiology practices a few conditions, although distinctly uncommon, may be seen (Table 13).

Table 12: Skin abnormalities and cardiovascular disorders

- Cyanosis
 - Central—intracardiac and intrapulmonary right-to-left shunt
 - Peripheral—low cardiac output, increased peripheral oxygen extraction
- Methemoglobinemia—bluish discoloration of the skin
 - Hereditary (rare) and acquired (nitrate and nitrite toxicity)
- Jaundice—yellow discoloration
 - Prosthetic valve malfunction—hemolysis
 - Portopulmonary hypertension
 - Severe congestive hepatopathy
- Bronze discoloration—slaty color of the skin
 - Primary or secondary hemochromatosis
 - Atrial or ventricular arrhythmias
 - Restrictive cardiomyopathy, dilated cardiomyopathy
- Amiodarone skin toxicity—benign
- Butterfly rash of the face—lupus erythematosus
 - Valvular disease (Libman–Sack endocarditis)
 - Pulmonary arterial hypertension
- Malar flush of the face—
 - Severe mitral stenosis
 - Severe precapillary pulmonary hypertension
- Plantar and palmar keratosis and wooly hair—naxos disease
 - Arrhythmogenic right ventricular dysplasia

Contd...

Contd...

Table 12: Skin abnormalities and cardiovascular disorders

- Telangiectasia of lips, tongue and buccal mucous membrane—
 - Osler–Weber–Rendu syndrome
 - Arteriovenous malformations
- Xanthomatosis—tendon xanthoma, xanthoma in the palmar crease, with or without xanthelasma—familial hyperlipidemia
 - Premature coronary artery disease
- Cutaneous lentiginosis—LEOPARD syndrome
 - Conduction defects, congenital pulmonary stenosis
- Petechiae and purpuric skin rash—bacterial endocarditis
 - Valvular heart disease
- Blotchy cyanosis—carcinoid heart disease
 - Right-sided valvular heart disease
- Livid reticularis—reticular purplish skin rash
 - Lupus erythematosus
 - Valvular heart disease, pulmonary hypertension
 - Blue toes syndrome
 - Cholesterol emboli
- Atrophic skin lesions—necrobiosis diabetorum
 - Increased risks of cardiovascular disease
- Annular skin rash with clear center—Lyme disease
 - Heart block, myocarditis
- Tightening of the skin, flexion contractures of the fingers, telangiectasia-scleroderma, CREST syndrome
 - Pulmonary arterial hypertension

Table 13: Musculoskeletal abnormalities associated with cardiovascular disorders

- Marfan's syndrome
 - Aortic regurgitation, mitral regurgitation, aortic disease
- Ehler–Danlos syndrome
 - Mitral regurgitation, aortic disease
- Turner's syndrome
 - Coarctation of aorta
- Holt–Oram syndrome
 - Atrial septal defect
- Down syndrome
 - Ventricular septal defect, atrioventricular cushion defects
- Rheumatoid arthritis
 - Aortic regurgitation, heart block
- Ankylosing spondylitis
 - Aortic and mitral valve disease, heart block
- Clubbing of the fingers and toes
 - Congenital cyanotic heart disease, bacterial endocarditis
- Straight back syndrome
 - Mitral valve prolapse
- Clubbing of fingers and toes
 - Congenital cyanotic heart disease, bacterial endocarditis

Finger and toes should be examined for clubbing. The drumstick type of clubbing is seen in cardiovascular diseases, such as congenital cyanotic heart disease and bacterial endocarditis. Bacterial endocarditis can be also associated

with splinter hemorrhage, Janeway and Osler nodes and valvular regurgitations. Heberden's nodes, which are usually seen in the fingers, result from osteoarthritis and are not associated with cardiovascular disorder.

Measurement of Arterial Pressure

At present, in most institutions, automated techniques are used for the measurement of blood pressure. The various techniques of measuring blood pressure, their advantages and disadvantages, and pitfalls are discussed in the section on clinical hypertension.

Examination of the Jugular Venous Pulse

Careful examination of the jugular venous pulse and pressure provides information regarding the hemodynamic changes in the right side of the heart. It has been suggested that for estimation of jugular venous pressure, it is easier and preferable to examine the external jugular vein. However, it has also been suggested that if pulsation is present and visible, the examination of the internal jugular veins is preferable to that of the external jugular veins, as the internal jugular veins are in a direct line with the superior vena cava.

Estimation of Jugular Venous Pressure

The jugular venous pressure can be estimated by examining either external or internal jugular veins (Fig. 1). Conventionally, the upper torso is elevated to 30–40 degrees and the top of the venous pulsation is determined and 5 cm is added to the height assuming that right atrium is located 5 cm below the sternal angle (angle of Louis). However, it has been suggested that 10 cm should be added rather than 5 cm, if the torso is elevated to 45 degrees or greater. It should be appreciated that the external jugular venous pulse may not be recognized in patients with a fat and short neck. Kinking and thrombotic obstruction of the external jugular veins may also cause a spuriously higher central venous pressure.

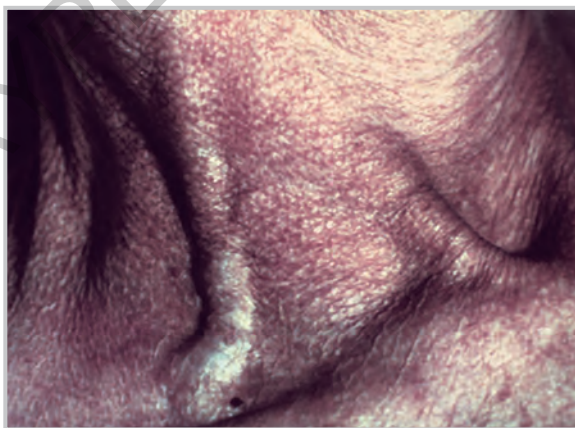


FIGURE 1: Courses of the external and internal jugular veins. The external jugular vein runs from lateral to the medial side of the neck across the sternocleidomastoid muscle. The internal jugular vein starts at the root of the neck in between the two heads of the sternocleidomastoid muscle and runs superiorly toward the angle of the jaw

Table 14: Causes of increased central venous and right atrial pressures

- Tricuspid valve obstruction—
 - Rheumatic tricuspid valve stenosis (usually associated with mitral and/or aortic valve disease)
 - Right atrial myxoma
 - Carcinoid heart disease
 - Neoplastic disease
- Right ventricular failure—
 - Systolic—
 - Primary-RV infarction
 - Secondary-pulmonary hypertension
 - Diastolic—
 - Right ventricular hypertrophy
 - Pericardial disease
 - Pericardial effusion
 - Constrictive pericarditis
- Primary tricuspid regurgitation—
 - Traumatic
 - Ruptured chordae
 - Ebstein's anomaly
 - Carcinoid heart disease
 - Rheumatic heart disease
 - Neoplastic disease
- Generalized volume overload—
 - Glomerulonephritis
 - Anemia
 - Large atriovenous communications
 - Isolated right ventricular volume overload
 - Atrial septal defects

Elevated central venous pressure suggests that the right atrial pressure is elevated. The upper limit of normal right atrial pressure in the supine position is about 7 mm Hg. Some of the causes of increased central venous and right atrial pressures are summarized in Table 14.

Jugular Venous Pulsations

The jugular venous pulse characters are best analyzed by examining the internal jugular veins. When the right atrial pressure waveforms are recorded during cardiac catheterization, three positive waves (a, c and v) and two negative waves (x and y descents) are recognized. The “a” wave occurs during atrial systole with increased right atrial pressure due to atrial contraction (Fig. 2). The “c” wave is related to bulging of the closed tricuspid valve into the right atrium at the beginning of the right ventricular systole. The “x” descent is primarily due to atrial relaxation with a fall in right atrial pressure. The downward descent of the tricuspid valve apparatus also contributes to the genesis of the “x” descent.

After complete relaxation of the right atrium and the nadir of “x” descent, the right atrial pressure rises as the systemic venous return to the right atrium continues. With the onset of right ventricular systole when the tricuspid valve closes, the right atrial pressure rises and the “v” wave begins. The right atrial pressure continues to rise as the systemic venous return to the right atrium continues.

The peak of the “v” wave coincides with the end of the right ventricular systole and can be recognized by timing with the down slope of the carotid pulse. The “y” descent begins with the opening of the tricuspid valve and continues during the rapid filling phase of the right ventricle with a concurrent pressure decline in the right atrium.

The hepatojugular reflux, also called abdominojugular reflux, is assessed during sustained abdominal compression by applying firm pressure over the abdomen for 10–15 seconds. Normally, during abdominal compression, the jugular venous pressure increases transiently by 1–3 cm.

Arterial Pulse

The contour, character and amplitude of the arterial pulses are related to left ventricular stroke volume, left ventricular velocity of ejection, aortic dP/dT,

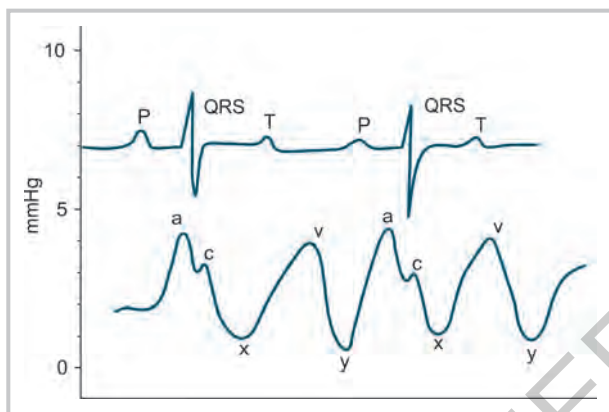


FIGURE 2: The schematic illustrations of right atrial pressure waveforms, which reveal three positive “a,” “c” and “v” waves and two negative “x” and “y” descent. The “a” wave occurs during atrial systole following P wave of the electrocardiogram. The “c” wave occurs at the onset of right ventricular systole when the closed tricuspid valve bulges into the right atrium. It occurs just after the QRS complex of the electrocardiogram. The “x” descent is related to atrial relaxation. The peak of the “v” wave coincides with the end of right ventricular systole. It coincides with the end of the T-wave of the electrocardiogram. The “y” descent is caused by the opening of the tricuspid valve and during the rapid filling phase (P: P wave; QRS: QRS complex; T: T wave. Normally the magnitude of “a” wave is greater than “v” wave and is less than 7 mm Hg)

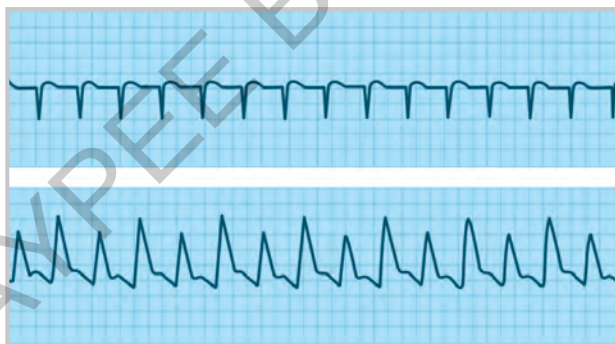


FIGURE 3: The characteristic “strong and weak alternating sequences of arterial pulses” in a patient with systolic heart failure are illustrated. The upper panel is an electrocardiogram showing normal sinus rhythm. The lower panel is directly recorded arterial pressure, showing alternating higher and lower arterial pressure

systemic vascular resistance, and the capacity and compliance of the arterial vascular system.

Examination of the volume and contours of arterial pulses provides important diagnostic clues regarding the underlying etiology of the pathophysiologic condition. Decreased amplitude may reflect reduced stroke volume irrespective of its etiology. Pulsus alternans in presence of regular rhythm indicates reduced left ventricular ejection fraction (Fig. 3).

Table 15: Cardiovascular physical examination**Palpable precordial impulses**

- Prominent systolic left parasternal impulse
 - RV failure
- Sustained LV apical impulse
 - Reduced LVEF
 - Increased LV mass
- Palpable PA impulse
 - Left-to-right shunt
 - Pulmonary hypertension
 - Pulmonary stenosis

(Abbreviations: LV: Left ventricle; RV: Right ventricle; LVEF: Left ventricle ejection fraction; PA: Pulmonary artery)

It should be appreciated that the absence of pulsus alternans does not exclude systolic heart failure. However, the presence of pulsus alternans is almost diagnostic of reduced left ventricular ejection fraction, although it is present in only about 10% of patients with chronic systolic heart failure. In patients with acute coronary syndrome, in approximately 25% of patients pulsus alternans can be appreciated.

The pulsus paradoxus is characterized by a fall in arterial pressure during inspiration more than 10 mm Hg. Normally systolic arterial pressure falls during inspiration by an average of 8–12 mm Hg. In cardiac tamponade there is a substantially greater decrease in the arterial pressure during inspiration. The magnitude of the pulsus paradoxus can be better appreciated if the blood pressure is recorded by the sphygmomanometer. The cuff pressure should be decreased slowly. The systolic pressure at expiration is noted. With a further reduction of cuff pressure, the systolic pressure during inspiration is noted and the difference between these two systolic blood pressures provides an estimate of pulsus paradoxus. More severe the tamponade, a greater fall in arterial pressure occurs during inspiration.

Examination of the Precordial Pulsation

Precordial cardiovascular pulsations are best appreciated with the patient in supine position with the upper torso elevated not more than 45 degree. During inspection, the left ventricular apical impulse is usually visible over the left fifth intercostal space medial to the anterior axillary line. In patients with severe volume overload of the left ventricle, such as due to severe mitral or aortic regurgitation, an accentuated left ventricular apical impulse along with pulsation of the entire precordium may be visible. The left ventricular apical impulse, also called apex beat, is examined with the patient in a partial left lateral decubitus position. A few clinical conditions and mechanisms of precordial impulses are summarized in Table 15.

Auscultation

The heart sounds are schematically illustrated in Figure 4. The analysis of the heart sounds should precede analysis of the heart murmurs. The high-frequency heart sounds, such as first (S1) and second (S2) and murmurs due to aortic and mitral regurgitation are better appreciated with the use of the diaphragm of the stethoscope. The lower frequency heart sounds, such as third (S3) and fourth (S4) and mid-diastolic rumbles are better heard with the bell of the stethoscope.

The S1 occurs just before the upstroke of the carotid pulse at the beginning of the isovolumic systole. At the bedside, S1 and S2 are best recognized by timing with carotid pulse upstroke and down stroke, respectively.

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