ww.jaypeebrothers.com , for detailed information on medicine books, visit our website www.jaypeebrothers.com





Handbook of Pulmonary 8

Handbook of Pulmonary & Critical Care Medicine

Editor-in-Chief **SK Jindal**

Foreword **Randeep Guleria**



Contents

1.	Introduction SK Jindal • History 1 • Respiratory System Function 2 • Ventilation-Perfusion (V/Q) Relationships 2 • Respiratory Defenses 3	1
2.	 Applied Respiratory Physics SK Jindal, VK Jindal Atom, Element, Molecule, and Compound 4 Molecular Movement 5 Atomic and Molecular Weights 5 Physical Properties of Gases 5 Gas Laws 8 Gas Solution and Tension 10 Vapor 10 Expression of Gas Volumes and Pressures 11 Flow of Gases 11 Deposition 13 Diffusion 13 	4
3.	 History and Physical Examination Prahlad Rai Gupta History Taking 15 History of Treatment 17 Physical Examination 18 	15
4.	Pulmonary Function TestsAN Aggarwal• Spirometry 22• Peak Expiratory Flow 25• Static Lung Volumes 26• Diffusing Capacity of Lungs 27• Exercise Testing 28• Other Tests 30	22
5.	Interpretation of Arterial Blood Gases and Acid-Base Abnormalities Aditya Jindal • Basic Concepts 33 • Overview of Acid-Base Pathophysiology in the Body 33 • Types of Acid-Base Disorders 34 • Anion Gap 36 • Acid-Base Disorders 37	32

6.	Tuberculosis: OverviewStuti Agarwal, Romica Latawa, Indu Verma• Route and Spread of Infection 40• Mycobacterial Groups 41• Mycobacterial Identification 42• Mycobacterial Drug Resistance 43	40
7.	 Immunology and Pathogenesis Madhur Kalyan, Krishna K Singh, Indu Verma Mycobacterium Tuberculosis Infection and Overview of Immunopathogenesis 44 Immune Responses to Tuberculosis 46 	44
8.	 Pulmonary Tuberculosis: Clinical Features and Diagnosis S Kashyap, Malay Sarkar Postprimary Pulmonary Tuberculosis 50 Symptoms and Signs 51 Tuberculosis in the Elderly 52 Miliary Tuberculosis 52 HIV and Tuberculosis 53 Pleural Effusion 53 Paradoxical Response 53 Physical Examination 53 Diagnosis of Tuberculosis 54 Extrapulmonary Tuberculosis 57 	50
9.	Molecular Diagnosis of Tuberculosis Rama Murthy Sakamuri, Mamta Kalra, Indu Verma, Suman Laal • Diagnosis of Tuberculosis: Beyond the Microscopy 58	58
10.	Management of TuberculosisD BeheraPrevention of Drug Resistance63Early Bactericidal Activity64Sterilizing Action64New Patients65Previously Treated Cases67	63
11.	 Prevention of Tuberculosis Rajesh N Solanki, Jaydeep Odhwani, Kumar Utsav Primordial Prevention 68 Primary Prevention 68 Secondary Prevention 70 Tertiary Prevention 73 	68
12.	 Extrapulmonary Tuberculosis AK Janmeja, PR Mohapatra, Deepak Aggarwal Diagnosis 74 Treatment of Extrapulmonary Tuberculosis 75 Lymph Node Tuberculosis 75 Pleural Effusion 76 Bone and Joint Tuberculosis 76 Central Nervous System Tuberculosis 79 	74

	 Abdominal Tuberculosis 81 Genitourinary Tuberculosis 82 Skin Tuberculosis 83 Pericardial Tuberculosis 83 Hepatic Tuberculosis 84 	
13.	Multidrug Resistant Tuberculosis Surendra K Sharma, Dinkar Bhasin • Definitions 86 • Diagnosis 87 • Management 87	86
14.	 Treatment of Tuberculosis in Special Situations Rajendra Prasad, Nikhil Gupta Treatment of Tuberculosis in Pregnancy and Lactation 91 Treatment of Tuberculosis in Renal Insufficiency 92 Treatment of Tuberculosis in Liver Disease 93 	91
15.	Tuberculosis and Human Immunodeficiency Virus InfectionAditya Jindal, SK Jindal• Epidemiology 95• Pathogenesis 95• Clinical Features 97• Diagnosis 98• Management 98	95
16.	Nontuberculous Mycobacterial Diseases PS Shankar, SK Jindal • Classification 102 • Human Disease 103 • Summary 105	102
17.	 Community-acquired Pneumonia Charles Feldman, Ronald Anderson Microbial Etiology 106 Risk Factors 107 Pathogenesis with Particular Reference to the Pneumococcus 107 Diagnostic Testing 108 Prognosis 109 Treatment 110 	106
18.	Pulmonary Fungal InfectionsArunaloke Chakrabarti• Types of Infections112	112
19.	 Nosocomial Pneumonia Vishwanath Gella, SK Jindal Definitions 118 Pathogenesis 119 Prevention of Hospital-acquired Pneumonia and Ventilator-associated Pneumonia 119 Diagnosis 121 Treatment 123 	118

Titality	aboolt of Failhonary and official outo inculorito	
20.	Lung Abscess <i>C Ravindran, Jyothi E</i> • Epidemiology 125 • Classification 125 • Etiology 126 • Pathogenesis 126 • Pathology 126 • Clinical Features 127 • Laboratory Diagnosis 127 • Complications 128 • Treatment 129 • Prognosis 130	125
21.	Bronchiectasis and Cystic Fibrosis David Honeybourne 131 Bronchiectasis 131 • Pathology 131 • Physiology 132 • Etiology 132 • Symptoms and Signs 132 • Diagnosis 133 • Microbiology 134 • Treatment 134 • Complications 136 • Prognosis 136 • Diagnosis 137 • Clinical Features 138 • Infections and Treatment 138	131
22.	 Anaerobic Bacterial Infections of the Lungs and the Pleura Ashok Shah, Chandramani Panjabi Pathophysiology 141 Other Predisposing Factors 141 Natural History and Clinical Classification 142 Anaerobes and Upper Respiratory Syndromes 143 Clinical Features 144 Laboratory Diagnosis 145 Treatment 145 	141
23.	 Bronchial Asthma Epidemiology SK Jindal Epidemiology 148 Disease Burden 149 Risk Factors of Asthma 149 	147
24.	Airway Inflammation and Remodeling Ruby Pawankar, Shu Hashimoto, Miyuki Hayashi, Shingo Yamanishi, Manabu Nonaka • Chronic Inflammation in Allergic Rhinitis and Asthma 154	153

• Remodeling in Asthma 156

		Contents	xxix
25. As Lie •	sthma Diagnosis esel D'silva, Parameswaran Nair Clinical Diagnosis 158 Tests for Diagnosis and Monitoring 159 Diagnostic Challenges 162	158	
26. Co Sic •	ontrol and Management of Stable Asthma dney S Braman, Gwen Skloot Goals of Asthma Treatment 165 Essential Components of Asthma Care 166	165	
27. Ac Aa •	cute Asthma Exacerbations <i>ditya Jindal</i> Triggers Causing Exacerbations 171 Diagnosis and Evaluation of Severity 172 Management 173	171	
28. Al Vil •	l lergen Desensitization kram Jaggi Definition 177 Mechanisms of Allergen Immunotherapy 177	177	
29. Pa Bh • •	atient Education in Asthma barat Bhushan Sharma, Virendra Singh Goals of Asthma Education Programs 184 Benefits of Asthma Education Programs 185 Methods and Settings 185 Asthma Education Program Components 185 Problems in Patient Education 186	184	
30. Pł Nu Co • • • • •	narmacotherapy of Bronchial Asthma asrat Shafiq, Samir Malhotra pontrollers: Anti-inflammatory Agents 187 Corticosteroids 187 Leukotriene Receptor Antagonists 189 Mast Cell Stabilizers 191 elievers—Bronchodilators 192 Beta-2 Adrenergic Agonists 192 Methylxanthines (Xanthines) 196 Anticholinergic Agents 198	187	
31. Al Ritu • • •	Ilergic Bronchopulmonary Aspergillosis tesh Agarwal Epidemiology 200 Pathogenesis 201 Pathology 201 Clinical Features 202 Laboratory Findings 202 Diagnosis and Diagnostic Criteria 205 Management 205 Allergic Bronchopulmonary Mycosis 207	200	

32.	 Burden of Chronic Obstructive Pulmonary Disease Monica Barne, Sundeep Salvi Mortality due to Chronic Obstructive Pulmonary Disease 208 Prevalence of Chronic Obstructive Pulmonary Disease 209 Disability Adjusted Life Years (DALYS) due to Chronic Obstructive Pulmonary Disease 211 	208
33.	 Risk Factors for Chronic Obstructive Pulmonary Disease Sneha Limaye, Sundeep Salvi 213 Tobacco Smoking 213 Environmental Tobacco Smoke 214 Household Air Pollution 214 Mosquito Coil Smoke 215 Outdoor Air Pollution 215 Chronic Obstructive Pulmonary Disease Associated with Occupational Exposures 215 Chronic Obstructive Pulmonary Disease Associated with Pulmonary Tuberculosis 216 Chronic Asthma 216 Genetic Factors 216 Socioeconomic Status 217 	213
34.	 Pathophysiology of Chronic Obstructive Pulmonary Disease Bill Brashier, Sundeep Salvi, Baishakhi Ghosh Inflammatory Changes 218 New Insights into Small Airway Obstruction 221 Chronic Obstructive Pulmonary Disease as a Disease of Systemic Inflammation 221 	218
35.	 Systemic Manifestations and Comorbidities of Chronic Obstructive Pulmonary Disease SK Jindal, PS Shankar Pathogenesis 223 Systemic Manifestations 224 Therapeutic Considerations 228 	223
36.	 Treatment of Chronic Obstructive Pulmonary Disease Peter J Barnes Risk Factors and their Prevention 229 Pharmacotherapy 230 Supplementary Oxygen 235 Antibiotics 236 Other Drug Therapies 236 Nonpharmacological Treatments 238 	229
37.	Acute Exacerbations of Chronic Obstructive Pulmonary Disease Raja Dhar, AG Ghoshal • Copd Exacerbation 239	239
38.	 Pulmonary Rehabilitation Rachael A Evans, Roger S Goldstein Role and Definition of Pulmonary Rehabilitation 245 Changing Pulmonary Rehabilitation Population— Whom to Refer? 246 	245

		Contents	xxxi
 Outcome Measures 247 Core Components of a Pulmonary Rehabilitation Program Maintenance 249 Mobility Aids 250 Rehabilitation Team 250 Setting 250 Exacerbations 250 Performance Enhancement 250 	248		
 39. Bullous Lung Diseases Aditya Jindal, Gyanendra Agrawal Pathogenesis 252 Etiology 253 Clinical Presentation 254 Radiologic Features 254 Pulmonary Function Tests 254 Natural History 255 Complications 255 Treatment 255 		252	
 40. Upper and Central Airways Obstruction VR Pattabhi Raman Physiological Considerations 257 Clinical Features 258 Diagnosis 258 Acute Upper Airway Obstruction 259 Chronic Upper Airway Obstruction 261 Therapeutic Considerations 264 		257	
 41. Interstitial Lung Diseases Nagarjuna V Maturu, Dheeraj Gupta Etiology and Classification 266 Epidemiology 268 Pathology 268 Pathogenesis 268 Diagnostic Approach 269 Treatment 271 Acute Exacerbation of ILD 273 Prognosis 274 		266	
 42. Idiopathic Interstitial Pneumonias H Shigemitsu, Ngozi Orjioke, Carmen Luraschi-Monjagatta Diagnosis 275 Histological Features 276 Idiopathic Pulmonary Fibrosis 276 Nonspecific Interstitial Pneumonia 278 Desquamative Interstitial Pneumonia 279 Respiratory Bronchiolitis-associated Interstitial Lung Disease 280 Cryptogenic Organizing Pneumonia 281 Acute Interstitial Pneumonia 282 Lymphoid Interstitial Pneumonia 282 Idiopathic Pleuroparenchymal Fibroelastosis 283 Unclassifiable Idiopathic Interstitial Pneumonia 284 		275	

43.	SarcoidosisDheeraj Gupta, Sahajal Dhooria, Om P Sharma• Etiology and Risk Factors 285• Pathogenesis and Immunology 286• Pathology 286• Clinical Features 287• Diagnosis 289• Treatment 291• Prognosis and Mortality 293	285
44.	 Pulmonary Eosinophilic Disorders Subhash Varma, Aditya Jindal Eosinophils 294 Pulmonary Eosinophilic Disorders 296 Approach to Diagnosis and Conclusion 304 	294
45.	Infiltrative and Deposition Diseases Pralay Sarkar, Arunabh Talwar • Pulmonary Amyloidosis 305 • Lysosomal Storage Disorders 307	305
46.	 Bronchiolitis Gyanendra Agrawal, Dheeraj Gupta General Features of Bronchiolar Disorders 312 Clinical Presentations 312 Practical Approach for Diagnosis of Bronchiolar Disorders 315 	312
47.	 High-altitude Problems Ajay Handa Physical Changes with Altitude 317 Physiological Adaptation to High Altitude 317 Specific Altitude-related Illnesses 318 Effects of High Altitude on Existing Lung Diseases 320 	317
48.	Aviation and Space Travel Ajay Handa • Respiratory Physiology with Altitude 322 • Preflight Assessment 323 • Prescribing In-flight oxygen 324 • Space Travel 325 • Microgravity and Weightlessness 325	322
49.	Lung Disease in Coal Workers Harakh V Dedhia, Daniel E Banks • Clinical Features of Coal Dust Exposure 327 • Pathology of Coal Worker Pneumoconiosis 329 • Management of CMDLD 330	327
50.	Silicosis PS Shankar, SK Jindal Occupational Exposure 333 Pathogenesis 334 Forms of Silicosis 334 Clinical Features 336	333

	Contents	xxxi
 Diagnosis 336 Prognosis 339 Treatment 339 		
 51. Berylliosis PS Shankar Acute Beryllium Disease 340 Chronic Beryllium Disease 340 Pathogenesis 341 Clinical and Radiological Features 341 Diagnosis 342 Treatment 342 	340	
 52. Metal-induced Lung Disease Dilip V Maydeo, Nikhil C Sarangdhar Types 344 Epidemiology 344 Pathogenesis 345 Types of Immune Responses in Metal-induced Lung Disease 345 Clinical Presentation and Diagnosis 345 Approach 346 Treatment 347 	344	
 53. Health Risks of Asbestos Fiber Inhalation Daniel E Banks, Harakh V Dedhia Asbestosis 349 Asbestos Fibers and the Pleural Space 350 Diffuse Pleural Thickening: Fibrosis of the Visceral Pleura 350 Pleural Plaques: Fibrosis of the Parietal Pleura 351 Malignant Mesothelioma 352 Lung Cancer 353 	348	
 54. Occupational Asthma PS Shankar, G Gaude Agents Causing Occupational Asthma 355 Pathogenetic Mechanisms of Occupational Asthma 357 Diagnosis 357 Management 359 Prognosis 360 	354	
 55. Hypersensitivity Pneumonitis PS Shankar Etiology 361 Pathogenesis 362 Pathology 363 Clinical Presentation 363 Diagnosis 364 Management and Prevention 365 Prognosis 366 	361	
 56. Toxic Inhalations and Thermal Lung Injuries VK Vijayan, N Goel, R Caroli Determinants of Inhalational Lung Injury 367 Clinical Presentations of Inhalational Injury 368 	367	

	 Systemic Illnesses from Inhaled Toxins 371 Smoke Inhalation Lung Injury 372 Management 372 	
57.	 Drug-induced Respiratory Disease William J Martin Jr Drugs Associated with Respiratory Toxicity 373 Diagnosis and Management of Drug-induced Respiratory Disease 376 	373
58.	 Epidemiology and Etiopathogenesis of Lung Cancer Nagarjuna V Maturu, Navneet Singh Lung Cancer in India 378 Histological Patterns 378 Risk Factors 379 Molecular Biology of Lung Cancer 383 	378
59.	Pathology of Lung TumorsAmanjit Bal, Ashim Das• Preinvasive Lesions 385• Classification of Lung Cancer 386• Epithelial Tumors 386• Neuroendocrine Lesions of the Lung 388• Staging of Lung Tumors 389	385
60.	Lung Cancer: Clinical Manifestations Javid Ahmad Malik • Local Manifestations 391 • Metastatic Manifestations 393 • Endocrine Syndromes 396 • Neurological Syndromes 397 • Hematological Syndromes 399 • Skeletal 399 • Miscellaneous Syndromes 400	391
61.	 Diagnosis and Staging of Lung Cancer Nagarjuna V Maturu, Ajmal Khan, Navneet Singh Diagnosis of Lung Cancer 401 Staging of Nonsmall-cell Lung Cancer 403 Staging of Small-cell Lung Cancer 405 	401
62.	 Approach to Management of Lung Cancer in India Navneet Singh, Nagarjuna V Maturu, Digambar Behera Treatment of Lung Cancer 407 Palliation 413 	407
63.	 Targeted Agents for Nonsmall Cell Lung Cancer Nagarjuna V Maturu, Navneet Singh Epidermal Growth Factor Receptor–Tyrosine Kinase Inhibitors 415 Anaplastic Lymphoma Kinase Inhibitors 418 Vascular Endothelial Growth Factor Inhibitors 419 	415
64.	 Hematopoietic and Lymphoid Neoplasm of Lungs Gaurav Prakash, Pankaj Malhotra Lymphomas 420 Lymphomatoid Granulomatosis 425 	420

		Contents	xxxv
	• Secondary Involvement of Lung by Other Systemic Hematopoietic and Lymphoid Disorders 426		
65.	 Solitary Pulmonary Nodule Alladi Mohan, B Vijayalakshmi Devi, Abha Chandra Terminology 428 Etiology 429 Clinical Evaluation 430 Imaging Studies 430 Management 435 	428	
66.	 Mediastinal Disorders Arjun Srinivasan, SK Jindal Imaging of Mediastinum 437 Diseases of Mediastinum 438 Tumors and Cysts of Mediastinum 441 	436	
67.	Diseases of the Chest Wall Balamugesh T • Kyphoscoliosis 447 • Thoracoplasty 449 • Pectus Excavatum 450 • Pectus Carinatum 450 • Ankylosing Spondylosis 450 • Obesity 451 • Flail Chest 452 • Miscellaneous Conditions 452	447	
68.	 Diffuse Alveolar Hemorrhage Stagaki E, Karakontaki F, Polychronopoulos V Diffuse Alveolar Hemorrhage Syndromes 454 Causes 457 Other Causes of Diffuse Alveolar Hemorrhage 464 	454	
69.	 Pulmonary Hypertension: A Third World Perspective Lakshmi Mudambi, Zeenat Safdar Clinical Features 467 Physical Examination 468 Diagnostic Evaluation 468 Pathophysiology 470 Management 471 	466	
70.	 Pulmonary Thromboembolism Devasahayam J Christopher, Richa Gupta Pathophysiology 473 Risk Factors 475 Clinical Features 475 Diagnosis 476 Management 478 	473	
71.	 Pulmonary Vascular Malformations Gautam Ahluwalia Hereditary Hemorrhagic Telangiectasias or Rendu–Osler–Weber Syndrome 483 	482	

	 Pathogenesis 483 Clinical Features 484 Investigations 484 Management 485 Other Pulmonary Vascular Malformations 485 	
72.	 Approach to Respiratory Sleep Disorders Ruchi Bansal Sleep History 488 Physical Examination 489 Nocturnal Polysomnography 490 Out-of-center Sleep Testing 490 Sleep Questionnaires 491 Respiratory Disorders During Sleep 491 	488
73.	Respiratory Sleep DisordersAditya Jindal• Classification 493	493
74.	Respiratory FailureAbinash Singh Paul, Ritesh Agarwal• Classification 499• Mechanisms 500• Clinical Manifestations 502• Diagnosis 502• Treatment 503	499
75.	Acute Respiratory Distress Syndrome Jean I Keddissi, D Robert McCaffree • Etiology 507 • Clinical Picture 508 • Pathophysiology 508 • Management 509 • Prognosis and Outcome 513	507
76.	 Sepsis Sean E Hesselbacher, Walter G Shakespeare, Kalpalatha K Guntupalli Pathogenesis 514 Clinical Features and Evaluation 515 Prognosis 516 Management 517 Goals of Care 520 Special Considerations 521 	514
77.	Nonpulmonary Critical Care Liziamma George, Mark Astiz Gastrointestinal Disease in Critical Care 522 Hematology in Critical Care 526 Renal Disease in Critical Care 528 Endocrine Emergencies in Critical Care 529 Neurological Disorders in Critical Care 531	522

	Contents	xxxvii
 78. Critical Care in Nonpulmonary Conditions: Poisoning, Envenomation, and Environmental Injuries Dhruva Chaudhry, Inderpaul Singh Sehgal Poisoning 535 Envenomation 541 Environmental Injuries 544 	535	
 79. Pulmonary Hypertension in the Intensive Care Unit Charles Peng, Roxana Sulica Right Heart in Health and Disease 548 Pulmonary Hypertension in the Critically III Patient 549 Right Ventricular Failure in Patients with Preexisting Pulmonary Arterial Hypertension 551 Management of the Pulmonary Arterial Hypertension Patient with Decompensated Right Heart Failure 552 Perioperative Management of the Patient with Pulmonary Arterial Hypertension 554 	548	
 80. Mechanical Ventilation: General Principles and Modes GC Khilnani, Vijay Hadda Indications of Mechanical Ventilation 555 Basic Aspects of Mechanical Ventilation 556 Modes of Mechanical Ventilation 557 Newer Modes of Mechanical Ventilation 565 Initiating Mechanical Ventilation 567 Complications of Mechanical Ventilation 568 	555	
 81. Noninvasive Ventilation GC Khilnani, Vijay Hadda Technical Aspect of Noninvasive Ventilation 570 Steps to Successful Provision of Noninvasive Positive Pressure Ventilation 573 Clinical Uses of Noninvasive Positive Pressure Ventilation: Evidence and Recommendations 575 	570	
 82. Blood Gas Monitoring Inderpaul Singh Sehgal, Ritesh Agarwal Arterial Sampling 579 Arterial Cannulation 580 Noninvasive Blood Gas Monitoring 581 	579	
 83. Cutaneous Capnography Preyas Vaidya, Arvind H Kate, Prashant Chhajed Site for Measurement 585 Factors Influencing PcCO₂ Monitoring 586 Medical Applications of PcCO₂ Monitoring 586 Clinical Settings for the Use of Cutaneous Capnography 587 	585	
 84. Nutritional Management and General Care in the Intensive Care Unit Inderpaul Singh Sehgal, Navneet Singh Malnutrition in Critical Illness 591 Refeeding Syndrome 593 	591	

	 Assessment of Nutritional Status in Critically III Patients 593 Goals and Principles of Nutritional Support 593 Timing of Initiation of Nutritional Support 594 Route of Administration of Nutritional Support 594 Quantity and Volume of Nutrition Support 595 Delivery of Enteral Nutrition and Its Determinants 596 General Care in ICU 598 	
85.	 Management of Complex Airways Diseases Rubal Patel, Atul C Mehta Difficult Airway Situations 601 Indications for Artificial Airway 602 Techniques 605 Alternative Airway Techniques 607 	601
86.	 Analgesia and Sedation in the ICU Karan Madan, Ritesh Agarwal Teamwork (Multidisciplinary Management) and Patient-focused Care 609 Initial Evaluation and Medication Reconciliation 610 Consequences of Off-target Sedation and Analgesia 610 Assessment of Pain, Sedation, and Agitation in the ICU 610 Objective Measurement of the Cerebral Activity in the ICU 612 Management of Analgesia and Sedation in the ICU 612 Recent Developments and Novel Approaches 618 	609
87.	 Weaning from Mechanical Ventilation Ajmal Khan, Ritesh Agarwal Pathophysiology of Weaning 622 Outcome of Weaning 623 Assessment for Weaning 623 Techniques of Weaning 624 	621
88.	 Hyperbaric Oxygen Therapy PS Tampi, SK Jindal Rationale of Hyperbaric Oxygen 628 Other Physiological Effects of Hyperbaric Oxygenation 629 Beneficial Effects of HBO₂ 629 Mechanism of Action of HBO₂ 629 Indications 630 HBO₂ in Pediatric Age Group 633 Potential New Indications 633 Contraindications 633 Complications 634 	628
89.	 Pleura: Anatomy and Physiology Srinivas Rajagopala Anatomy of the Pleura 635 Development of the Pleural Membranes 636 Histology of the Pleura 636 Pleural Fluid: Normal Volume and Cellular Contents 636 Physiology and Pathophysiology of Pleural Fluid Turnover 637 Physiological Changes with a Pleural Effusion 638 	635

• Physiological Changes with Pneumothorax 638

	Pleural Manometry 638Pleural Ultrasound 639	
90.	Tubercular Pleural EffusionPranab Baruwa, Kripesh Ranjan Sarmah• Pathology and Pathogenesis 640• Clinical Features 641• Diagnosis 641• Management 645• Complication of TB Pleural Effusion 646	640
91.	 Parapneumonic Effusion and Empyema Devasahayam J Christopher Definitions 648 Pathogenesis 649 Epidemiology 649 Bacteriology 650 Clinical Features and Diagnosis 650 Treatment 652 	648
92.	Malignant Pleural Effusions and PleurodesisSrinivas Rajagopala• Etiology of Malignant Effusions 655• Pathogenesis of Metastasis and Effusions 656• Clinical Presentation 656• Radiological Findings 657• Diagnosis 657• Management 659• Prognosis 663	655
93.	 Pneumothorax Uma Devraj, GA D'Souza Definitions 664 Pathophysiology 665 Resolution of Pneumothorax 665 Etiology 665 Laboratory Investigations and Diagnosis 667 Recurrence Rates 668 Treatment 668 	664
94.	Malignant Pleural MesotheliomaArun S Shet, Girish Raju, GA D'SouzaEpidemiology674Pathogenesis675Pathology675Clinical Presentation676Diagnostic Approach676Treatment676	674
95.	Pulmonary Involvement in Connective Tissue DiseasesOm P Sharma, Aditya Jindal• Rheumatoid Arthritis• Systemic Sclerosis• Sjögren's Syndrome• 686	680

xxxix

Contents

	 Systemic Lupus Erythematosus 687 Dermatomyositis and Polymyositis 691 Ankylosing Spondylitis 692 Mixed Connective Tissue Disease 693 	
96.	 Pulmonary Manifestations of Other System Diseases Ajmal Khan, SK Jindal Cardiovascular Diseases 695 Neuromuscular Diseases 697 Endocrine Disorders 703 Gastrointestinal Diseases 706 Hepatic Disorders 709 Renal Diseases 712 	695
97.	 Pulmonary Involvement in Tropical Diseases Sanjay Jain, SK Jindal Malaria 714 Typhoid 716 Leptospirosis 717 Dengue 718 Amebiasis 719 	714
98.	 Pulmonary Diseases in Pregnancy Lakhbir K Dhaliwal, Preeti Verma, Umesh Jindal Dyspnea During Pregnancy 722 Asthma in Pregnancy 722 Pneumonia in Pregnancy 726 Tuberculosis and Pregnancy 730 Pulmonary Thromboembolism 732 Pregnancy-specific Problems 734 	721
99.	 Rare Lung Diseases Sanjeev Mehta, PS Shankar Pulmonary Alveolar Phospholipoproteinosis 737 Pulmonary Calcification and Ossification Syndromes 740 Pulmonary Alveolar Microlithiasis 742 	737
100.	 Ethics in Respiratory Care Basil Varkey Ethics Education 744 Ethics in End-of-life Care 749 A Conceptual Model for Patient Care 751 	744
101.	 End-of-Life Care Jeba S Jenifer, SK Jindal Components 754 Common Symptoms 754 Diagnosing Dying and Providing Terminal Care 757 End-of-life Care in the Intensive Care Unit 758 	753
Index		761

CHAPTER 4

Pulmonary Function Tests

AN Aggarwal

INTRODUCTION

There is as yet no single test that can provide sufficiently detailed information on all aspects of lung function. Instead, depending on the clinical scenario, one must do one or more procedures to answer a particular question. Further, the available options vary greatly in terms of ease of conducting the test, equipment and technician requirements, test performance characteristics, and procedure cost.

SPIROMETRY

Spirometry is the most common and most widely used lung function test, although its true potential still needs to be realized. One needs to pay careful attention to follow the standard procedures while performing and interpreting the test. Because the residual volume in lungs cannot be exhaled, spirometric measurements are limited to the vital capacity and its subdivisions (Fig. 4.1).

Indications and Contraindications

The most common indication for doing the test is a functional evaluation of patients with lung disease. The presence of spirometric abnormalities, as well as the degree of impairment, provides useful information about the disease severity and pulmonary reserve of the patient. Serial measurements can provide information about disease progression, as well as response to prescribed treatment. The test also has an important role in clinical trials. Spirometry is also used as a screening tool for studies in epidemiologic surveys, and to screen at-risk populations for subclinical disease (for example,



Fig. 4.1: Various lung volumes and capacities in relation to the spirometry tracing. Note that residual volume cannot be determined through conventional spirometry.

preoperative assessment, or detecting chronic obstructive pulmonary disease (COPD) among asymptomatic smokers). The test is also utilized in occupational setting, both for detecting work-related respiratory disorders, and for disability assessment in symptomatic people (for example, as part of compensation procedures). Finally, spirometry is an important research tool for understanding pathophysiology and temporal course of several diseases.

Any benefit from the information obtained through this test should be carefully weighed against patient discomfort and risk. The test is better avoided in pregnant and severely dyspneic patients. It should also not be carried out in patients where pressure swings due to a forced expiratory maneuver can worsen existing conditions (such as ruptured tympanic membrane, bronchopleural fistula, ongoing hemoptysis, etc.). Uncooperative patients, and those on life support systems, should also not undergo the test.

Equipment

A wide range of apparatus, ranging from handheld portable devices to large equipment, and from predominantly manual to completely automated systems, is available to perform spirometry. Although many factors such as cost, patient load, clinical requirements, etc. determine the choice of machine, it is important to use one that confirms to some minimum technical specifications necessary to obtain valid results.

Most commercially available spirometers nowadays are computerized systems that employ a transducer to convert a mechanical signal to an electrical one, and display the output in a fashion understood by the operator. These equipment can be divided into two broad categories: (1) volume displacement spirometers and (2) flow-sensing spirometers. The former work with volume as the primary output, and flow is a derived parameter. Such machines can have

a water seal, a dry-rolling seal, or a bellows type design. Flow sensing devices can either be electronic turbines, or use electronic pneumotachometers (sensors that estimate airflow from the change in pressure occurring across a suitable resistance), which in turn can have a flow-resistive, a heated wire, or an ultrasonic design. As opposed to volume displacement spirometers, these machines measure flow as the primary signal, which is time-integrated to yield volume estimates.

Reference Values

The basic purpose of pulmonary function testing is to identify persons with abnormal lung function. To know what is abnormal, we must first define what is normal. Predicted normal values can be obtained from studies carried out in healthy subjects. They are usually in the form of a regression equation describing the predicted value as a function of gender and anthropometric data (e.g. height, weight, etc.), and differ greatly with ethnicity. Any value below the predicted normal is not necessarily reduced, since the normal value is a range rather than a fixed point. This introduces the concept of "lower limit of normal" or LLN, which can be defined in several ways. The simplest (and most widely used) method is to use a fixed percentage of predicted value. For example, a value less than 80% of predicted FEV1 can be considered abnormal. However, there is very little statistical or physiological basis for such an approach. A more valid approach is to use lower 95% confidence limits of the regression equation, or subtract 1.645 times the standard error of estimate of the regression equation from the predicted value, to define the LLN. Any value below the corresponding LLN is considered abnormal. It is very important to use norms derived from individuals largely similar to the patients being generally tested at any pulmonary function laboratory. Therefore standard Caucasian norms, often incorporated into spirometer softwares, should be avoided, and locally appropriate reference equations preferred wherever available.

Interpretation and Patterns in Common Disorders

Interpreting lung function data is not just about looking at numbers generated by the spirometer. Both the volume-time curve and the flow-volume loop must also be evaluated with regard to their technical quality, size and shape, and various components, before making a final interpretation. Often such graphical analysis provides additional important information not obtainable from the numerical data. If available, the postbronchodilator graphs should also be similarly evaluated and compared to baseline curves. The clinical data provided in the requisition form is equally important in helping to reach any conclusion, especially in borderline situations.

Broadly, the interpretation of spirometric data involves only three numerical variables: FEV1, VC and FEV1/VC. The largest observed values of FEV1 and VC available from among at least three acceptable and reproducible tests should be used as the key parameters for interpretation, even if these individual observations are derived from different test maneuvers. If both forced and relaxed VC maneuvers have been performed, the larger value of VC

amongst the FVC and SVC measurements should be used for interpretation. The large numbers of other variables, often available from computerized spirometer outputs, usually provide no additional information, and are best excluded from a standard interpretative algorithm.

Any spirometry record with normal FEV1, VC and FEV1/VC (i.e. all values more than their corresponding LLN values) should be interpreted as normal. Any spirometry record with FEV1/VC value below its predicted LLN should be interpreted as having an obstructive abnormality. This approach is superior to the use of fixed cut-offs in correctly identifying patients with airflow limitation, especially among the elderly.

Any spirometry record with a normal FEV1/VC (i.e. value above corresponding LLN), coupled with a reduced VC (i.e. value below corresponding LLN), is suggestive of a restrictive abnormality. In situations where statistically valid LLN figures are not available (or not practical to use, as in field settings), observed VC ratio less than 80% of predicted value is often used to define reduction in VC. Restrictive defects are common in conditions with loss of functioning lung parenchyma (e.g. diffuse parenchymal lung diseases, lung collapse/atelectasis, pneumonia, after lung resection). Such defects are also observed in neuromuscular diseases (due to reduction in generation of force needed for a FVC maneuver) as well as disorders of chest wall and pleura (e.g. massive pleural effusion, pleural fibrosis, obesity, and kyphoscoliosis). True restriction is defined as reduction in total lung capacity. A mixed (obstructive plus restrictive) defect also cannot be diagnosed solely based on spirometry. A disproportionately low VC in face of a reduced FEV1/ VC can either represent air trapping (with consequent increase in RV at the expense of VC, as in severe emphysema), or a true reduction in TLC (as in COPD with pneumonia). There is no universally accepted scheme of severity categorization.

The flow-volume loop may also provide a clue to underlying pathology. A small and concave or scooped curve suggests obstructive disorder. A small curve with steep slope suggests restriction. A small and flat curve suggests central airway obstruction. In disorders with variable intrathoracic obstruction, only the expiratory component of the loop is flat, whereas in disorders with variable extrathoracic obstruction, only the inspiratory component is flat. Both components are flat in lesions causing fixed airway obstruction.

Bronchodilator responsiveness (BDR) is considered to be present if the increase in FEV1 and/or VC (15–30 minutes after inhalation of 400 μ g salbutamol) in the postbronchodilator study is both more than 12% and more than 200 mL over baseline values. Although an oversimplification, patients with asthma tend to have BDR much more frequently than those with COPD. It must be noted that lack of BDR does not necessarily imply poor clinical response to bronchodilators in either condition.

PEAK EXPIRATORY FLOW

Peak expiratory flow (PEF) is defined as highest flow achieved from a maximum forced expiratory maneuver started without hesitation from a position of

maximal lung inflation. It can be measured either as a part of the spirometry procedure on the same instrument (with values derived from the flow volume curve), or separately using peak flow meters. The first meter specifically designed to measure this index of lung function was developed more than 50 years ago (Wright meter). Subsequently, a more portable, lower cost version (the "Mini-Wright" peak flow meter) was developed, and other designs and copies have since then become available across the world.

Although PEF is fairly well reproducible for an individual, the normal range of PEF in healthy individuals is rather wide. As a result, predicted values of PEF cannot be used to detect lung disease, since there is substantial overlap between values in patients with lung diseases and normal persons. Further, since PEF recordings are both flow and volume dependent, they tend to get reduced in both obstructive and restrictive disorders. Hence in general notion that diminished PEF is a marker of airway obstruction is also not correct. While a normal PEF can reliably rule out airway obstruction, a low PEF does not necessarily indicate the same. The degree of reduction in PEF does not correlate well with the severity of obstruction described by the degree of reduction in FEV1. PEF measurements generally underestimate the degree of airway obstruction, as determined from FEV1 measurements.

STATIC LUNG VOLUMES

Since spirometry cannot measure RV, it is not possible to determine FRC and TLC from this test. Other techniques are needed for the purpose. These methods are generally based on principles in which airflow velocity plays no role (in contrast to spirometry), and hence the term "static lung volumes" is often used for these measurements. Three techniques may be used: (a) open circuit nitrogen washout, (b) closed circuit inert gas dilution, and (c) whole body plethysmography. Determination of static lung volumes is helpful in ascertaining true restrictive physiology, and differentiating between obstructive and restrictive disorders. Comparison between TLC estimated through gas dilution and plethysmographic methods can also quantify the extent of air trapping within the lungs.

Whole Body Plethysmography

In contrast to gas dilution techniques, plethysmography measures the total volume of air in the thoracic cavity, including gas trapped in bullae and other noncommunicating spaces (e.g. air within pleura or esophagus). Plethysmographically determined FRC is therefore often referred to as thoracic gas volume (TGV). Although several different types of body plethysmographs are available, the "volume constant" type is the most widely used.

Interpretation

A decrease in TLC is diagnostic of a restrictive defect. In parenchymal restriction (e.g. lung fibrosis), RV and TLC are reduced proportionately,

resulting in a normal RV/TLC ratio. In extrapulmonary restriction (e.g. chest wall or neuromuscular disorders), RV is usually normal (or sometimes even increased), resulting in an increased RV/TLC ratio. On the other hand, TLC might be increased in acromegaly, or in conditions like emphysema, as a result of air trapping. An increase in RV/TLC ratio, with an obstructive defect on spirometry, is a good indicator of air trapping. In conditions characterized by non-communicating air in the lungs (e.g. emphysema, bullae, etc.), whole body plethysmography provides a better estimate of the lung volume, since gas dilution techniques measure only the volume of air that is freely exchanged during breathing. In fact, the difference in volumes calculated by the two techniques may provide some indication to the volume of noncommunicating air present in the lungs. Estimation of static lung volumes is also necessary to diagnose mixed obstructive-restrictive defects, with a combination of reduced FEV1/VC ratio and reduced TLC.

DIFFUSING CAPACITY OF LUNGS

Measurement of pulmonary diffusing capacity allows us to assess the ability of lungs to transport gas from inspired air to the red blood cells in pulmonary capillary network. It is, however, a misnomer, since gas transfer does not depend solely on diffusion across the alveocapillary membrane, and it is not a "capacity" in that there is no theoretical maximal limit. Many laboratories therefore employ the term "transfer factor" instead.

The diffusing capacity for carbon monoxide (DLCO) is the generally measured index. This is because carbon monoxide uptake is easily measurable, and the gas essentially follows the same pathway as oxygen during transport from alveolar air to red blood cells, and ultimate binding to hemoglobin. DLCO is the uptake of carbon monoxide from lungs per unit time per unit of carbon monoxide driving pressure. The test is usually performed for screening for diffuse lung diseases and pulmonary vascular disorders, precise characterization of airflow limitation, differential diagnosis and severity assessment of restrictive ventilatory defects, disability evaluation, and preoperative assessment. There are no absolute contraindications. However, for technical reasons, most machines cannot measure DLCO in individuals with an extremely low VC (usually <1.5 L) or severely dyspneic patients unable to hold breath for a sufficient time. The test cannot be performed on many patients receiving supplemental oxygen, as this needs to be discontinued before and throughout the test procedure.

Methodology

Diffusing capacity for carbon monoxide can be measured using a single breath method, an intrabreath method, or a rebreathing technique. The first is most commonly used, as it is simpler and better standardized. DLCO is calculated from the total lung volume, breath-hold time, and the initial and final alveolar carbon monoxide concentrations. It is a product of the subject's total lung capacity and rate of carbon monoxide uptake during the breath-hold time. An

estimate of the total lung capacity and the initial alveolar carbon monoxide concentration is obtained from the tracer gas concentration in exhaled gas.

The test can be repeated after an interval of at least 5 minutes. Generally, a mean value is reported from two acceptable tests whose results agree within 2 mL/min/mm Hg.

Interpretation

In contrast to other pulmonary function tests, observed DLCO values must be normalized to key nonrespiratory variables. Hemoglobin concentration is important as it removes the carbon monoxide from the blood, thus providing a nearly constant gradient for gas transfer. Anemic patients may thus have a lower DLCO, and the measurement should therefore be corrected for this factor in such patients. Smokers tend to have a small baseline level of blood carbon monoxide, and thus the transfer gradient may be less than for nonsmokers. If available, blood carboxyhemoglobin levels may be used to compensate for this anomaly.

As with other lung function tests, the patient's result is interpreted by comparing it with a corresponding reference value. It must be noted that the degree of variability of DLCO (both for a given subject and for the population) is much higher than the results of other lung function tests. In case serial tests are performed, a DLCO result can also be compared to previous values to detect any sizeable change. Normalizing the DLCO to patient's alveolar volume may provide additional information about the reason for an abnormal result. When alveolar volume is reduced (either because of true restriction or due to noncommunicating air spaces), the ratio of DLCO to alveolar volume is relatively preserved. The ratio is however decreased if alveolar volume is increased (as in emphysema) or normal (as in anemia, or nonperfusion of ventilated alveoli).

EXERCISE TESTING

Exercise testing is generally indicated for (a) evaluation of exercise intolerance, (b) evaluation of unexplained breathlessness, (c) preoperative assessment, and (d) formulation of exercise prescriptions. Detailed evaluation can give some clue about whether the underlying disorder is predominantly cardiovascular or respiratory in origin. Exercise testing can range from gross and crude assessments to highly detailed and standardized evaluation using computerized equipment. The two most regularly employed types of exercise testing for evaluating pulmonary diseases are the 6-minute walk test (6MWT) and a formal complete cardiopulmonary exercise testing (CPET).

Basic Modalities

Stair climbing remains the most basic exercise test that asks patients to climb stairs till they get limited by symptoms. There is, however, no consensus on how to standardize the procedure, and several variations (such as climbing at own

pace or at brisk pace, climbing with or without holding handrails) are used. Results are generally reported as number of stairs or number of flights of stairs.

Step tests ask the subject to walk up and down on a stool or bench at a specified rate. Several variations exist with regard to height of steps, number of steps, and the frequency of step-ups. The most popular is the Master two-step test. The test is well-suited to field use, and subjects can achieve close to their maximal exercise capacity.

The 6MWT is a simple procedure that assesses the maximum distance that a subject can walk on flat surface at his/her own pace in 6 minutes. The test provides a global estimate functional capacity, but does not provide any specific information on individual systems (cardiac, pulmonary, hematologic, and musculoskeletal) involved in exercise. The test is also sensitive to patient effort. The 6MWT is the most popular among the basic exercise tests as it is simple, practical and well standardized, and involves an activity familiar to almost everyone. A measured corridor, usually about 30 meters, is used, and the subject walks to and fro in this space at a self-determined pace. If a long corridor is not available, the test can be performed on a treadmill, although pacing and control are not as optimal and the distance walked is usually less. The test has good reproducibility and good correlation with other measures of functional status. For this reason, the 6MWT is the preferred investigation when a complete CPET is not available. Norms for healthy Indian men have recently become available.

The shuttle walk test uses an audio signal from a metronome to dictate the walking pace. Walking speed is incrementally increased every minute while the subject walks to and fro on a 10-meter straight path. The test is terminated when the subject can no longer maintain the required speed. Hence, the test correlates better with maximal symptom limited tests such as the CPET, rather than the submaximal tests like 6MWT. However, the test is more complicated than 6MWT, and may result in more frequent cardiac complications in the absence of electrocardiographic monitoring.

Exercise testing may sometimes be used to diagnose airway hyperreactivity due to exercise in patients with unexplained dyspnea. Exercise induced bronchospasm (EIB) is described as a self-limiting bronchospastic event occurring immediately after strong exercise. Typically, greater than 10–15% reduction in FEV1 and/or FVC is observed. EIB is caused by loss of heat, water, or both, from the airways during exercise.

Cardiopulmonary Exercise Testing

The CPET is a more complex investigation that involves exercise at incrementally increasing intensity. The test is terminated when symptoms limit further exercise, or the maximal exercise capacity is achieved. A computerized protocol provides breath-by-breath information on respiratory gas exchange, airflow, oxygen consumption, carbon dioxide production, and cardiac variables (such as heart rate, blood pressure, etc.). The subject exercises on either a treadmill or on a bicycle ergometer; the latter may however be preferable as work rate can be directly measured.

Electrocardiographic and noninvasive blood pressure monitoring accompanies the test. In addition, oxygen saturation is continuously monitored through pulse oximetry. Real-time data on ventilatory and gas exchange parameters is obtained by asking the subject to breathe through a mouthpiece connected to a spirometer and metabolic cart. Flow, volume, and exhaled oxygen and carbon dioxide concentrations are measured. Either incremental or constant work protocol can be used. The test is terminated when the subject (a) gets exhausted, fatigued or distressed, (b) develops signs of cardiovascular instability (ischemia, arrhythmia, substantial blood pressure elevation, etc.), or (c) develops significant hypoxemia.

Although data for a large number of monitored and calculated variables is generated, interpretation and clinical correlation depends on judicious integration of all available information. No single parameter is diagnostic of a cause for exercise limitation. Four basic measurements are critical in describing the response to exercise: oxygen consumption, carbon dioxide production, heart rate, and minute ventilation. Under steady state conditions, measured oxygen uptake (VO₂) equals metabolic oxygen consumption, and measured carbon dioxide output (VCO₂) is the same as its metabolic production. Both VO₂ and VCO₂ can be mathematically computed from expired gas concentrations.

The test is highly sensitive in identification of subclinical disease than other lung function tests conducted at rest. Therefore, CPET can be performed for preoperative assessment, disability evaluation, and selection of candidates for heart and/or lung transplantation. The test is also useful in determining whether breathlessness results from cardiac or pulmonary component among patients who have disorders of both organ systems.

OTHER TESTS

Airway Hyperresponsiveness

Airway hyperresponsiveness (AHR), a characteristic feature of bronchial asthma, is an increased sensitivity of airways to a variety of inhaled agents. AHR is classically measured using inhalation challenges with airway constrictor agonists, such as histamine or methacholine, which result in direct bronchoconstriction. Recently, inhaled mannitol solution has also become available for this purpose and acts by inducing osmotic changes in the airway. Exercise can also be used to test for AHR.

Airway Resistance

Airway resistance can be clinically estimated using several approaches such as interrupter technique, forced oscillation technique, and whole body plethysmography. The interrupter technique is the simplest, and requires monitoring airway pressure and airflow. Airway resistance is not normally determined for clinical purposes, but may provide additional information in the evaluation of patients suspected to have obstructive disorders. The range of normal airway resistance is not well defined, and generally values higher than 2.8 cm $H_2O/L/s$ are considered abnormal.

Pulmonary Mechanics

The elastic properties of the lungs are determined by relating alterations in volume of air in the lungs to the corresponding changes in the lung's recoil. A body plethysmograph is usually used to determine the static pulmonary mechanics. Lung recoil force is measured as the transpulmonary pressure. This is the difference between the alveolar and pleural pressures; the former is measured at mouth under static conditions with shutter at mouth closed and glottis open, and the latter is quantified through pressure measurement from a thin balloon placed in the lower third of esophagus and connected to a pressure transducer.

Respiratory Muscle Function

Estimation of maximal respiratory pressures is a simple technique to assess global respiratory muscle function. A manometer is used to record highest pressures during maximal inhalation and exhalation, which can be sustained for at least 1 second. Maximal expiratory pressure (MEP) is roughly twice as much as maximal inspiratory pressures (MIP). The test is commonly used during evaluation of respiratory muscle weakness in patients with neuromuscular disorders. It is also used as one of the several parameters to assess weaning potential in patients receiving mechanical ventilation. Abnormal muscle strength is identified by comparing observed values to reference data; such data is also available for the Indian population. Values are generally higher among men, and decline with age. A reduction in both MIP and MEP indicates generalized skeletal muscle weakness. A low MIP with normal MEP suggests isolated inspiratory muscle weakness (usually diaphragmatic). Isolated expiratory muscle weakness (normal MIP and low MEP) is rare.

The sniff nasal inspiratory pressure (SNIP) is a noninvasive test of inspiratory strength. The test is performed by wedging a catheter into one nostril and asking the subject to sniff through the other nostril. Pressure measured in the obstructed nostril. The SNIP correlates strongly with transdiaphragmatic pressure and the MIP, but provides no information on expiratory muscle function.

Transdiaphragmatic pressure can be measured after insertion of esophageal and gastric balloon catheters. This allows functional assessment during inspiration, expiration, a sniff, a cough, or phrenic nerve stimulation. Although the technique is highly complex and invasive, it is the best measure of respiratory muscle strength. However, a wide normal range limits its clinical utility.

Salient Features

- Presents a concise, yet fairly comprehensive information for day-to-day
- · Abridged form of Textbook of Pulmonary & Critical Care Medicine (2nd edition)
- Includes 101 chapters to cover clinical respiratory medicine, critical care, sleep medicine and respiratory perspectives
- It is reader-friendly with simple language
- Written by eminent authors from across the globe
- Useful for educators, pulmonologists, internists, intensivists, patients with lung diseases.

SK Jindal MD FAMS FNCCP FICS FCCP is a distinguished Clinician and Medical Teacher. He is an Emeritus Professor (Pulmonary Medicine), Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India, with a long teaching and research career. He has extensively contributed to the specialty with the introduction of the 1st postdoctoral course (DM) course in Pulmonary and Critical Care Medicine in India. He has authored several books, published a large number of research papers and served on the Editorial boards of many scientific and professional journals. He achievements by several national and international organizations including the Outstanding Educator Award of American Thoracic Society in 2011.

Available at all medical bookstores or buy online at www.jaypeebrothers.com



JAYPEE BROTHERS Medical Publishers (P) Ltd. **TAYPED** www.jaypeebrothers.com

Join us on ffacebook.com/JaypeeMedicalPublishers

