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Physiotherapy in Cardiopulmonary Conditions

Mariya Jiandani
Megha Sandeep Sheth
Neepa Hiren Pandya
Priya Singh Rangey

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Contents

Section 1: Cardiopulmonary System: Function and Dysfunction

1. Cardiovascular System and its Dysfunction 3
Narasimman Swaminathan, Neepa H Pandya

Cardiovascular System Function 3
Neepa H Pandya

Function of the Heart 3
Coronary Circulation 7

Cardiovascular System Dysfunction 9
Narasimman Swaminathan

Causes of Cardiac Dysfunction 9
Pathophysiology of Cardiac Dysfunction 9
Signs and Symptoms of Heart Failure 10
Physiotherapy Clinical Decision-Making 12

2. Respiratory System and its Dysfunction 14
Jaimala Shetye

Structure of the Respiratory System 14
Functions of the Respiratory System 17
Dysfunctions of the Respiratory System 20

3. ICF with Reference to Cardiopulmonary System 26
Vimal Telang, Mariya Jiandani

Models of Disability and Evolution to ICF 27
ICF Coding 30
ICF as an Outcome Measure 30
ICF Core Sets 30
Goal Setting Made Easy with ICF 34

4. Cardiopulmonary Response to Exercise 39
Veena Kiran Nambiar

Cardiovascular System 40
Short-term or Acute Cardiac Responses to Exercise 41
Stroke Volume Response to Submaximal and Maximal Exercise 42
Heart Rate Regulation 43
Long-term or Chronic Adaptations of the Heart to Exercise 44
Long-term Responses of the Nervous Control System 45
Vascular System 45
Pulmonary System 48
Short-term or Acute Ventilatory Responses to Exercise 50
Determinants of Maximal Oxygen Consumption during Dynamic Exercise 56

Section 2: Clinical Evaluation, Relevant Investigations and Pharmacological Management: Physiotherapy Perspective

5. Clinical Evaluation and Documentation of Heart and Lung Function 61
Titiksha Pol

History Taking or Subjective Evaluation 61
Objective Examination 66
Proforma 76

6. Biochemical Investigations 79
Hardini Jay Parikh

Serum Enzymes and Cardiac Biomarkers 80
Lipid Profile 82
Coagulation Profile 83
Electrolytes 84
Complete Blood Cell Count 85
Blood Sugar Profile 86
Creatinine 88
Blood Urea Nitrogen 88
C-reactive Protein 88
Investigations for Respiratory System Assessment 88
Liver Function Tests 88
Other Important Clinical Investigations 89

7. Radiological Investigations 93
Ruju Doshi

X-ray 93
Ultrasound 113
Computed Tomography Scan 113
Magnetic Resonance Imaging 120
Positron Emission Tomography-Computed Tomography 121

8. Pulmonary Function Testing 122
Tarpan Shah

Important Terminologies Related to Pulmonary Function Testing 123
Methods of Pulmonary Function Testing 124
Indications for Pulmonary Function Testing 128
Contraindications 128
Risks Involved in Pulmonary Function Testing 128
Bedside Pulmonary Function Testing 129
Pulmonary Function Testing Measurement Instruments/ Devices 130
Graphs in Pulmonary Function Testing 131

Approach to Pulmonary Function Testing Interpretation 132

9. Evaluation of Arterial Blood Gases and Acid-Base Balance 138
Khalil Ahmed Dalvi, Titiksha Pol

Importance of Arterial Blood Gas 138
Components of an Arterial Blood Gas Report 138
Steps to Interpret an Arterial Blood Gas Report 144
Clinical Examples of Arterial Blood Gas Interpretation 145

10. Electrocardiography 147
Hardik Parab

Normal Electrocardiogram Waveform 148
Interpretation of ECG 149

11. Special Investigations 161
Hardik Parab

Electrocardiography 162
Pulmonary Function Test 162
Exercise Oximetry 162
Functional Exercise Tests 162
Ankle Brachial Index 162
Echocardiography 163
Bronchoscopy 164
Cardiac Biomarkers 165
Holter Monitoring 166
Cardiac Computed Tomography 167
Cardiac Catheterization and Angiogram 167
Magnetic Resonance Imaging 169
Perfusion Scans 171
Implantable Event Recorder 172
Transtelephonic Pacemaker or Implantable Cardioverter/ Defibrillator Transmission 172
Electrophysiology Studies 173

12. Exercise Tolerance Tests 176
Aditi Ketkar Berry, Kushal Madan, Abraham Samuel Babu

Evaluating Exercise Intolerance 176
Procedure for Testing 176
Interpretation of Test Results 180
Integrating Imaging and Physiological Assessments with Exercise Testing 182
Evaluating Exercise Capacity during the COVID-19 Pandemic 182

13. Disability Evaluation in Cardiorespiratory Dysfunction 184
Ravindran R

Disability Evaluation of the Respiratory System 185
Disability Evaluation of the Cardiovascular System 187

14. Pharmacological Management: Drugs Used in Heart and Lung Diseases 198
Supriya Malhotra, Falguni Majmudar, Amee Pandya

Drugs Used for Cardiovascular Therapeutics 199
Drugs for Hypertension 199
Drugs for Heart Failure 203
Drugs for Ischemic Heart Disease 208
Drugs for Arrhythmias 210
Drugs for Peripheral Vascular Disease 212
Drug Therapy in Respiratory Therapeutics 213
Drug Therapy for Bronchial Asthma and Chronic Obstructive Pulmonary Disease 213
Mucoregulators 215
Mucolytics 215
Expectorants 215
Antitussives 216
Occupational and Environmental Lung Disorders 216
Bronchiectasis 216
Cystic Fibrosis 216
Interstitial Lung Disease 216
Sleep Apnea 216

15. Drugs Affecting Physical Performance 218
Falguni Majmudar, Supriya Malhotra, Nehal Parikh

Performance-enhancing Drugs 219
Performance-affecting Drugs 221
Drugs Causing Sexual Dysfunction 222

Section 3: Physiotherapy Approaches in Management of Cardiopulmonary Dysfunction

16. Patient Education 227
Poorvi Devani

Patient-centered Care Model 227
Benefits of Patient Education 228
Approaches to Patient Education 229
A Model for Patient Education 229
Suggestions for Enhancing the Patient Education Process 232
Factors Affecting Patient Education 232

17. Humidification and Nebulization 234
Mariya Jiandani, Farah Jiandani

Definition of Terms 234
Process of Humidification 234
Humidifiers 235
Nebulization (Aerosol Therapy) 238

18. Bronchial Hygiene and Use of Assistive Devices 245
Mariya Jiandani

Normal and Impaired Mucociliary Clearance 245
Techniques of Bronchial Hygiene or Airway Clearance 247
Techniques of Airway Clearance 248
Assistive Devices 258
Clinical Decision-making in Technique Selection 262

19. Therapeutic Body Positioning 265
KV Haribabu, Gauri Mayank Afle

Various Body Positions 266
Effects of Therapeutic Body Positioning 268

Modes of Giving Body Positioning 271
Therapeutic Body Positioning in Lung Pathologies 272

20. Strategies to Improve Lung Volumes 275
Anuprita Thakur
Therapeutic Approaches to Increasing Lung Volume 276
Mechanical Devices to Increase Lung Volume 279
Complementary Therapies to Improve Lung Volume 285
Technology to Improve Lung Volume 285

21. Work of Breathing 288
Rachna Arora
What is Work of Breathing? 289
Work of Breathing in Disease 291
Factors Affecting Work of Breathing 292
Assessment of a Breathless Patient 293
Management of a Breathless Patient 295
Physiotherapy Interventions to Reduce Work of Breathing 296

22. Respiratory Muscle Dysfunction and Training 300
Vidhi Thakar, Neepa H Pandya
Respiratory Musculature Mechanics 300
Respiratory Muscle Dysfunction 301
Evaluation of Respiratory Muscles 302
Training of the Respiratory Muscles 304

23. Energy Conservation Techniques 309
Maniarasi Shunmugam
Measurement of Energy Expenditure 310
Ventilatory Muscle Demand 310
Energy Conservation 312
Assessment 312
Aims and Goals 313
Energy Conservation Techniques 313
Benefits of Energy Conservation in Chronic Obstructive Pulmonary Disease 314
Educating the Patient 315
Bioenergetic Therapy 315

Section 4: Physiotherapy Management of Common Cardiopulmonary Conditions

24. Airway and Parenchymal Diseases 319
Ankita Asher, Neepa H Pandya
Airway Diseases 319
Chronic Obstructive Pulmonary Disease 319
Asthma 322
Bronchiectasis 322
Parenchymal Diseases 324
Cystic Fibrosis 324
Pneumonia 324
Interstitial Lung Diseases 325
Chronic Fibrosing Interstitial Pneumonias 325
Hydatid Cyst/Echinococcal Disease 327
Lung Abscess 327
Exercise Performance in Chronic Lung Diseases 328

25. Disorders of Pleura and Thoracic Cage 332
Bela Agarwal
Anatomy and Physiology of Pleura 332
Pleural Disorders 336
Disorders of the Thoracic Cage 346

26. Physiotherapy in Infectious Disease-COVID-19 352
Mariya Jiandani
Overview of SARS-CoV-2 353
Physiotherapy in COVID-19 360
Post-acute COVID-19 Syndrome 366
Physiotherapy in Post-COVID-19 367
Safety Considerations 374

27. Diseases of the Cardiovascular System 379
Pooja Akhtar, Neepa H Pandya
Overview 380
Coronary Artery Disease 380
Neepa H Pandya
Pathophysiology 381
Impact on Exercise Performance 382
Clinical Features 383
Assessment 384
Medical Management 384
Valvular Heart Diseases 385
Pooja Akhtar
Mitral Stenosis 386
Mitral Regurgitation 389
Aortic Stenosis 391
Aortic Regurgitation 393
Heart Failure 395
Pooja Akhtar
Classification of Heart Failure 395
Pathophysiology 395
Precipitating Causes of Heart Failure 397
Clinical Manifestations 398
Investigations 399
Medical Management 400
Physiotherapy Management of Chronic Heart Failure 401

28. Cardiopulmonary Physiotherapy in Neuromuscular Disorders 406
Neepa H Pandya, Mihir Mehta
Pathophysiology 406
Physiotherapy Management 410

29. Physiotherapy in Cardiac Surgeries 417
Sushama A Bhandare
Coronary Artery Bypass Graft 418
Minimal Invasive Cardiac Surgery 419
Valve Replacement Surgery 419

Congenital Heart Diseases and Diseases of the Great Vessels 420
Complications in Cardiac Surgeries 426
Physiotherapy Management 428

30. Physiotherapy in Pulmonary Surgeries 439
Sushama A Bhandare
Indications of Pulmonary Resection 439
Types of Pulmonary Surgeries/Resection 440
Incision/Thoracotomy 441
Preoperative Investigations 442
Complications of Pulmonary Surgery 443
Postoperative Drainage 443
Postoperative Problem List after Lung Surgery 445
Physiotherapy Management 445

31. Physiotherapy in Abdominal Surgeries 451
Megha Sheth, Neepa H Pandya
Postoperative Complications 451
Physiotherapy Assessment in Abdominal Surgery Patients 453
Physiotherapy Management 454

32. Yoga: A Physiotherapist's Perspective 458
Nilima Patel
Introduction: Yoga—A Psychophysical Spiritual Approach in Health Care 458
Introduction to Ashtanga Yoga 459
Ashtanga Yoga: Principles and Guidelines for a Healthy Heart 460
Chair Yoga for Cardiopulmonary Health 467

33. Yoga and Cardiopulmonary Conditions 473
Rashmi Mahulkar Muthal
Role of Yoga Therapy in Obstructive Airway Disease 473
Integrated Approach of Pranayama Therapy in Asthma 474
Cascade of Biomechanical Changes Occurring in Bronchial Asthma 477
Neurophysiological Changes Occurring in Asthmatic Patients as an Effect of Pranayama 478
Integrated Approach of Pranayama 478
Mechanism of Action of Pranayama Upon Increase in Breath-holding Period 480
Role of Yoga Therapy in Coronary Artery Disease 480
Changes in the Cardiovascular System with Integrated Yogic Practices at the Root Level 480
Effects of Yoga on the Autonomic Nervous System 481
Effect of Yoga on Blood Vessels 481
Yoga and Coronary Artery Diseases 482

34. Nutritional Counseling in Cardiopulmonary Conditions 486
Shivani Sheth, Charmi Shah, Neeta Vyas
Importance of Nutritional Counseling 487
Interference of Diet with Cardiac and Respiratory Drugs 487
Food Items to be Avoided in Cardiac and Respiratory Conditions 487
Dietary Guidelines for Heart Diseases 488
Postcardiac Surgery Nutrient Recommendations 490

Section 5: Physiotherapy for Intensive Care Unit

35. Intensive Care Unit Setup and Equipment 495
Sapna D Gupta, Jaldipsinh N Gohil
Guidelines for Intensive Care Unit Setup and Equipment 496
Categorization of Services for the Critically Ill Patient 497
Recommendations for Intensive Care Unit Admission 497
Fire Safety in Intensive Care Unit 497
Intensive Care Unit Equipment 499

36. Mechanical Ventilation and Weaning 510
Farah Jiandani, Mariya Jiandani
Respiratory Failure 510
Mechanical Ventilator 511
Types of Mechanical Ventilators 512
Phases of a Mechanical Ventilator 513
Modes of Mechanical Ventilation 513
Settings 517
Complications of Mechanical Ventilation 520
Weaning 520

37. Oxygen Therapy 525
Harita Vyas
Hypoxia and its Features 526
Indications/Goals of Oxygen Therapy 526
Oxygen Therapy as a Drug 527
Oxygen Delivery Systems 528
Long-term Oxygen Therapy and Portable Devices 531
Oxygen Therapy in Pediatric Population 532
Delivery Approach for Oxygen Therapy and Physiotherapy 533
British Thoracic Society Guidelines for Oxygen Delivery for Various Diseases 533
Exercise and Oxygen Therapy 533
Hazards of Oxygen Therapy for Healthcare Professionals 534

38. Physiotherapy in the Intensive Care Unit 536
Razia Nagarwala, Mariya Jiandani
Intensive Care Unit Environment 537
Patient in the Intensive Care Unit 537
Intensive Care Unit-related Complications 537
Goals of Physiotherapy in Intensive Care Unit 538
Techniques to Prevent Complication and Optimize Care for Early Discharge from ICU 539
Early Mobilization 546
Safety Criteria 549
Benefits of Physiotherapy Intervention in ICU 549

39. Physiotherapy Management in an Organ Transplant Unit 552
Dipali Rana, Neeta Vyas
Benefits of Physiotherapy in Transplantation 553
Comprehensive Examination 553
Early Postoperative Physiotherapy 554
Physiotherapy in the Intensive Care Unit 554

40. Physiotherapy in a Cancer Intensive Care Unit 559
Anuradha Abhijeet Daptardar, Ajeeta Mohan Kulkarni
Cancer Treatment and its Side Effects 560
Surgery 560
Postoperative Complications 563
Chemotherapy 564
Radiotherapy 564
Enhanced Recovery after Surgery 565
Palliative Care 566

41. Pediatric and Neonatal Intensive Care Unit 570
Priya Singh Rangey
Anatomy and Physiology of the Pediatric Cardiorespiratory System 571
PICU and NICU Equipment and Setup 571
Reasons for Admission to NICU and PICU 575
Assessment 575
Management 576
Management of Specific Neonatal Disorders 582
Dying Children 582
Care of Parents 583

42. Cardiopulmonary Resuscitation 585
Priyanshu Rathod
Introduction to Cardiopulmonary Resuscitation (CPR) 585
Cardiac Arrest Versus Heart Attack 585
Basic Skills when Giving Cardiopulmonary Resuscitation 586
Adult Cardiopulmonary Resuscitation and Automated External Defibrillator Skills 590
Child Cardiopulmonary Resuscitation 591
Infant Cardiopulmonary Resuscitation 592
General Breathing Problems 592
Helping a Choking Adult 592
Using Epinephrine Pens 593
Safety Measures in Infective Conditions—Cardiopulmonary Resuscitation during the COVID-19 Pandemic 593

Section 6: Rehabilitation

43. Cardiac Rehabilitation 597
Neepa H Pandya, Sarla V Bhatt
Overview 597
Exercise Response in Cardiac Patients 598
Rationale for Exercise Training 598
Exercise Prescription 599
Exercise Training Program 600
Phases of Cardiac Rehabilitation 604
Risks Associated with Exercise Training 604
Boundaries of Exercise Training Session 607
Outcomes of Cardiac Rehabilitation 607

44. Pulmonary Rehabilitation 609
Mariya Jiandani, Neepa H Pandya
Overview 609
Common Impairments Associated with Respiratory Diseases 610
Definition of Pulmonary Rehabilitation 611
Program Goals 611
Types of Pulmonary Rehabilitation Programs 612
Components of Pulmonary Rehabilitation 612
Strength of Evidence for Pulmonary Rehabilitation 622
Management of Acute Exacerbations 622
Outcomes of Pulmonary Rehabilitation 623
Disease-specific Considerations in Pulmonary Rehabilitation 623

45. Telerehabilitation 627
Mrinmayee Koltharkar, Mariya Jiandani
Telemedicine Around the World and in India 627
History of Telerehabilitation 628
Barriers for Center-based Physical Rehabilitation 629
Advantages of Telerehabilitation 629
Concerns of Telerehabilitation 629
Fields Involved in Telerehabilitation 630
Modes of Delivery 630
Use of Mobile Technology in Telerehabilitation 631
Principles for the Use of Telerehabilitation 631
Clinical Application 632
Future Perspectives 637

46. Cardiopulmonary Rehabilitation: Future Trends 640
Anup Bhat, Ganesha Poojari, Akhila Satyamurthy, Abraham Samuel Babu
Current State of Cardiac and Pulmonary Rehabilitation 640
Impact of COVID-19 on Cardiac and Pulmonary Rehabilitation 641
Strategies to Advance Cardiopulmonary Rehabilitation 642
Future of Cardiopulmonary Rehabilitation 644

47. Palliative Care in Cardiopulmonary Conditions 647
Anjali Ravindra Bhise
Multi- and Interdisciplinary Team 648
Physical Therapy 648
Physiotherapy Management for Common Symptoms 650
Pulmonary Rehabilitation Versus Palliative Care 652

Index 657

2 Respiratory System and its Dysfunction

Jaimala Shetye

LEARNING OBJECTIVES

At the end of the chapter, the reader will be able to:

- Understand the structure and function of the respiratory system.
- Understand the dysfunction of the respiratory system.

CHAPTER OUTLINE

- Introduction
- Structure of the Respiratory System
  - Upper Respiratory Tract
  - Lower Respiratory Tract
  - Mucociliary Escalator
  - Alveolar Capillary Membrane
  - Lung Interstitium or the Extracellular Matrix
  - Surfactant
  - Rib Cage and Pleura
  - Control of Respiration
- Functions of the Respiratory System
  - Air Conditioning Function of the Upper Respiratory Tract
  - Defense Mechanisms Existing in the Lower Respiratory Tract
  - Highly Efficient Breathing Function
  - Ventilation and Perfusion
- Assessment of Lung Function
- Dysfunctions of the Respiratory System
  - Dysfunction of the Upper Respiratory Tract
  - Dysfunction of the Lower Respiratory Tract
  - Dysfunction of the Mucociliary Escalator
  - Impaired Cough
  - Respiratory System Dysfunction in Obstructive Lung Diseases
  - Respiratory Dysfunction in Restrictive Lung Disease

INTRODUCTION

The respiratory system is a unique system. It is continuously in contact with the external environment and hence it is easily influenced by the change in the external environment. At the end of a deep breath, the volume of air in the lungs is about 80%, in the blood amounts to 10%, and only the remaining 10% is in the lung tissue. This small mass of tissue is spread over an extremely large area—nearly the size of a tennis court, thus providing a large surface area for gaseous exchange. An important feature of the respiratory system is that it cannot be voluntarily kept closed to avoid environmental irritants and hence it has a strong in-built defense mechanism at many levels in the upper respiratory tract (URT) and the lower respiratory tract (LRT). Understanding the structure and function of the respiratory system is a prerequisite to know about the cause of dysfunctions of the respiratory system. Thus, a clinical reasoning for the dysfunctions and hence the symptoms can be understood. This understanding helps us to plan effective management toward overcoming the limitations imposed by these dysfunctions or, to a certain extent, reverse the dysfunction where possible.

STRUCTURE OF THE RESPIRATORY SYSTEM

The respiratory system consists of respiratory apparatus and its neural control. The respiratory apparatus can be divided into URT and LRT.

Upper Respiratory Tract (URT)

The URT includes:

- Nose (two nostrils) which is the entry point of the environmental air into the lungs
- Paranasal sinuses
- Nasopharynx
- Oropharynx
- Laryngopharynx
- Larynx

The larynx is the terminal part of URT responsible for vocalization. The conchae in the nostrils act as a heat and moisture exchanger for the inhaled air that passes into the lungs. The turbulence caused by the flowing air due to the structure of conchae helps to exchange heat such that the inhaled air is brought to body temperature by the time it

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reaches the carina irrespective of the temperature. The conchae in the nostrils increase surface area (166 cm^2) for humidification, heating/cooling, and filtering of air.

Lower Respiratory Tract (LRT)

The LRT begins with the trachea. It has 23 generations. Generation is the level where the next division of the airway takes place **(Fig. 2.1)**.

- Thus, the trachea is the first generation.
- The right and left main bronchi are the second generation. The first generation has a single airway and the second has two: the right and left main bronchi.
- The third generation comprises lobar bronchi, three on the left and two on the right, amounting to five airways in the third generation.
- The lobar bronchi divide into segmental branches. On the right side the segmental branches are—apical, anterior and posterior for apical lobe; medial and lateral for middle lobe; and apical, anterior basal, posterior basal, medial basal and lateral basal for lower lobe. On the left side, the medial basal is generally missing, amounting to nine segmental bronchi on the left. Thus, the fourth generation has 10 on right and 9 on left amounting to 19 bronchi.
- The airways further divide and redivide to form further generations.

There are a total of 23 generations of the tracheobronchial tree of which—the first 14 generations consist of *conducting zone*; the 15th generation is the *transitional zone*; and the 16th to 23rd generations form the *respiratory zone*. The respiratory zone consists of respiratory bronchioles, alveolar ducts, and alveolar sac. It is here in the respiratory zone that an exchange of oxygen, carbon dioxide, and toxins takes place. This zone is the *functional unit of gaseous exchange*.

As the branching of airways takes place, some structural changes occur according to the function of different levels of bronchi.

Structural changes in the airway from the 1st to the 23rd generation of tracheobronchial tree

The *trachea* is the toughest part of the tracheobronchial tree as it is the only extrathoracic part of the LRT. It needs to have a mechanism to prevent the collapse of its walls during the pressure changes causing inspiration and expiration. For this purpose, the tracheal wall has cartilages which offer it the required toughness to prevent the collapse of airway walls during expiration. They are "C" shaped, the open part of the "C" placed posteriorly to avoid injury to the esophagus which is in proximity to the trachea posteriorly. It is seen that from the trachea to the 17th generation (terminal bronchioles), the amount of cartilage, smooth muscle cells, and cilia go on decreasing in an amount such that the respiratory zone is fully devoid of these components to prevent them from hindering gaseous exchange. Also, from the 17th generation onward, alveolar enlargements are seen on the walls of the bronchioles. These projections go on increasing with every generation and finally end in the alveolar sac. This is responsible for the large cross-sectional area which favors fast and efficient gaseous exchange.

Mucociliary Escalator

The mucociliary escalator, also known as mucociliary blanket, functions throughout life **(Fig. 2.2)**.

- *Mucus*: It contains 95% of its component as water, glycoproteins, carbohydrates, lipids, DNA, and cellular

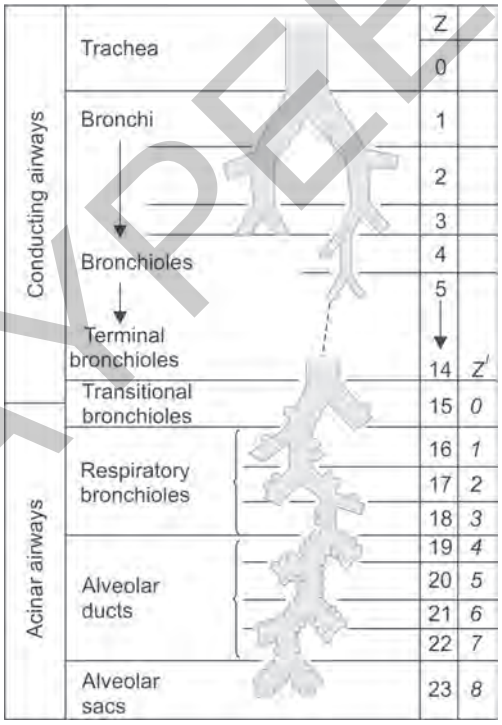


Fig. 2.1: Model of airway branching.

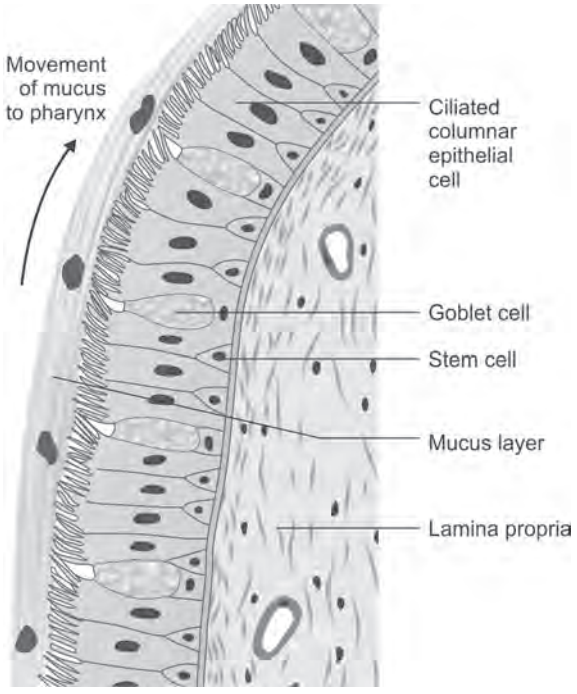


Fig. 2.2: Mucociliary blanket.
(For color version, see Plate 3)

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debris. The water-rich mucus provides for humidification of the inhaled air as it passes over it during inspiration. Mucous membranes provide up to 650–1,000 mL of water/day to humidify the air. Hence, adequate hydration is vital for the continuous ciliary activity.

- *Cilia*: Each epithelial cell has approximately 200 cilia. The tips of these cilia are clawed. The cilia are embedded in a layer of periciliary fluid. The cilia beat synchronously in this periciliary fluid at approximately 20 strokes per second. The speed of their movement is 0.5 mm/min in small airways and 20 mm/min in the trachea. The periciliary fluid also needs to be in desired consistency. The cilia continuously beat upwards. During the effective stroke, the claws of the cilia grip the lower layer of the mucus which is just above the cilia and push it upward; during the recovery stroke, the cilia retract in the periciliary fluid and come to their original position only for the next effective stroke. Thus, the mucus blanket is continuously moved cephalad up to the larynx as cilia are present up to this level in the LRT. From here, normally the mucus is swallowed which is not noticeable. If the mucus is in large amount as in some lung conditions, then it is expelled out voluntarily. Both excessive water content and reduced water content of the periciliary fluid disturb the normal ciliary activity.

The particles which cannot be expelled by this mucociliary escalator are expelled by cough.

Alveolar Capillary Membrane

The seat of gaseous exchange is the alveolar capillary membrane, also called the *gas exchange barrier*. Oxygen, on its way from alveolar gas to the hemoglobin of the red blood cells, passes through pulmonary surfactant, alveolar epithelium, interstitium, capillary endothelium, plasma, and finally the erythrocyte **(Fig. 2.3)**.

- Alveolar epithelium is made up of epithelial cells, alveolar macrophages, and fibroblasts. The epithelial cells form the structural support of the alveoli. The alveolar macrophages are freely moving cells which phagocytose bacteria and foreign particles that have escaped previous defense mechanisms like the propulsion by the mucociliary escalator or expulsion by cough reflex.
- The fibroblasts synthesize collagen and elastin which are the main components of the lung interstitium. On repeated disease or insult of the lung tissue, a large amount of collagen and elastin fibers are laid down which finally results in interstitial fibrosis.
- The interstitium fills the space between the alveolar epithelium and the capillary endothelium.

Lung Interstitium or the Extracellular Matrix

The alveolar capillary membrane or the gas exchange barrier is built of three minimal tissue layers:

1. An endothelium lining the capillaries
2. An epithelium lining the airspaces
3. An interstitial layer to house the connective tissue fibers

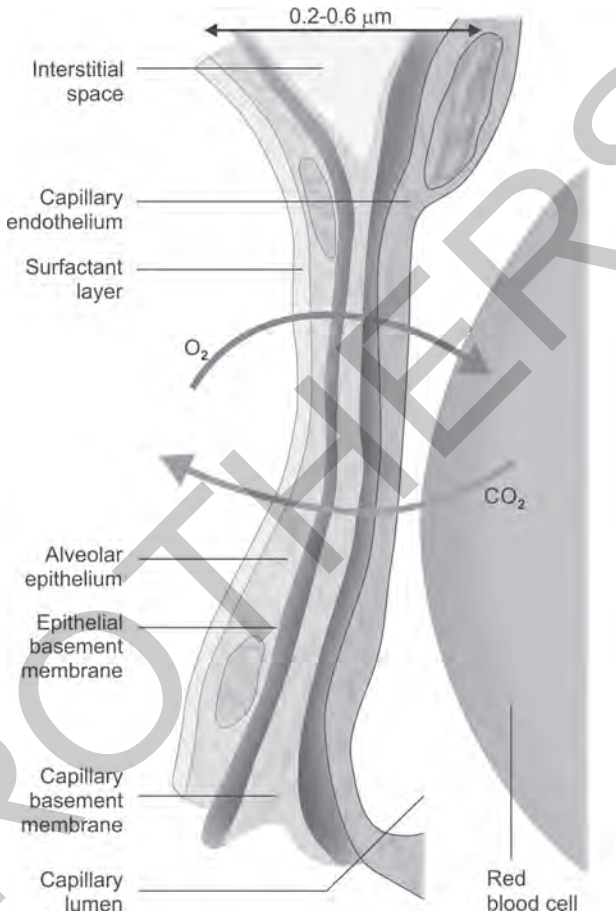


Fig. 2.3: Alveolar capillary membrane.
(For color version, see Plate 3)

The lung interstitium, also called the pulmonary extracellular matrix (ECM), determines the tissue architecture of the lung and provides mechanical stability and elastic recoil, which is essential for physiological lung function. The interstitial ECM of the alveoli is composed of a relaxed meshwork, with collagens and elastin as important core proteins. The pulmonary ECM is composed of two main structural types:

1. *Basement membranes*: These are thin sheets of glycoproteins that cover the basal side of epithelia and endothelia and surround muscle, fat, and peripheral nerve cells.
2. *Interstitial matrices*: They form a loose and fibril-like meshwork which interconnects structural cell types within tissues and thereby maintains the three-dimensional (3D) cohesiveness and is responsible for the biomechanical behavior of the lung.

Biochemical and biomechanical signals initiated by the ECM instruct cellular function and differentiation. Thus, the ECM plays a decisive role in lung development, tissue remodeling, and maintaining adult homeostasis.

The interstitium of the alveolar wall is continuous with the perivascular spaces and forms the route for drainage of fluid from the capillaries to the lymphatics.

Composition of the Extracellular Matrix

Collagens constitute a major portion of the protein content of the lung and are severely altered in many lung diseases.

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They provide the 3D frameworks for the lung tissue. The collagens have great tensile strength but low elasticity; hence, they contribute to the overarching architecture of the lung, whereas the large elastic fibers, which are characterized by low tensile strength and high elasticity, provide the lung with its necessary compliance and elastic recoil.

Surfactant

Surfactant is composed of around 90% lipids, mainly saturated phosphatidylcholine, and around 10% proteins.

- Surfactant prevents alveolar atelectasis by a reduction of the alveolar surface tension which prevents the formation of intra-alveolar edema.
- Another function of surfactant is the immunomodulatory function in the innate host defense system.

Two surfactant proteins—surfactant proteins A (SP-A) and surfactant proteins D (SP-D), are members of a family of innate immune proteins, known as collectins, that bind pathogens and facilitate the clearance by immune cells.

Rib Cage and Pleura

The rib cage consists of 12 pairs of ribs, their costal cartilages, and the sternum. The 12 ribs are attached to the 12 thoracic vertebrae posteriorly. Most attach anteriorly via their costal cartilages to the sternum. Ribs 1–7 are called true ribs and 8–12 are called false ribs, the last two ribs being floating ribs as they are not attached to the sternum at all. The lung is enclosed in the rib cage and is lined by the visceral pleura and the parietal pleura with a potential space called the interpleural space which is filled with a thin film of serous pleural fluid which lubricates the pleural surfaces and allows the layers of pleura to slide smoothly over each other during respiration. The two pleurae are in a close association such that they can slide easily over one another allowing inspiration and expiration without effort but they cannot be pulled apart due to the negative pressure that exists between the two.

Control of Respiration

Respiration is controlled by two dominant systems:

1. Central nervous system
2. Extracerebral system

Caudal pons is responsible for a sustained inspiratory drive and acts as the "apneustic center," and an area in the rostral and lateral portions of the pons presumably inhibits the apneustic drive and acts as the "pneumotaxic center".

- The chemoreceptors and mechanoreceptors are other factors regulating the respiration.

Any deviation of structure from the normal affects the function of the system.

Muscles of Inspiration

The *diaphragm* needs a special mention, as it is the primary muscle of inspiration contributing to 70% of tidal volume (TV). It separates the thoracic cavity from the abdominal cavity and is innervated by the phrenic nerve.

- At rest, the diaphragm is dome-shaped due to a balance between the functional residual volume of the lungs and the abdominal contents.
- On contraction, the diaphragm descends over the abdominal viscera and flattens. This causes the lower ribs to move outward and upward, thereby dropping the intrathoracic pressure.
- Thus, the diaphragm contraction brings about a 3D increase in thoracic volume.
  - The vertical diameter of the thoracic cage increases due to the downward descent.
  - The anteroposterior and lateral diameters of the lower rib cage increase due to the upward and outward movement of the lower ribs as explained above.
- As the diaphragm descends down, the intra-abdominal pressure increases. This increase in pressure along with the support offered by the visceral organs in the abdominal cavity plays a crucial role in enabling an effective contraction of the diaphragm.

The movement of the rib cage is also affected by the contraction of other inspiratory muscles such as the *intercostals* and the *scalene*.

- They bring about the *pump handle and the bucket handle* movements of the ribs, thereby increasing all the three dimensions of the rib cage.

The *sternocleidomastoid and serratus anterior* act as accessory muscles of inspiration.

- They function when the ventilatory demand increases, as in exercise or in respiratory diseases causing the fatigue of primary muscles of inspiration.
- They are able to act as accessory muscles of respiration because of their favorable placement, as their attachment to the rib cage helps them to move the rib cage and assists the prime movers of respiration when they undergo fatigue or when the respiratory demand increases.

Expiration

- Quiet expiration is a *passive* process and occurs because of the recoil of the lung tissue on cessation of inspiration.
- Strong *abdominal* muscles are required for forced expiratory maneuvers such as coughing or huffing, thus causing airway clearance.

FUNCTIONS OF THE RESPIRATORY SYSTEM

The respiratory system is often underestimated when it is said that the function of the respiratory system is breathing; because breathing is just one of the many important functions of the respiratory system. The respiratory system works with the circulatory system to provide oxygen and to remove the waste products of metabolism. It also helps to regulate the pH of the blood, provides for a sense of smell, and helps to generate resonance in speech.

The human respiratory system is the only system in the body which is so open to the external environment and gives easy access to foreign particles as it cannot be forced shut for

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a longer period of time like the eyes or mouth. The protective function plays an important role in providing this security which is so important.

Air Conditioning Function of the Upper Respiratory Tract

The air that enters the nose has to be filtered for foreign particles, brought to body temperature, and 100% humidified before it reaches the carina.

- The large particles in the inhaled air are caught up in the nasal hair. This prevents them from entering the airways; thereby, preventing injury or irritation.
- The smaller particles that miss this trap are caught in the mucus blanket which lines the URT and the LRT. Goblet cells in the mucus membrane secrete 100 mL/day of mucous which aids in trapping inspired particles, and prevents them from entering the LRT.
- The trapped particles are then moved up by the mucociliary escalator.

Warming up of air is regulated by the structure of the nostrils.

- The conchae in the nostrils act as a heat and moisture exchanger for the inhaled air that passes into the lungs.
- The turbulence caused by the flowing air due to the structure of conchae helps to exchange heat such that the inhaled air is brought to body temperature by the time it reaches the carina irrespective of the temperature of the inhaled air.

The mucous layer which has 95% of water content also helps to humidify the inhaled air.

It is important to note that this filtration, warming, and humidification are optimal conditions required for efficient diffusion at the alveolar capillary membrane.

Defense Mechanisms Existing in the Lower Respiratory Tract

The finer particles that evade the trap of mucociliary blanket are carried further in the LRT and may act as an irritant stimulus and stimulate the cough reflex which expels the irritant with forced violent expiration.

Cough

It is a powerful reflex responsible for airway clearance. Cough can be voluntary or involuntary. The cough receptors are located in the pleura, between the larynx and the fourth generation of the bronchi, i.e., the segmental bronchi, and are also present in the external auditory canal. These receptors are stimulated by irritants causing inflammation and causing chemical, mechanical or thermal stimulation.

- The cough is characterized by sudden deep inspiration up to 90% of the total lung capacity, followed by the sudden closure of the glottis.
- This results in the trapping of a large amount of air in the lungs.
- This suddenly increases the intrathoracic pressure to up to 300 mm Hg narrowing the trachea and the bronchi.
- This is followed by a sudden opening of the glottis and the trapped air explodes outward at a speed of up to 500 mph.
- The resultant shearing force wipes the secretion or irritant off the airway wall.

Strong muscles of respiration, good compliance, elasticity, and an intact neural input are the key requirements for an effective cough.

The particles that evade even this level reach the alveoli. The free-moving macrophages provide the final protection by engulfing and degrading these irritants and protecting the respiratory system.

Highly Efficient Breathing Function

Breathing consists of inspiration and expiration, the ratio of inspiration to expiration being 1:2 or 1:3.

- Contraction of the inspiratory muscles brings about expansion of the thoracic cage resulting in reduction in the intrathoracic and intra-alveolar pressure.
- The pressure gradient between the intra-alveolar and the environmental pressure causes the air to enter the lungs during inspiration.
- Expiration is a passive process triggered by relaxation of the inspiratory muscles causing the fall back of the thoracic cage to the previous dimension leading to reversal of pressure and expiration. The expiration occurs because of the passive elastic recoil of the lung tissue.
- Thus, the work of breathing is ensured to be low such that only 4% of the total body oxygen consumption at rest is being utilized for breathing, which explains the efficiency of the respiratory system.

Impairment in any of these functions of the respiratory system results in respiratory dysfunction increasing the work of breathing.

Ventilation and Perfusion

A synchronized function of the respiratory system and cardiovascular system ensures adequate ventilation–perfusion throughout life. The ventilation/perfusion ratio ($\dot{V}/\dot{Q}$ ratio or V/Q ratio) is a ratio used to assess the efficiency and adequacy of the matching of two variables. Here, V is the volume of air in the alveoli, taking part in diffusion ($\dot{V}$ indicates the rate of flow instead of volume). Q is the volume of blood in the capillaries supplying the alveoli and participating in diffusion ($\dot{Q}$ indicates the rate of flow instead of volume). The V/Q ratio or the $\dot{V}/\dot{Q}$ ratio is different for different areas of the lung. Gravity has a profound effect to influence this ratio, and hence a change in body position changes this ratio in various regions of the lung **(Box 2.1)**.

The V/Q ratio is calculated as follows:

The minute ventilation is approximately 5 L [TV × breaths in a minute] = (500 mL × 10 breaths).

The cardiac output is approximately 6 L [heart rate (HR) × stroke volume] = (75 × 80 mL).

The V/Q ratio is 5 L/6 L = 0.8.

This ratio increases toward the apex and decreases toward the base of the lung as explained above.

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Box 2.1: Effect of gravity on ventilation-perfusion (V/Q).

In erect standing position, the V/Q ratio goes on decreasing from the apex to the base of the lung. This is because of the effect of gravity. Gravity has an influence on air as well as on blood. Hence, the ventilation, as well as perfusion, increase from the apex to the base. However, the increase in perfusion is more than the increase in ventilation from the apex to the base. The between-region differences in ventilation are much less as compared to those for blood flow across lung regions.

Hence, the ratio decreases from a preferentially ventilated apex to the preferentially perfused base. The middle zone of the lungs is considered to have relatively equal ventilation and perfusion with a mean V/Q ratio of 0.8.

Gaseous Exchange

Gas exchange is the primary function of the respiratory system and is essential for ensuring a constant supply of oxygen to the tissues as well as removing carbon dioxide to prevent its accumulation.

- It is the process by which oxygen and carbon dioxide move between the capillary blood and the lungs.
- Oxygen-deficient, carbon dioxide-rich blood returns to the right side of the heart through two large veins, the superior vena cava and the inferior vena cava. The blood enters the right side of the heart from where it is sent to the pulmonary circulation through the pulmonary artery for getting oxygenated.
- The site at which diffusion of oxygen and carbon dioxide takes place is called the *alveolar capillary membrane or barrier*. Its thickness averages to about 1 μm (1/10,000 of a centimeter, or 0.000039 inches). A detailed description of this alveolar capillary membrane is given earlier in the chapter.
- The partial pressure of oxygen in the capillary blood at the alveolar capillary membrane is lower (40 mm Hg) than the partial pressure of oxygen in the inhaled atmospheric air in the alveoli (160 mm Hg) available for gaseous exchange.
- The partial pressure of carbon dioxide in the capillary blood (46 mm Hg) at the alveolar capillary membrane is higher than the partial pressure of carbon dioxide in the inhaled atmospheric air in the alveoli (0.2 mm Hg) available for gaseous exchange.
- Both the gases physiologically flow from higher pressure to lower pressure ensuring oxygenation and expulsion of carbon dioxide from the capillary blood, both at the same time.
- This oxygenated blood then enters the left side of the heart through pulmonary veins. From here, it is pumped by the heart as cardiac output for systemic perfusion.
- The cycle continues. Arterial hypoxemia (reduced PaO_2) is caused by alveolar hypoventilation.
- It is important to note that the diffusion across the alveolar capillary membrane continuously occurs throughout life without interruption during inspiration, expiration as well as inspiratory and expiratory hold.
- The residual volume, which is the volume of air remaining inside the lung even after complete forced expiration, and the functional residual capacity (FRC), which is the volume of air remaining inside the lungs after normal quiet expiration, ensure smooth uninterrupted gaseous exchange without causing swings in the gaseous pressures. Any structural impairment of this very delicate yet efficient alveolar capillary membrane affects gaseous exchange which is reflected as altered arterial blood gases.
- A huge surface area accommodated in the small thoracic cavity makes gaseous exchange easy. As we have seen previously, this large surface area for diffusion is because of the specific structure of the tracheobronchial tree.

Anatomic Dead Space

This is defined as the volume of air that enters the airways and the lungs but does not take part in gaseous exchange.

- In order to fill the alveoli to capacity, air must incidentally occupy spaces of the terminal bronchioles, bronchi, and trachea (together called the conducting zone), but these structures do not exhibit the thin epithelial–capillary interface for gaseous exchange.
- Thus, the TV of 500 mL does not fully take part in gaseous exchange.
- Out of this 500 mL, nearly 150 mL of inhaled tidal breath remains in the conducting zone of the lungs. Thus, only 350 mL of air that is present in the respiratory zone, i.e., from the 17th generation onward, takes part in gaseous diffusion.
- The ventilation that occurs in the respiratory zone is called *alveolar ventilation*.
- The space in the conducting zone is called the anatomical dead space. The ventilation that occurs at this level is thus called *dead space ventilation*.
- In pathological conditions of the lung wherein the perfusion in an area of the lung is impaired, e.g., pulmonary embolus, the air that ventilates the affected alveoli is not able to undergo gaseous exchange due to impaired blood supply in that region due to the embolus.
- The volume of air in these unperfused areas of the lung along with anatomic dead space is called the *physiologic dead space*.
- In certain conditions of the lung such as lung collapse or lung fibrosis, the perfusion of the affected area is normal but there is absence of ventilation in the region. In such a situation, the deoxygenated blood supplied to this affected area of the lung gets passed on further without gaseous exchange. This is called an "*intrapulmonary shunt*." In this situation, a portion of the cardiac output goes from the right side of the heart to the left without getting oxygenated in the lung for the reasons mentioned above.

Clinical Pointer

A change in body position can affect the ventilation–perfusion ratio in conditions where there is presence of dead space and intrapulmonary shunt. Thus, body position has a profound influence and hence, the importance of body positioning in pulmonary pathologies.

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DYSFUNCTIONS OF THE RESPIRATORY SYSTEM

Dysfunction of the Upper Respiratory Tract

The air conditioning function of the URT gets disrupted in certain conditions:

- In conditions with obstruction in cavity, such as nasal septal deviation and space-occupying lesions in the nasal cavity, the nasal passage is bypassed and the person breathes through the mouth. The warming, humidification, and filtration of the inhaled air are compromised.
- When an endotracheal tube is inserted, the URT is bypassed. The patient either voluntarily inhales the air or if the patient is on mechanical ventilator support, the machine delivers the inhaled air. Even in this situation, the URT is bypassed and the filtration, warming, and humidification of the inhaled air need to be replaced from outside. Thus, the need for providing humidified air and use of a heat moisture exchange filter in the ventilator circuit arises.
- The URT is also bypassed in some head, neck, and face surgery in which extensive excision of tissue necessitates permanent tracheostomy. The tracheostomy opening thus needs to be covered by the permeable cover which allows inhalation of the air but at the same time filters large particles in the inhaled air. For the same reason, humidification with a nebulizer is an essential routine for such patients.
- Inflammation in the URT and the sinuses results in conditions such as rhinitis and sinusitis.

Dysfunction of the Lower Respiratory Tract

As we know, the LRT has two distinct zones and accordingly has two distinct functions:

- The first is the conducting zone from the trachea to the 14th generation of the tracheo-bronchial tree. Infection in this region may result in dilatation or fibrosis of the conducting airways, resulting in an obstruction or restriction. This results in impaired conduction of inhaled air to the respiratory zone.
- The respiratory zone extends from 16th to 23rd generation of the tracheo-bronchial tree (refer to the structure of the respiratory system above). This zone participates in gaseous exchange, i.e., uptake of oxygen from the alveolar air and expulsion of carbon dioxide and toxins from the deoxygenated blood. The ventilation in this region of the lungs is called alveolar ventilation. A reduction in alveolar ventilation results in hypoxemia due to inadequate transfer of oxygen to the capillary blood.
- Alveolar ventilation is affected in asthma. In acute exacerbation of asthma, the bronchospasm, inflammatory edema in the airway wall, and excessive secretion of goblet cells result in narrowing of the lumen of the airways. During inspiration, the work of inspiratory muscles helps to widen the airways and allows adequate inhalation. However, during expiration which is a passive process, there is an early airway closure which results in the trapping of excess air in the lungs at each breath. The alveoli are thus unable to sufficiently empty before the next inspiration, resulting in reduced alveolar ventilation and thus affecting gaseous exchange.
- Any disease that disrupts the alveolar capillary membrane such as acute respiratory distress syndrome and infections such as pulmonary tuberculosis, bronchiectasis, and fibrosis as recently observed in COVID-19 affect alveolar ventilation.
- Any thickening or widening of the area between the alveolar air and the capillary blood results in reduced gaseous exchange, e.g., pulmonary edema, pneumonia, pulmonary interstitial lung diseases, etc.
- Certain conditions resolve after medical treatment, e.g., pulmonary edema, or when the resolution of pneumonia takes place with effective antibiotics, but some conditions result in a long-lasting dysfunction as seen in fibrosis of the lung interstitium due to any cause.

Dysfunction of the Mucociliary Escalator

The dysfunction of this assembly results in the accumulation of secretion in the airways. The stagnation of such secretions impairs ventilation. The secretions stimulate the cough reflex and result in productive cough as seen in bronchiectasis and cystic fibrosis.

Since the mucociliary escalator functions throughout life, it needs certain optimal conditions to do so.

- The mucus contains 95% of its component as water, and hence inadequate hydration or excessive loss of moisture due to hyperventilation results in drying of this substance which impairs the ciliary activity.
- Periciliary fluid also needs adequate hydration and desired consistency. An excessive amount of this layer as in excessive humidification, will prevent the ciliary tip from reaching the base of the mucosal layer and hence prevent cephalad progression of the mucus. Reduced water content as in dehydration also causes ciliary dysfunction.
- This is clinically important as patients with respiratory diseases show tachypnea and lose excess fluid with each breath or cough. Those particles which cannot be expelled by this mucociliary escalator are expelled by cough reflex, which is the next defense mechanism of the respiratory system.

Impaired Cough

Cough can be impaired under many conditions:

- Cough begins with a sudden deep inspiration. This first stage of cough requires strong inspiratory muscles such as the diaphragm, sternocleidomastoid, serratus anterior, scalene, and intercostal muscles. Any condition affecting the strength of inspiratory muscles, e.g., higher center dysfunction as in opium poisoning or bulbar affection in head injury, results in insufficient cough. Neurogenic weakness can be seen in Guillain–Barre syndrome and motor neuron disease which results in low lung volumes.

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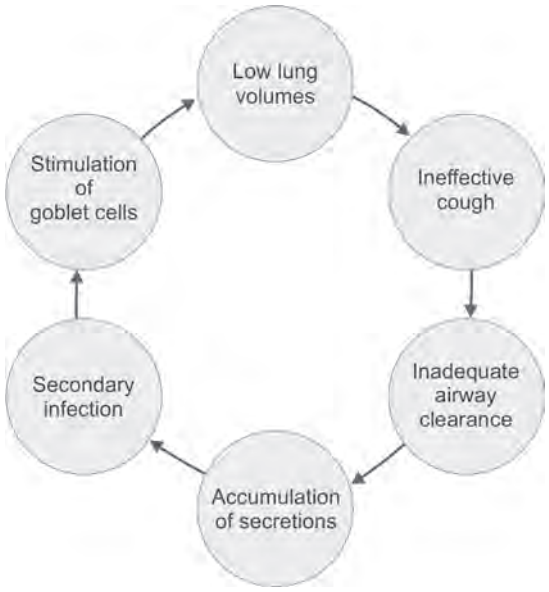


Fig. 2.4: Vicious cycle of ineffective cough.

- A sudden deep inspiration up to 90% of the total lung capacity requires a compliant lung and chest wall. Lung compliance can be reduced in restrictive conditions of the lung and chest wall, which greatly reduces the lung volume. Airflow limitation may cause the cough to be weak and/or ineffective. In bronchiectatic segments of the airways, there is lack of airflow and hence the cough is ineffective.
- The final stage of cough is the forceful expulsion of trapped air at high speed. This requires strong muscles of expiration such as the abdominal muscles, trunk muscles, and intercostal muscles. Weak abdominal muscles as seen in quadriplegia, paraplegia, and hemiplegia cannot produce the desired expiratory force. Inadequate and hence ineffective cough results in accumulation of secretions which results in secondary infection which in turn results in increased activation of goblet cells, thus forming a vicious cycle **(Fig. 2.4)**. Expiratory muscle weakness decreases the cough-induced dynamic compression of the airways rendering the cough ineffective.
- Pain because of conditions such as surgical incision or fractured ribs can suppress cough.

Respiratory System Dysfunction in Obstructive Lung Diseases

The cause of obstruction varies in different obstructive lung conditions, such as asthma and bronchitis.

Asthma and Bronchitis

- There is a spasm of the bronchial smooth muscles, edema in the bronchial wall, and increased activity of the goblet cells causing hypersecretion of mucus resulting in narrowing of the bronchial lumen.
- This is of less significance during inspiration as the force generated by the inspiratory muscles can overcome it.
- However, during expiration the force of smooth muscle spasm and the elastic recoil, both are directed towards narrowing of the airway lumen.
- This results in early airway closure during expiration and hence air trapping.
- Thus, there is inadequate emptying of the lungs at the end of expiration which adversely affects alveolar ventilation.

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is a group of lung diseases which demonstrate irreversible obstruction of the airways. COPD includes emphysema and chronic bronchitis. The cause of obstruction in chronic bronchitis is similar to bronchitis as explained above.

- The characteristic pathological changes in COPD include chronic inflammation, increased number of specific inflammatory cell types in different parts of the lung, and structural changes resulting from repeated injury and subsequent repair. These changes are found to persist even after cessation of smoking.
- The hallmarks of pathological changes within the ECM in COPD, including emphysema, are the extensive destruction of elastic fibers by ECM-degrading enzymes released by inflammatory cells, mainly macrophages, neutrophils, and T cells.
- Also, emphysema is characterized by the destruction of the alveoli. Many alveoli coalesce to form one big alveolus resulting in a reduced surface area for diffusion.
- The pathophysiological aspects of COPD, which are mainly caused by smoke exposure, include airflow obstruction and hyperinflation.
- The airflow limitation in COPD is caused by three interrelated processes:
  1. Small airway remodeling leading to a thickening of airway walls
  2. Loss of small airways
  3. Enlargement of the respiratory air spaces in the alveoli, a condition called *emphysema*
- Consequently, the transpulmonary pressure dissemination is disrupted, causing hyperinflation of the lungs. These structural alterations cause biomechanical changes that manifest as loss of the elastic recoil of the lung.
- As seen previously, the ECM represents the main stress-bearing component in the respiratory system; the loss of elastic recoil in pulmonary emphysema suggests that elastin, the core component within elastic fibers, is the major target of decomposition.
- The result is an excessively compliant lung in addition to loss of elasticity leading to loss of radial traction and consequent narrowing. Such a lung is unable to maintain the lumen of the airways patent till the desired amount of air is allowed to escape from the lungs during exhalation.

Thus, obstructive diseases show the presence of ventilatory dysfunction as well as dysfunction of gaseous exchange.

- Another cause of ventilatory dysfunction in COPD is weakness of the respiratory muscles.

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- COPD is a primary lung condition, but its systemic manifestations have also been observed. Systemic inflammation and chronic hypoxia are common features in COPD. This is one of the few causes of respiratory muscle weakness in COPD.
- The trapping of air, due to early airway closure, results in hyperinflation of the lungs. This places the diaphragm lower in the abdominal cavity rendering it flat and shortened, causing weakness of the diaphragm. This enforced caudal position of the diaphragm alters its angle of pull during inspiration. Instead of pulling the lower rib cage upward and outward during inspiration, as in normal, it pulls the ribs inward and downward, which is clinically seen as *Hoover's sign*.
- Similarly, due to air trapping, the rib cage is unable to assume its normal end-expiratory dimension and alignment. The increased intercostal spaces even at the end of expiration put the intercostal muscles at a mechanical disadvantage for the next inspiration.
- The intercostal muscles remain lax, and hence we see intercostal indrawing during inspiration due to sucking in of these lax intercostal spaces because of negative intrathoracic pressure during inspiration.
- Consequently, there is outward bulging of the intercostal spaces during expiration as the intrathoracic pressure increases during expiration, thus resulting in increased work of breathing, which further challenges the muscles of inspiration to work harder thus further weakening them.

Exercise Tolerance in Chronic Obstructive Pulmonary Disease

Ventilation–perfusion inequality is inevitable in COPD and leads to hypoxemia with or without carbon dioxide retention.

- Exercise capacity is reduced in obstructive conditions due to the dynamic hyperinflation which results from the increase in minute ventilation.
- The end-expiratory lung volume increases during exercise. This greatly compromises the lung expansion and causes dyspnea.

Dynamic Hyperinflation

During normal quiet breathing, i.e., TV breathing, the FRC is maintained constant and hence inspiratory capacity is maintained and not compromised.

- With exercise, the demand for the respiratory system increases which is provided by an increase in the respiratory rate.
- Increased respiratory rate reduces the expiratory time resulting in trapping of air within the lungs during exercise. It increases the end-expiratory lung volume and compromises the inspiratory capacity which limits the exercise capacity of the individual. This is called dynamic hyperinflation.

Clinical Pointer

Pursed lip breathing produces a weak positive pressure in the mouth during expiration and helps to control this dynamic hyperinflation to a certain extent as it allows emptying of the lungs.

Respiratory Dysfunction in Restrictive Lung Disease

Restrictive lung diseases are those in which there is restriction of the expansion of the lung.

- This restriction is either because of alteration in the lung parenchyma or because of disease of the pleura, chest wall, or neuro-musculoskeletal apparatus.
- Thus the lung volumes are reduced, but the airway resistance (related to the lung volume) is not increased which differentiates it from obstructive diseases.
- The restriction is clinically evident as reduced chest expansion which can be measured and a rapid shallow breathing with use of accessory muscles which can be observed.

Restrictive diseases can be classified based on the cause of restriction as restrictive diseases of the lung and restrictive diseases of the chest wall.

Restrictive Diseases of the Lung

Examples of restriction of the lung include diffuse interstitial fibrosis, pulmonary tuberculosis, pleural effusion, atelectasis, fibrosis of pleura, intrathoracic space-occupying lesions compressing the lung, endobronchial tumors, and pulmonary edema.

Diffuse interstitial fibrosis

- The primary functional impairment in fibrosis is affection of the gaseous exchange at the alveolar-capillary membrane.
- Diffuse interstitial fibrosis is also referred to as idiopathic pulmonary fibrosis, interstitial pneumonia, and cryptogenic fibrosing alveolitis. The end result is fibrosis which is typical of many pulmonary conditions such as interstitial lung disease, sarcoidosis, scleroderma, pneumoconiosis, cystic fibrosis, and others. Certain drugs used as medications for other ailments cause interstitial fibrosis. Examples of such drugs are busulfan, nitrofurantoin, amiodarone, bleomycin, and some antineoplastic drugs. Oxygen, when given in high concentration, causes acute toxic changes with subsequent interstitial fibrosis.
- In interstitial fibrosis, there is thickening of the interstitium of the alveolar wall. Fibroblasts appear to lay down thick collagen bundles. Eventually, there is destruction of the alveolar architecture and the scarring ends up with multiple air-filled spaces formed by dilated terminal and respiratory bronchioles. It is referred to as "honey comb" appearance of the lung.
- In interstitial pulmonary fibrosis (IPF), the repetitive lung injury and subsequent repair mechanisms lead to ongoing destruction of the elastic parenchymal lung tissue, which is constantly replaced by stiff scar tissue, comprising large and condensed cell–ECM aggregates known as fibroblastic foci within the pulmonary interstitium. Progressive tissue remodeling leads to traction bronchiectasis, thickened interlobular septae, and subpleural honeycombing. All these microscopic and macroscopic changes contribute to a progressive loss in gas exchange, with subsequent deteriorating lung function, breathlessness; finally, leading towards respiratory failure.

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Impaired exercise tolerance in interstitial fibrosis

- Patients with mild diffuse interstitial fibrosis may show impaired pulmonary function on exercise than at rest. The maximal oxygen uptake and carbon dioxide output are severely reduced. As the exercise progresses, the limited TV greatly increases the respiratory rate. The impaired diffusion is an important feature of the lung due to thickening of the interstitium. It results in ventilation–perfusion mismatch.
- Another factor responsible for reduced exercise tolerance in patients with restrictive disorder is the small rise in cardiac output. Much of the capillary bed gets obliterated with interstitial fibrosis resulting in increased pulmonary vascular resistance. This further greatly increases on exercise resulting in less increase in cardiac output. An abnormally low cardiac output in the presence of ventilation–perfusion mismatch results in hypoxemia.

Pulmonary tuberculosis

Pulmonary tuberculosis (TB) is very common in India.

- A postpulmonary tubercular sequel is a fibrocavitatory lesion with dilatation of airways resulting in a mixed obstructive restrictive picture.
- Post-tubercular bronchiectasis also poses impaired airway clearance as discussed earlier.

Pleural effusion

- Pleural effusion is collection of pleural fluid in the intrapleural space.
- A longstanding pleural effusion results in a rigid contracted fibrotic pleura that splints the lung and prevents its expansion.

Other restrictive conditions of the lung include atelectasis, fibrosis of pleura, intrathoracic space-occupying lesions compressing the lung, endobronchial tumors, and pulmonary edema compromising the air entry into the lungs.

Thus, in restrictive conditions of the lung, the chest expansion is found to be reduced even when the thoracic movements or control of respiration is not impaired.

Restrictive Diseases of the Chest Wall

In these conditions, the lung may be unimpaired but the restriction is imposed by the inability of the rib cage to perform desired rib cage movements due to various reasons. This can be seen in impaired neural control or restriction of the rib cage.

Examples of impaired neural control are as follows:

- Suppression of higher centers controlling respiration
- Spinal cord injury
- Myasthenia gravis wherein the neuromuscular junction is impaired
- Peripheral neuropathy like Guillain–Barre syndrome

Examples of restriction of the skeletal component of the rib cage are as follows:

- Kyphoscoliosis
- Ankylosing spondylitis
- Obesity
- Chest burns

Impairments in Some Restrictive Conditions

Spinal cord injury: The dysfunction caused by spinal cord injury depends on the level of injury.

- One of the main causes of disability in spinal cord injury is due to diaphragm dysfunction. In high spinal cord injury, the diaphragm is unable to contract due to its paralysis and the patient requires immediate mechanical ventilator support. In cervical injury below the fifth cervical level, there will be paralysis of abdominal and other accessory muscles but the diaphragm will be spared. This is seen as paradoxical breathing of the upper chest.
- It is important to evaluate the ability of the diaphragm to move. This can be done by placing the fingertips just below the lower border of the rib cage anteriorly bilaterally approximately 6–8 cm laterally to the xiphoid process during breathing. As the person inhales, the fingertips of both the hands are pushed away from the lower ribs indicating the downward descent of the diaphragm.
- Fatigue of the diaphragm is a common feature of respiratory conditions and contributes to hypoventilation.

Restriction of the chest wall is evident in kyphoscoliosis, girdle burns over the chest that have healed with scarring, paralysis of the muscles of respiration, rheumatoid arthritis, ankylosing spondylitis, obesity, thoracoplasty, median sternotomy for coronary artery bypass grafting, pectus excavatum, etc.

- Chest wall diseases, such as both scoliosis and kyphosis, have the potential to cause restriction of the lung. They alter the alignment of the rib cage, neck, and shoulders which places not only the primary but also the accessory muscles of respiration at a mechanical disadvantage.
- In ankylosing spondylitis, the gradual but progressive immobility of the vertebral joints and the costovertebral joints reduces the bucket handle and pump handle movements of the ribs which are so important for normal lung expansion.
- Ankylosing spondylitis also shows systemic inflammation and hence weakness of skeletal and respiratory muscles.
- In restrictive conditions, the resultant hypoventilation favors lung infections increasing the morbidity and hence the disability.

Measurement of Lung Dysfunction

The lung dysfunctions need to be identified and documented for diagnostic and prognostic purposes. Documentation of respiratory dysfunction includes the following:

- Spirometry findings
- Gas exchange characteristics
- Diffusing capacity
- Exercise tolerance capacity

As has been pointed out before, the respiratory function has a great impact on the exercise performance. At rest, the normal lung has an enormous reserve of functions.

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The oxygen uptake and carbon dioxide output can be increased 10-fold under normal circumstances. Most importantly, these increases occur without any fall in arterial pO_2 or a rise in arterial pCO_2. To explore minor dysfunction of gaseous exchange, we need to utilize the stress of exercise.

Self-assessment of disability by the patient is often variable and may be misguiding. Exercise tests provide a documentable objective measure of disability imposed by the respiratory disorder on the patient. In patients with respiratory disorders, submaximal tests, like the 6-minute walk test or the shuttle walk test, can prove more practical than the gold standard treadmill test using standard protocols. This is because of the respiratory limitation of exercise coming into play earlier than the desired target HR.

In the recent COVID-19 pandemic, physiotherapy guidelines highlight the role of desaturation after the 6-minute walk test as an indication of impaired gaseous exchange and advocate its implication in clinical decision-making.

- *Assessment of neuromuscular dysfunction*: Neuromuscular dysfunction of the respiratory system can be assessed by strength of the respiratory muscles in terms of the maximum negative and positive mouth pressures created during forceful inspiration and expiration, respectively. The maximum inspiratory pressure (MIP) helps to understand the capacity of the inspiratory muscles to create negative mouth pressure during forced deep inspiration. Maximum expiratory pressure (MEP) measures the strength of expiratory muscles.
- *Assessment of quality of life*: Chronic respiratory dysfunction has a profound impact on the quality of life of the person with a respiratory disease. For the generic measure of quality of life, questionnaires given below can be of help:
  - WHO Disability Assessment Schedule (WHO-DAS 2.0) *or*
  - Short Form Health Survey (SF-36) developed by RAND

  Disease-specific questionnaires would be:
  - Chronic respiratory disease questionnaire (CRDQ)* *or*
  - St George's Respiratory Questionnaire (SGRQ)#

Both these questionnaires need a licensing agreement and a fee is required to use the questionnaire. The contact address is given in the reference section.

Summary

Understanding the anatomy, function, and pathophysiology helps us to identify the clinical reasoning for any dysfunction. This makes appropriate clinical decision-making easy. This approach reveals that although the obstructive and restrictive conditions have similar symptoms, such as dyspnea, reduced exercise tolerance, and respiratory muscle fatigue, their cause is different and hence the therapeutic approach will be different.

REVIEW QUESTIONS

1. What are the defense mechanisms offered by the respiratory system in humans?
2. Discuss the mechanism of the mucociliary escalator.
3. Explain the structure of lung interstitium.
4. Explain the biomechanics of the diaphragm during inspiration.
5. Explain the concept of dead space and shunt.
6. Explain the various phases of cough and the importance of cough in maintaining airway clearance.
7. What are the causes of reduced exercise tolerance in obstructive and restrictive disorders?
8. How are lung volumes and flows affected in obstructive and restrictive lung disorders?
9. What are the objective measures to assess lung function?

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* CRDQ/CRQ: Please contact: Jane Howe, Licensing Officer, Office of Research Contracts & Intellectual Property, McMaster University, GH 306G, 1280 Main Street, W. Hamilton, Ontario, CANADA L8S 4L8, Telephone: 1-877-836-9235. Fax: 905-540-8019 Email: orcip@mcmaster.ca

St George's Respiratory Questionnaire. St George's Health Status Research Team, Department of Physiological Medicine, St George's Hospital Medical School, London SW17 ORE UK. Please contact Yvonne Forde at sgrq@sgul.ac.uk http://www.healthstatus.sgul.ac.uk/downloads/sgrq_scoring_sheet.htm Last seen 7/10/2020. https://tbcindia.gov.in/WriteReadData/India%20TB%20Report%202019.pdf

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Physiotherapy in Cardiopulmonary Conditions

The goal of the authors when writing this book was to provide a textbook and a reference book for students, clinicians and academicians. It contains important and common cardiopulmonary dysfunctions encountered amongst patients. The book is divided into six sections depending on the content of the chapters comprising details of foundational concepts, clinical evaluation and investigations, physiotherapy techniques used in cardiopulmonary rehabilitation, common cardiopulmonary dysfunctions and rehabilitation. Clinical examples provide a clear understanding of the assessment and management processes. It is a unique depiction of all topics related to cardiopulmonary medicine and physiotherapy.

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- Clinical examples for ease of understanding
- Review questions for exam going students.

Mariya Jiandani MSc (PT) FAIMER fellow PhD guide is an Associate Professor at Physiotherapy School and Centre, Seth Gordhandas Sunderdas Medical College and the King Edward Memorial Hospital, Mumbai, India. She has been working in the field of Cardiorespiratory Physiotherapy for more than 30 years.

Megha Sandeep Sheth MPT PhD guide is Lecturer at SBB College of Physiotherapy, Ahmedabad, India, since 20 years, performing EMG/NCV at the institute since almost 7 years. She has over 100 publications in national and international journals.

Neepa Hiren Pandya PT PhD is active in the field of cardiopulmonary physiotherapy since last 25 years. She is working as an associate physiotherapist at Zydus Hospital, Vadodara, Gujarat, India. She is a renowned clinician and academician in the field of cardiorespiratory and vascular physiotherapy.

Priya Singh Rangey MPT (Rehabilitation) PhD Scholar is an Assistant Professor at LJ Institute of Physiotherapy, LJ University, Ahmedabad, Gujarat, India with an academic and clinical experience of 7 years. She has several publications in national and international journals.

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