



GUIDELINES

For

GENERAL PRACTITIONERS

2024

Press record

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FOREWORD

It is a great honor for me to write a foreword to [Guidelines for General Practitioners](#) by General Practitioners' society, Myanmar Medical Association (Central).

General practitioners are the primary health providers in the community looking after the majority of the people of our country. They are being trusted and depend upon by every families in the surrounding area where they practice. The first and foremost care by the General Practitioners are the most important for all the people.

Guidelines based on a critical appraisal of scientific evidence (evidence-based guidelines) clarify which interventions are of proved benefit and document the quality of the supporting data. They alert clinicians to interventions unsupported by good science, reinforce the importance and methods of critical appraisal, and call attention to ineffective, dangerous, and wasteful practices.

Clinical guidelines can improve the quality of clinical decisions. They offer explicit recommendations for clinicians who are uncertain about how to proceed, overturn the beliefs of doctors accustomed to outdated practices, improve the consistency of care, and provide authoritative recommendations that reassure practitioners about the appropriateness of their treatment policies.

The Myanmar Medical Association together with the GP society has been helping out with the CME and CPD program for the Member doctors both inhouse sessions and online courses. This guideline is one of the essential parts of this CPD for the GPs.

I would like to congratulate the GP society for their effort for producing this guideline and also, I would like to encourage them to review and updated regularly.



Professor Aye Aung
President

Myanmar Medical Association

April, 2024

PREFACE

We are writing this letter to express our sincerest gratitude and appreciation for the successful completion of the **second edition** of the **General Practitioners' Guidelines**. This accomplishment is the result of an exceptional collaborative effort, and we would like to extend our thanks to all those involved.

The General Practitioners' Guidelines has been an invaluable resource since its inception with the launch of the first edition in November 2017. As per the initial plan, the guidelines were intended to be updated every three years to ensure the most up-to-date information reaches Myanmar General Practitioners, enhancing their knowledge in primary healthcare and family health.

However, the unforeseen outbreak of the Covid-19 pandemic disrupted our plans and posed numerous challenges for the team. In-person meetings became impossible due to safety concerns, making it necessary for us to find alternative means of communication and collaboration. Despite the adversity faced, the team members demonstrated remarkable resilience and adaptability by utilizing online platforms and technology to continue the update process.

We would like to extend our deepest gratitude to the dedicated team members who persevered and worked tirelessly during these trying times. Their commitment, professionalism, and unwavering dedication to the project enabled us to overcome the obstacles posed by the pandemic and successfully complete the second edition of the guideline.

Furthermore, we would like to express our sincere appreciation to the specialist societies that actively contributed to the development of the guidelines. Their expertise and invaluable insights have ensured that the content remains current, accurate, and relevant, enabling our General Practitioners to provide the highest quality of care to their patients.

We would also like to extend our heartfelt thanks to the esteemed President of the Myanmar Medical Association, for their continuous support and guidance throughout this endeavor. Their leadership and unwavering commitment to advancing medical knowledge in Myanmar have been instrumental in the success of this Guidelines.

Moreover, the decision to distribute the guideline as electronic copies reflects our commitment to ensuring easy access for all Myanmar General Practitioners. By making it available in this format, we aim to facilitate the dissemination of updated knowledge, thus empowering our healthcare professionals to deliver the best possible care to the community.

In conclusion, we would like to express our deepest gratitude to all those who contributed to the development and distribution of the General Practitioners' Guidelines Second Edition. The unwavering supports and collective efforts have made a significant impact on enhancing primary healthcare and family health care in Myanmar.

Once again, thank you for your outstanding dedication, resilience, and invaluable contributions. We look forward to our continued collaboration in advancing medical knowledge and improving healthcare outcomes for all.

Dr Khine Soe Win and Dr Win Zaw
General Practitioners' Society (Central)
Myanmar Medical Association

April, 2024

EDITORIAL

It is my privilege to inform you that our updated and revised edition of “**Guidelines for General Practitioners**” will be published very soon and it is my great pleasure to be the editor-in-chief of this guideline book. There are various reasons for revising and updating the previous edition.

This is the fact that some important topics, for example, malaria and family violence are missing in the first edition and some clinical practice guidelines like Diabetes Management have been changed during the interim period. Of course, this opportunity arises due to the emergence of COVID-19 in the world. As all you know, Medicine is an ever-changing science; we need to consider updating our guidelines at least five- yearly. Hence the time is up now!

Education is achieved by assimilating information from many resources and readers of this book can enhance their learning experience in terms of reflecting in their daily Family/General Practice. We all take immense pride in contributing good educational resource dedicated to Myanmar General Practitioners. The editors and authors anticipate that the readers will both enjoy and profit from their work in preparing this volume.

Happy studying and learning,

Dr Win Lwin Thein
Editor-in chief
Vice President (GP Society)
April, 2024

ACKNOWLEDGEMENT

We would like to thank all our talented and hard-working colleagues who have contributed to the ongoing development of the **Guidelines for General Practitioners**.

Especially, we would like to highlight the significance of the second edition which appears when the family medicine development process in Myanmar is being idle. Many factors are impeding the developing process lately, which has been accelerated previously by the commitment of the MOHS, the medical universities, and the General Practitioners' Society before the COVID-19 pandemic started.

No one can deny that the Myanmar health care system is lacking a strong and effective primary care task force. The best solution to mend this defect is retraining the thousands of general practitioners who are working individually across the country. Here comes the role of family medicine to train these GPs and primary care doctors to be able to use its principles effectively and, in turn, strengthen primary care.

Many GPs are using some family medicine principles consciously or unconsciously in varying degree of competency. Person-centered care, continuity of care, and family-oriented care became the culture of most practices for a long time. But only a few GPs can enjoy the most effective coordinated care and seamless continuity of care with secondary and tertiary care providers. The reasons behind this would be the absence of standardization in general practitioners' service quality and unawareness of the value of family medicine practitioners by other specialties and the public.

To resolve this ambiguity, primary care doctors should be involved in the retraining programs and thereafter CME/CPD and other life-long-learning programs which prescribe family medicine curricula.

We also acknowledge the effort of the contributors to make this new edition more family medicine-oriented, in addition to the Family Medicine chapter at the beginning of the book. We genuinely believe that the new edition will be a better reference for the GP/FP who wants to practice quality primary care and for future family medicine programs in Myanmar.

Finally, we would like to thank all academic writers who contributed to the General Practice Guidelines-first edition. Without their kind support, this second edition could never have happened.

Regards,

Dr. Tin Aye and Dr. Kyaw Thu

General Practitioners' Society (Central), MMA

April, 2024

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SYMBOLS AND ABBREVIATIONS

AAA abdominal aortic aneurysm	COAD chronic obstructive airways disease
ABC airway, breathing, circulation	COC combined oral contraceptive
ABCD airway, breathing, circulation, dextrose	COCP combined oral contraceptive pill
ABO A, B and O blood groups	COPD chronic obstructive pulmonary disease
ACE angiotensin-converting enzyme	COX cyclooxygenase
ACEI angiotensin-converting enzyme inhibitor	CPA cardiopulmonary arrest
ACTH adrenocorticotrophic hormone	CPAP continuous positive airways pressure
ADHD attention deficit hyperactivity disorder	CPK creatine phosphokinase
ADT adult diphtheria vaccine	CPR cardiopulmonary resuscitation
AFP alpha-fetoprotein	CR controlled release
AI aortic incompetence	CREST calcinosis cutis; Raynaud's phenomenon; oesophageal involvement; sclerodactyly; telangiectasia
AIDS acquired immunodeficiency syndrome	CRF chronic renal failure
AHRA angiotensin II (2) reuptake antagonist	CR(K)F chronic renal (kidney) failure
AKF acute kidney failure	CRP C-reactive protein
ALE average life expectancy	CSF cerebrospinal fluid
ALL acute lymphocytic leukaemia	CT computerised tomography
ALP alkaline phosphatase	CTS carpal tunnel syndrome
ALT alanine aminotransferase	CVA cerebrovascular accident
AMI acute myocardial infarction	CVS cardiovascular system
AML acute myeloid leukaemia	CXR chest X-ray
ANA antinuclear antibody	DBP diastolic blood pressure
ANF antinuclear factor	DC direct current
AP anterior-posterior	DHA docosahexaenoic acid
APH ante-partum haemorrhage	DI diabetes insipidus
ASD atrial septal defect	DIC disseminated intravascular coagulation
ASIS anterior superior iliac spine	dL decilitre
ASOT antistreptolysin O titre	DMARDs disease modifying antirheumatic drugs
AST aspartate aminotransferase	DNA deoxyribose-nucleic acid
AV atrioventricular	DRABC defibrillation, resuscitation, airway, breathing, circulation
AZT azidothymidine	drug dosage bd—twice daily, tid/tds -three times daily, qid/qds -four times daily
BCC basal cell carcinoma	ds double strand
BCG bacille Calmette-Guérin	DS double strength
BMD bone mass density	DSM diagnostic and statistical manual (of mental disorders)
BMI body mass index	DU duodenal ulcer
BP blood pressure	DUB dysfunctional uterine bleeding
BPH benign prostatic hyperplasia	DVT deep venous thrombosis
Ca carcinoma	EBM Epstein-Barr mononucleosis (glandular fever)
CABG coronary artery bypass grafting	EBV Epstein-Barr virus
CAD coronary artery disease	ECG electrocardiogram
CAP community acquired pneumonia	ECT electroconvulsive therapy
CBT cognitive behaviour therapy	EDD expected due date
CCF congestive cardiac failure	EEG electroencephalogram
CCU coronary care unit	ELISA enzyme linked immunosorbent assay
CD4 T helper cell	ESRF end-stage renal failure
CD8 T suppressor cell	ESR(K)F end stage renal (kidney) failure
CDT combined diphtheria/tetanus vaccine	ERCP endoscopic retrograde cholangiopancreatography
CEA carcinoembryonic antigen	esp. especially
CFS chronic fatigue syndrome	ESR erythrocyte sedimentation rate
CHD coronary heart disease	FB foreign body
CHF chronic heart failure	FBE full blood count
CIN cervical intraepithelial neoplasia	
CK creatinine kinase	
CKD chronic kidney disease	
CKF chronic kidney failure	
CML chronic myeloid leukaemia	
CMV cytomegalovirus	
CNS central nervous system	

FEV1 forced expiratory volume in 1 second
fL femtolitre = (1e-15) litre
FSH follicle stimulating hormone
FUO fever of undetermined origin
FVC forced vital capacity
g gram
GA general anaesthetic
GABHS group A beta-haemolytic streptococcus
GBS Guillain-Barré syndrome
GFR glomerular filtration rate
GI glycaemic index
GIT gastrointestinal tract
GLP glucagon-like peptide
GnRH gonadotrophin-releasing hormone
GO gastro-oesophageal
GORD gastro-oesophageal refl ux
GP general practitioner
G-6-PD glucose-6-phosphate
GU gastric ulcer
HAV hepatitis A virus
anti-HAV hepatitis A antibody
Hb haemoglobin
HbA haemoglobin A
anti-HBc hepatitis B core antibody
HBeAg hepatitis B e antigen
anti-HBs hepatitis B surface antibody

HBsAg hepatitis B surface antigen
HBV hepatitis B virus
HCG human chorionic gonadotropin
HCV hepatitis C virus
anti-HCV hepatitis C virus antibody
HDL high-density lipoprotein
HEV hepatitis E virus
HFM hand, foot and mouth
HFV hepatitis F virus
HGV hepatitis G virus
HIV human immunodeficiency virus
HNPCC hereditary nonpolyposis colorectal cancer
HPV human papilloma virus
HRT hormone replacement therapy
HSV herpes simplex viral infection
IBS irritable bowel syndrome
ICE ice, compression, elevation
ICS inhaled corticosteroid
ICS intercondylar separation
ICT immunochromatographic test
IDDM insulin dependent diabetes mellitus
IDU injecting drug user
IgE immunoglobulin E
IgG immunoglobulin G
IgM immunoglobulin M
IHD ischaemic heart disease
IM, IMI intramuscular injection
inc. including
IPPV intermittent positive pressure variation
IR internal rotation
ITP idiopathic (or immune) thrombocytopenia
 purpura
IUCD intrauterine contraceptive device
IUGR intrauterine growth retardation

IV intravenous
IVI intravenous injection
IVP intravenous pyelogram
IVU intravenous urogram
JCA juvenile chronic arthritis
JVP jugular venous pulse
KA keratoacanthoma
kg kilogram
KOH potassium hydroxide
LA local anaesthetic
LABA long acting beta agonist
LBBB left branch bundle block
LBO large bowel obstruction
LBP low back pain
LDH/LH lactic dehydrogenase
LDL low-density lipoprotein
LFTs liver function tests
LH luteinising hormone
LHRH luteinising hormone releasing hormone
LIF left iliac fossa
LMN lower motor neurone
LNG levonorgestrel
LRTI lower respiratory tract infection
LSD lysergic acid
LUQ left upper quadrant
LUTS lower urinary tract symptoms
LV left ventricular
LVH left ventricular hypertrophy
mane in morning
MAOI monoamine oxidase inhibitor
mcg microgram (also µg)
MCV mean corpuscular volume
MDI metered dose inhaler
MDR multi-drug resistant TB
MI myocardial infarction
MRCP magnetic resonance cholangiography
MRI magnetic resonance imaging
MS multiple sclerosis
MSM men who have sex with men
MSU midstream urine
N normal
NAD no abnormality detected
NGU non-gonococcal urethritis
NHL non-Hodgkin's lymphoma
NIDDM non-insulin dependent diabetes mellitus
nocte at night
NSAIDs non-steroidal anti-inflammatory drugs
NSU non-specific urethritis
(o) taken orally
OA osteoarthritis
OCP oral contraceptive pill
OGTT oral glucose tolerance test
OSA obstructive sleep apnoea
OTC over the counter
PA posterior–anterior
PAN polyarteritis nodosa
Pap Papanicolaou
pc after meals
PCA percutaneous continuous analgesia
PCB post coital bleeding

PCL posterior cruciate ligament
PCOS polycystic ovarian syndrome
PCP pneumocystis carinii pneumonia
PCR polymerase chain reaction
PCV packed cell volume
PDA patent ductus arteriosus
PEF peak expiratory flow
PEFR peak expiratory flow rate
PET pre-eclamptic toxemia
PFT pulmonary function test
PH past history
PID pelvic inflammatory disease
PLISSIT permission: limited information: specific suggestion: intensive therapy
PMS premenstrual syndrome
PMT premenstrual tension
POP plaster of Paris
POP progestogen-only pill
PPI proton-pump inhibitor
PPROM preterm premature rupture of membranes
PR per rectum
prn as and when needed
PROM premature rupture of membranes
PSA prostate specific antigen
PSIS posterior superior iliac spine
PSVT paroxysmal supraventricular tachycardia
PT prothrombin time
PTC percutaneous transhepatic cholangiography
PU peptic ulcer
PUO pyrexia of undetermined origin
pv per vagina
qds, qid four times daily
RA rheumatoid arthritis
RBBB right branch bundle block
RBC red blood cell
RCT randomised controlled trial
RF rheumatic fever
Rh rhesus
RIB rest in bed
RICE rest, ice, compression, elevation
RIF right iliac fossa
RPR rapid plasma reagin
RR relative risk
RSV respiratory syncytial virus
RT reverse transcriptase
rtPA recombinant tissue plasminogen activator
SAH subarachnoid haemorrhage
SARS severe acute respiratory distress syndrome
SBE subacute bacterial endocarditis
SBO small bowel obstruction
SBP systolic blood pressure
SC/SCI subcutaneous/subcutaneous injection
SCC squamous cell carcinoma
SCG sodium cromoglycate
SIADH syndrome of secretion of inappropriate antidiuretic hormone
SIDS sudden infant death syndrome
SIJ sacroiliac joint
SL sublingual
SLE systemic lupus erythematosus
SLR straight leg raising
SND sensorineural deafness
SNHL sensorineural hearing loss
SNRI serotonin noradrenaline reuptake inhibitor
SOB shortness of breath
sp species
SR sustained release
SSRI selective serotonin reuptake inhibitor
SSS sick sinus syndrome
stat at once
STI sexually transmitted infection
SVC superior vena cava
SVT supraventricular tachycardia
T3 tri-iodothyronine
T4 thyroxine
TB tuberculosis
tds, tid three times daily
TENS transcutaneous electrical nerve stimulation
TFTs thyroid function tests
TG triglyceride
TIA transient ischaemic attack
TIBC total iron binding capacity
TM tympanic membrane
TMJ temporomandibular joint
TNF tissue necrosis factor
TOF tracheo-oesophageal fistula
TORCH toxoplasmosis, rubella, cytomegalovirus, herpes virus
TPHA Treponema pallidum haemagglutination test
TSE testicular self-examination
TSH thyroid-stimulating hormone
TT thrombin time
TV tidal volume
U units
UC ulcerative colitis
U & E urea and electrolytes
µg microgram
UMN upper motor neurone
URTI upper respiratory tract infection
US ultrasound
UTI urinary tract infection
U ultraviolet
VC vital capacity
VDRL Venereal Disease Reference Laboratory
VF ventricular fibrillation
VMA vanillyl mandelic acid
VSD ventricular septal defect
VT ventricular tachycardia
VUR vesico-ureteric reflux
VWD von Willebrand's disease
WBC white blood cells
WCC white cell count
WHO World Health Organization
WPW Wolff-Parkinson-White
XL sex linked

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Respiratory Problems

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CHAPTER 4

RESPIRATORY PROBLEMS

Chapter (4)

Respiratory Problem

1. Asthma In Adults
2. Chronic Obstructive Pulmonary Disease
3. Acute Respiratory Infection (ARI)
4. Pneumonia In Adults
5. Bronchiectasis
6. Pleural Effusion
7. Pneumothorax
8. Pulmonary Embolism
9. Respiratory Failure
10. Lung Cancer

ASTHMA IN ADULTS

DEFINITION

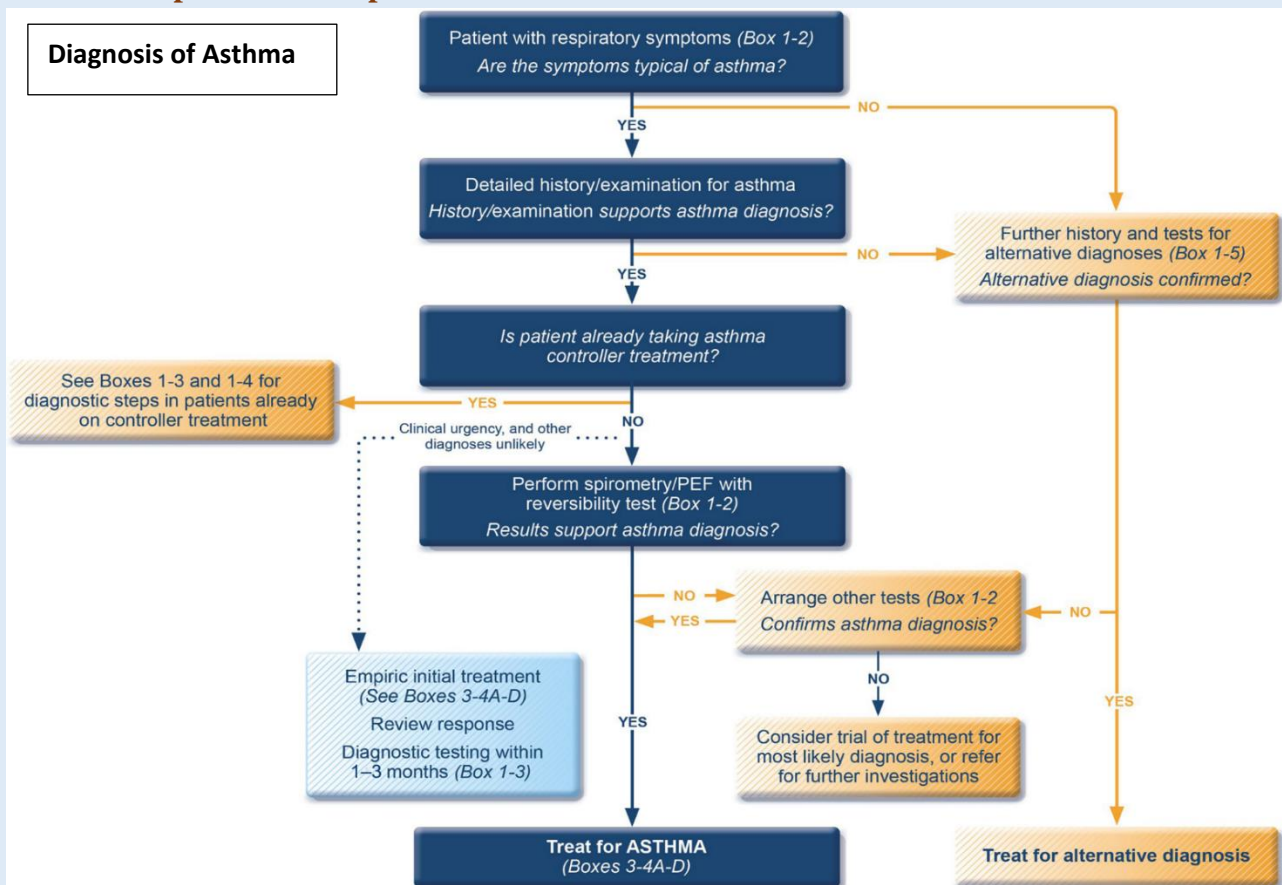
Asthma is a condition of paroxysmal reversible airways obstruction and has three characteristic features

- Airflow limitation - usually reversible spontaneously or with treatment
- Airway hyper-responsiveness to a wide range of stimuli
- Inflammation of bronchi.

DIAGNOSIS OF ASTHMA

Clinical features that increase probability of asthma > 1 of the following:

- **Wheeze**
- **Breathlessness**
- **Chest tightness**
- **Cough particularly if symptoms are worse:**
 - o at night/early morning
 - o with exercise, allergen and/or cold air exposure
 - o after aspirin/ blocker
- **Past History of atopy**
- **Family History of asthma and/or atopy**
- **Widespread wheeze**
- **Unexplained low FEV₁ or PEFR**
- **Unexplained eosinophilia**



Ref: <https://www.nature.com/articles/s41533-023-00330-1/figures/1>

SYMPTOMS/SIGNS OF A SEVERE ASTHMA ATTACK

- PEFR 30-50% predicted or best
- O₂ saturation \geq 92%
- unable to talk in sentences
- intercostal recession
- tachypnoea, respiratory rate $>$ 25 breaths/min
- tachycardia, heart rate \geq 110 bpm

LIFE-THREATENING SIGNS

- PEFR $<$ 33% predicted or best
- O₂ saturation $<$ 92%
- Arrhythmia
- Hypotension
- Cyanosis
- Exhaustion
- Poor respiratory effort
- Silent chest (inaudible wheeze)
- Altered consciousness

DIFFERENTIAL DIAGNOSIS

Airflow obstruction = FEV₁ / FVC $<$ 0.7

AIRFLOW OBSTRUCTION

- COPD
- Bronchiectasis
- Inhaled foreign body
- Obliterated bronchiolitis
- Large airway stenosis
- Lung cancer
- Sarcoidosis

NO AIRFLOW OBSTRUCTION

- Chronic cough syndrome
- Hyperventilation syndrome
- Vocal cord dysfunction Rhinitis
- Gastro-oesophageal reflux
- Heart failure
- Pulmonary fibrosis

ASTHMA MANAGEMENT IN PRACTICE

The aim of asthma management is to prevent exacerbations and asthma deaths, and to relieve and control symptoms

treatment to:

- Reduce symptoms and impact on lifestyle (e.g., absence from work/school)
- Minimize the need for reliever medication
- Prevent severe attacks/exacerbations

MANAGEMENT OF ACUTE ASTHMA

NON-PHARMACEUTICAL MEASURE

In addition to medicals, other therapies and strategies may be considered where relevant, to assist in symptom control and risk reduction. Some examples with consistent high-quality evidence are:

- **Smoking cessation advice:** at every visit, strongly encourage smokers to quit. Provide access to counselling and resources. Advise parents and carers to exclude smoking in rooms/cars used by children with asthma
- **Physical Activity:** encourage people with asthma to engage in regular physical activity because of its general health benefits; it may have a small management of exercise-induced bronchoconstriction
- **Investigation for occupational asthma:** ask all patients with adult-onset asthma about their work history. Identify and remove occupational sensitizers as soon as possible. Refer patients for expert advice, if available
- **Identify aspirin-exacerbated respiratory disease,** and before prescribing NSAIDs including aspirin, always ask about previous reaction

Although allergens may contribute to asthma symptoms in sensitized patients, allergen avoidance is not recommended as a general strategy for asthma. These strategies are often complex and expensive, and there are no validated methods for identifying those who are likely to benefit.

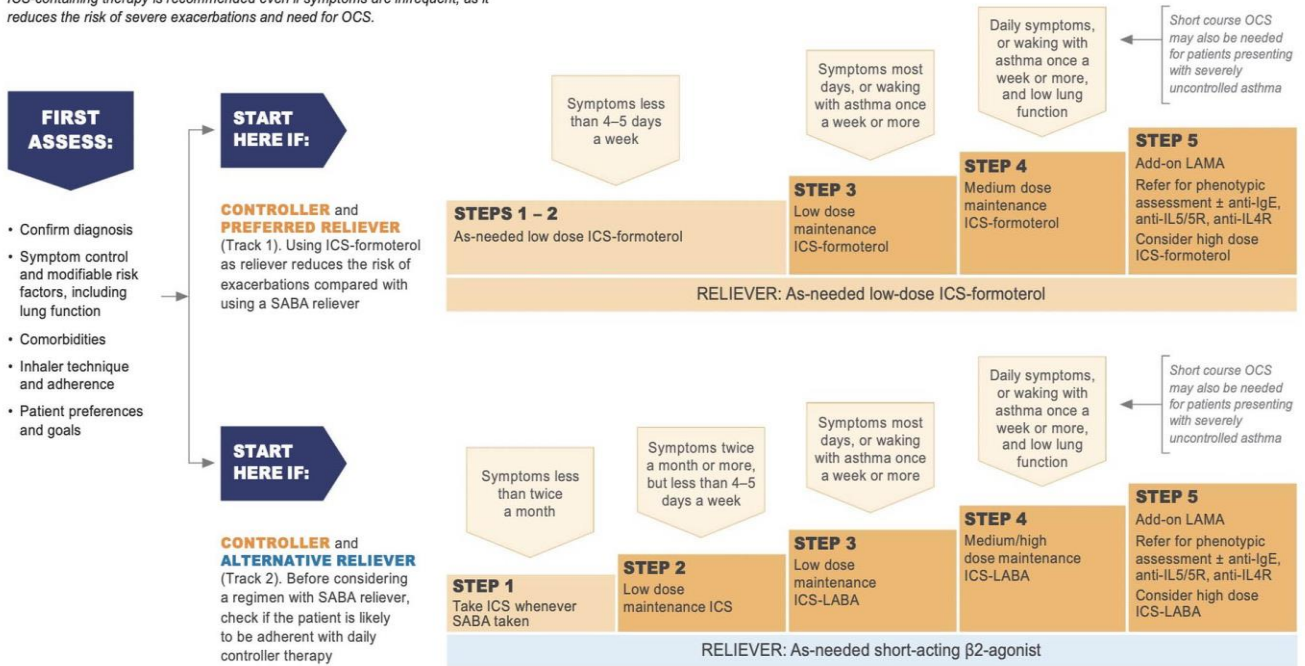
DRUG TREATMENT OF ASTHMA

- Use a stepwise approach
- Start at the step most appropriate to the initial severity of symptoms. The aim is to achieve early control of the condition.

STARTING TREATMENT

in adults and adolescents with a diagnosis of asthma

Track 1 is preferred if the patient is likely to be poorly adherent with daily controller ICS-containing therapy is recommended even if symptoms are infrequent, as it reduces the risk of severe exacerbations and need for OCS.

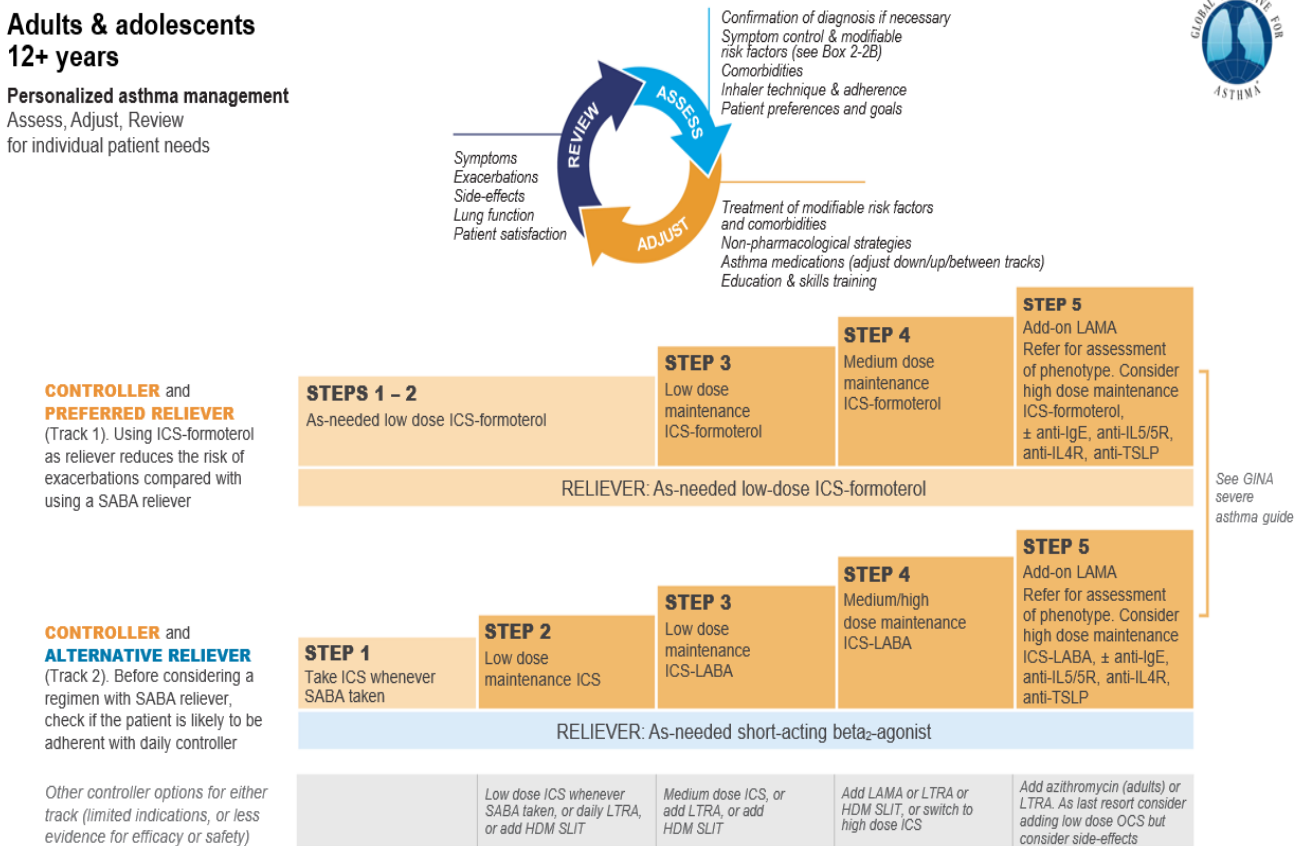


GINA 2021, Box 3-4Bi

© Global Initiative for Asthma, www.ginasthma.org

Adults & adolescents 12+ years

Personalized asthma management
Assess, Adjust, Review
for individual patient needs



GINA 2022, Box 3-5A

© Global Initiative for Asthma, www.ginasthma.org

Ref: <https://twitter.com/bigcatdoc/status/1389823874385199104/photo/1>

ASTHMA TREATMENT TRACKS FOR ADULTS AND ADOLESCENTS

The option for ongoing treatment for adults and adolescents have been obtained in the main treatment figure (Box) by showing two treatment tracks. The key difference between the tracks is the medication that is used for symptom relief: as-needed low dose ICS-formoterol in Track 1 (preferred) and as-needed SABA in Track 2

TRACK 1: THE RELIEVER IS AS-NEEDED LOW DOSE ICS-FORMOTEROL

This is the preferred approach recommended by GINA for adults and adolescents. Using low dose ICS-formoterol as reliever reduces the risk of severe exacerbations compared with regimens with SABA as reliever, with similar symptom control. With the approach:

- When as patient at any treatment step has asthma symptoms, they use low dose ICS-formoterol in a single inhaler for symptom relief.
- In steps 3-5, patients also take ICS-formoterol as their regular daily treatment. This is called ‘maintenance and reliever therapy (MART)

ICS-formoterol should not be used as the reliever by patients taking any other ICS-LABA

TRACK 2: THE RELIEVER IS AS-NEEDED SABA.

This is an alternative approach when Track 1 is not possible or is not preferred by a patient who has no exacerbations on their current therapy.

- **In step 1**, the patient takes a SABA and a low dose ICS together for symptom relief when a symptom occurs, either in combination inhaler or with the ICS taken right after the SABA
- **In Steps 2-5**, a SABA (alone) is used for symptom relief, and the patient takes ICS-containing controller medication regularly every day.

Before prescribing a regimen with SABA reliever, consider whether the patient is likely to be adherent with their ICS-containing controller therapy, as otherwise they will be at higher risk of exacerbations.

During ongoing treatment, treatment can be stepped up or down along one track, using the same reliever at each step, or it can be switched between tracks, according to the individual patient’s needs.

Before stepping up, check for common problems such as incorrect inhaler technique, poor adherence, and environmental exposures, and confirm that the symptoms are due to asthma (See Box 5) be scheduled. The frequency of review depends on the patient’s initial level of symptom control, their risk factors, their response to initial treatment, and their ability and willingness to engage in self-management with an action plan.

STEPPING UP ASTHMA TREATMENT

Asthma is a variable condition, and periodic adjustment of controller treatment by the clinician and/or patient may be needed.

Sustained step-up (for at least 2-3 months), if symptoms and/or exacerbations persist despite 2-3 months of controller treatment, assess the following common issue before considering a step-up

- Incorrect inhaler technique
- Poor adherence
- Modifiable risk factors, e.g., smoking
- Are symptoms due to comorbid conditions, e.g., allergic rhinitis

Short-term step-up (for 1-2 weeks) by clinician or by patient with written asthma action plan, e.g., during viral infection or allergen exposure

Day-today adjustment by patient with as-needed low dose ICS-formoterol for mild asthma, or ICS-formoterol as maintenance and reliever therapy. This is particularly effective in reducing severe exacerbations.

STEPPING DOWN TREATMENT WHEN ASTHMA IS WELL-CONTROLLED

Consider stepping down treatment once good asthma control has been achieved and maintained for 3 months, to find the lowest treatment that controls both symptoms and exacerbations, and minimizes side-effects.

- Choose an appropriate time for step-down (no respiratory infection, patient not travelling, not pregnant)
- Assess risk factors, including history of previous exacerbations or emergency department visit, and low lung function
- Document baseline status (symptom control and lung function), provide a written asthma action plan, monitor closely, and book a follow-up visit
- Step down through available formulations to reduce the ICS dose by 25-50% at 2-3-month intervals (see box 3-9 in full GINA 2021 report for details of how to step down different controller treatments)
- If asthma is well-controlled on low dose ICS or LTRA, as-needed low dose ICS-formoterol is a step-down option, based on three large studies in mild asthma. Smaller studies have shown that low dose ICS taken whenever SABA is taken (with combination or separate inhalers) is more effective as a step-down strategy than SABA alone.
- Do not completely stop ICS in adults or adolescents with asthma unless this is needed temporarily to confirm the diagnosis of asthma.
- Make sure a follow-up appointment is arranged.

BACKGROUND – THE RISKS OF “MILD” ASTHMA

- Patients with apparently mild asthma are still at risk of serious adverse events
 - 30-37% of adults with acute asthma
 - 16% of patients with near-fatal asthma
 - 15-27% of adults dying of asthmahad symptoms less than weekly in previous 3 months (Dusser Allergy 2007, Bergstrom 2008)
- Exacerbation triggers are unpredictable (viruses, pollens, pollution, poor adherence)
- Even 4-5 lifetime OCS courses increase the risk of osteoporosis, diabetes, cataract (*Price et al J Asthma Allerg 2018*)

(Global Initiative for Asthma www.ginasthma.org)

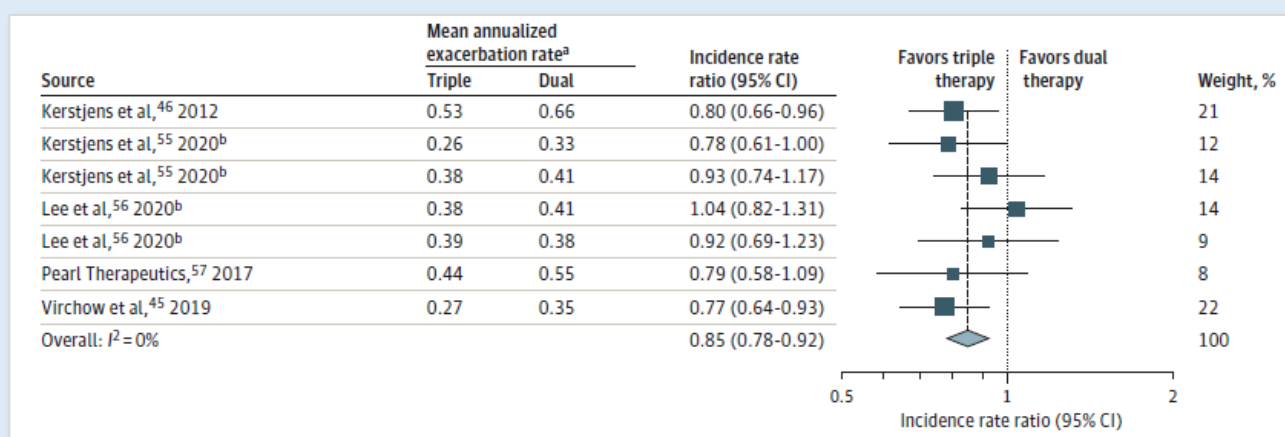
WHY NOT TREAT WITH SABA ALONE?

INHALED SABA HAS BEEN FIRST-LINE TREATMENT FOR ASTHMA FOR 50 YEARS

- Asthma was thought to be a disease of bronchoconstriction
- Role of SABA reinforced by rapid relief of symptoms and low cost
- Regular use of SABA, even for 1–2 weeks, is associated with increased AHR, reduced bronchodilator effect, increased allergic response, increased eosinophils (e.g., Hancox, 2000; Aldridge, 2000)
- Can lead to a vicious cycle encouraging overuse
- Over-use of SABA associated with ↑ exacerbations and ↑ mortality (e.g., Suissa 1994, Nwaru 2020)
- Starting treatment with SABA trains the patient to regard it as their primary asthma treatment
- The only previous option was daily ICS even when no symptoms, but adherence is extremely poor
- GINA changed its recommendation once evidence for a safe and effective alternative was available

OTHER CHANGES IN MEDICATION RECOMMENDATIONS FOR ≥12 YEARS

- Long-acting muscarinic antagonists (LAMA) should not be used as monotherapy for asthma (i.e. without ICS) because of increased risk of severe exacerbations (Baan, *Pulm Pharmacol Ther* 2021)
- Adding LAMA to ICS-LABA: GRADE review and meta-analysis (Kim, *JAMA* 2021) confirms previous findings
- Small increase in lung function (mean difference 0.08 L)
- No clinically important benefits for symptoms or quality of life → don't prescribe for dyspnea
- Modest overall reduction in exacerbations compared with ICS-LABA (risk ratio 0.83 [0.77, 0.90])



- Patients with exacerbations should receive at least medium dose ICS-LABA before considering add-on LAMA
- Chromone pMDIs (sodium cromoglycate, nedocromil sodium) have been discontinued globally

HOW TO INVESTIGATE UNCONTROLLED ASTHMA

Most patients can achieve good asthma control with ICS-containing treatment, but some patients do not, and further investigation is needed.

Box 5. How to investigate uncontrolled asthma in primary care

Watch patient using their inhaler. Discuss adherence and barriers to use	Compare inhaler technique with a device-specific checklist, and correct errors, recheck frequently. Have an empathic discussion about barriers to adherence.
↓	
Confirm the diagnosis of asthma	If lung function normal during symptoms, consider halving ICS dose and repeating lung function after 2-3 weeks
↓	
Remove potential risk factors. Assess and manage comorbidities	Check for risk factors or inducers such as smoking, beta0blockers, NSAIDs, allergen exposure. Check for comorbidities such as rhinitis, obesity, GERD, depression/anxiety.
↓	
Consider treatment step-up	Consider step-up to next treatment level. Use shared decision-making, and balance potential benefits and risks.
↓	
Refer to a specialist or severe asthma clinic	If asthma still uncontrolled after 3-6 months on Step 4 treatment, refer for expert advice. Refer earlier if asthma symptoms severe or doubts about diagnosis.

REVIEW AND MONITORING

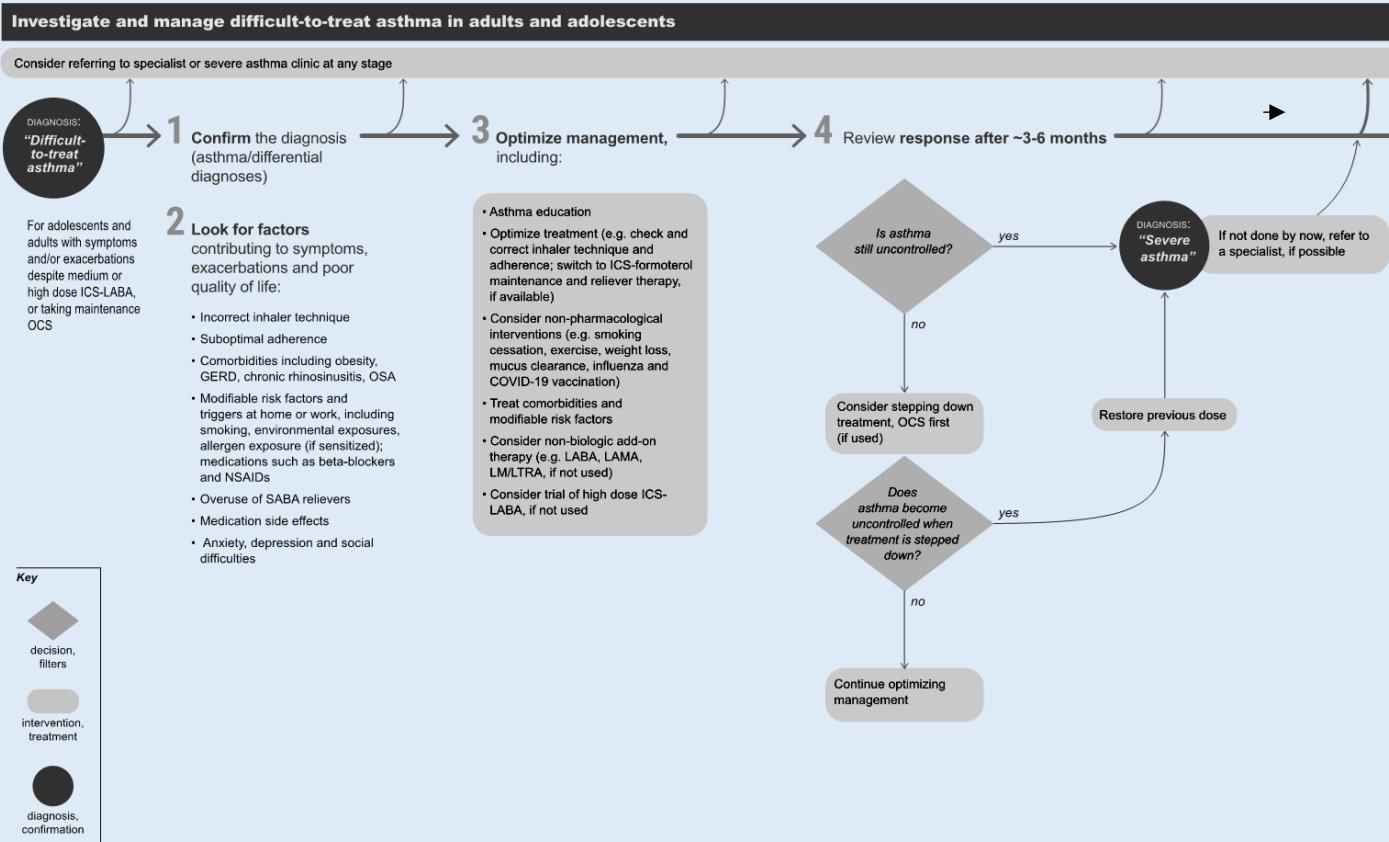
Aim to review all patients with asthma at least annually

- Check symptoms last seen. Use objective measures
In the last month
 1. Have you had any **difficulty sleeping** because of your asthma symptoms (including cough)?
 2. Have you had your usual asthma **symptoms during the day** (cough, wheeze, chest tightness, or breathlessness)?
 3. Has your asthma **interfered with usual activities**, e.g., house work, work/school etc.?
- Record smoking status and advice smokers to stop.
- Record any **exacerbations/acute attacks** since last seen
- Check **medication-inhaler** technique, problems, side effects.
- Check influenza/pneumococcal vaccination received.
- Review objective measures of lung function, e.g., home PEFr chart, PEFr/ spirometry at review.
- Address any problems or queries and educate about asthma.
- Agree management goals and date for further Review

Table 1. Levels of asthma control

Characteristics	Controlled (all of the following)	Partly controlled (any present in any week)	Uncontrolled
Daytime symptoms	≤ 2 per week	>2 per week	2-3 features of partly controlled asthma present in any week
Limitation of activities	None	Any	
Nocturnal symptoms/awakening	None	Any	

Need for rescue/reliever treatment	≤ 2 per week	>2 per week	
Lung function (PEF or FEV ₁)	Normal	$<80\%$ predicted or personal best (if known) on any day	



Ref: GINA guideline 2022

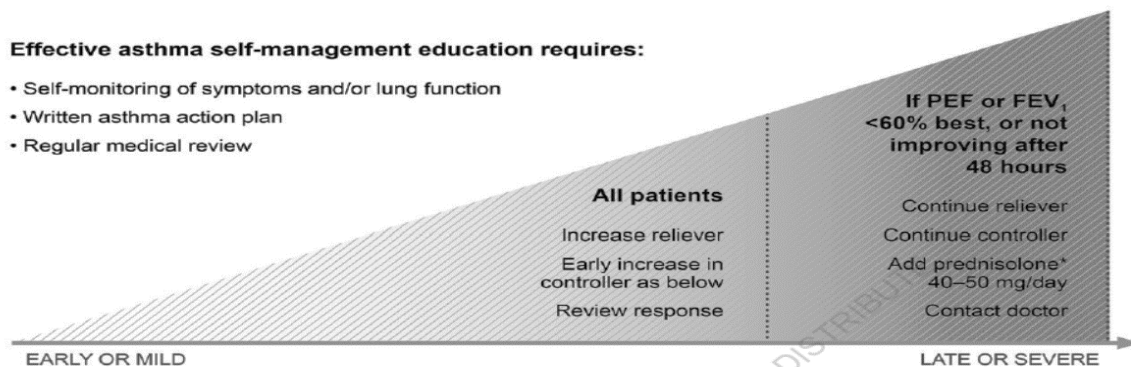
WRITTEN ASTHMA ACTION PLANS

All patients should be provided with a written asthma action plan appropriate for their level of asthma control and health literacy, so they know how to recognize and respond to worsening asthma.

Box 10. Self-management with a written action plan

Effective asthma self-management education requires:

- Self-monitoring of symptoms and/or lung function
- Written asthma action plan
- Regular medical review



The written asthma action plan should include:

- the patient's usual asthma medications
- when and how to increase medications, and start OCS if needed
- how to access medical care if symptoms fail to respond.

COVID-19 AND ASTHMA

- **Are people with asthma at increased risk of COVID-19, or severe COVID-19?**
 - People with asthma do not appear to be at increased risk of acquiring COVID-19, and systematic reviews have not shown an increased risk of severe COVID-19 in people with well-controlled, mild-to-moderate asthma
- **Are people with asthma at increased risk of COVID-19-related death?**
 - Overall, studies to date indicate that people with well-controlled asthma are not at increased risk of COVID-19-related death (*Williamson, Nature 2020; Liu et al JACI IP 2021*) and in one meta-analysis, mortality appeared to be lower than in people without asthma (*Hou, JACI IP 2021*).
 - However, the risk of COVID-19 death was increased in people who had recently needed OCS for their asthma (*Williamson, Nature 2020; Shi, Lancet RM 2022*) and in hospitalized patients with severe asthma (*Bloom, Lancet RM 2021*).
- **What are the implications for asthma management?**
 - It is important to continue good asthma management (as described in the GINA report), with strategies to maintain good symptom control, reduce the risk of severe exacerbations and minimise the need for OCS
- **Have there been more asthma exacerbations during the pandemic?**
 - No: in 2020–21, many countries saw a *decrease* in asthma exacerbations and influenza-related illness
 - The reasons are not precisely known, but may be due to public health measures such as handwashing, masks and social/physical distancing that reduced the incidence of other respiratory infections, including influenza (*Davies, Thorax 2021*)

COVID-19 AND ASTHMA MEDICATION

- Advise patients to continue taking their prescribed asthma medications, particularly inhaled corticosteroids
 - For patients with severe asthma, continue biologic therapy or OCS if prescribed
- Are inhaled corticosteroids (ICS) protective in COVID-19?
 - In one study of hospitalized patients aged ≥ 50 years with COVID-19, ICS use in those with asthma was associated with lower mortality than in patients without an underlying respiratory condition (*Bloom, Lancet RM 2021*)
- Make sure that all patients have a written asthma action plan, advising them to:
 - Increase controller and reliever medication when asthma worsens (see GINA report Box 4-2)
 - Take a short course of OCS when appropriate for severe asthma exacerbations
- When COVID-19 is confirmed or suspected, or local risk is moderate or high, avoid nebulizers where possible, to reduce the risk of spreading virus to health professionals and other patients/family
 - For bronchodilator administration, pressurized metered dose inhaler via a spacer is preferred except for acute severe asthma
 - Add a mouthpiece or mask to the spacer if required

COVID-19 AND ASTHMA – INFECTION CONTROL

- In healthcare facilities, follow local COVID-19 testing recommendations and infection control procedures if spirometry or peak flow measurement is needed (*e.g., Virant, JACI in Practice 2022*)
 - Use of an in-line filter minimizes the risk of transmission *during* spirometry, but many patients cough *after* performing spirometry; coach the patient to stay on the mouthpiece if they feel the need to cough
 - If spirometry is not available due to local infection control restrictions, and information about

- lung function is needed, consider asking patients to monitor lung function at home
- Follow local infection control procedures if other aerosol-generating procedures are needed
 - Nebulization, oxygen therapy (including nasal prongs), sputum induction, manual ventilation, non-invasive ventilation and intubation
- Follow local health advice about hygiene strategies and use of personal protective equipment, as new information becomes available in your country or region

COVID-19 VACCINE AND ASTHMA

- Have COVID-19 vaccines been studied in people with asthma?
 - Yes. Many types of COVID-19 vaccines have been studied and are being used worldwide
- Are COVID-19 vaccines safe in people with allergies?
 - In general, allergic reactions to vaccines are rare
 - Patients with a history of severe allergic reaction to a COVID-19 vaccine ingredient (e.g., polyethylene glycol for Pfizer/BioNTech or Moderna, or polysorbate 80 for AstraZeneca or J&J/Janssen), should receive a different COVID-19 vaccine. More details from ACIP in <https://www.cdc.gov/vaccines/hcp/acip-recs/vacc-specific/covid-19.html>
 - People with allergies to food, insect venom or other medications can safely receive COVID-19 vaccines
 - As always, patients should speak to their healthcare provider if they have concerns
 - Follow local advice about monitoring patients after COVID-19 vaccination
- Usual vaccine precautions apply, for example:
 - Ask if the patient has a history of allergy to any components of the vaccine
 - If the patient has a fever or another infection, delay vaccination until they are well
- Based on the risks and benefits, and with the above precautions, GINA recommends people with asthma should be up to date with COVID-19 vaccination (including booster doses, if available)
- COVID-19 vaccination and biologic therapy
 - We suggest that the first dose of asthma biologic therapy and COVID-19 vaccine should not be given on the same day, so that adverse effects of either can be more easily distinguished
- Influenza vaccination
 - Remind people with asthma to have an annual influenza vaccination
 - CDC now recommends that influenza vaccine and COVID-19 vaccine can be given on the same day
- After COVID-19 vaccination
 - Current advice from the United States Centres for Disease Control and Prevention (CDC) is that where there is substantial transmission of COVID-19, people will be better protected, even if they are fully vaccinated, if they wear a mask in indoor public settings; this will also reduce risk to others.

GINA will update advice about COVID-19 and asthma as new data become available

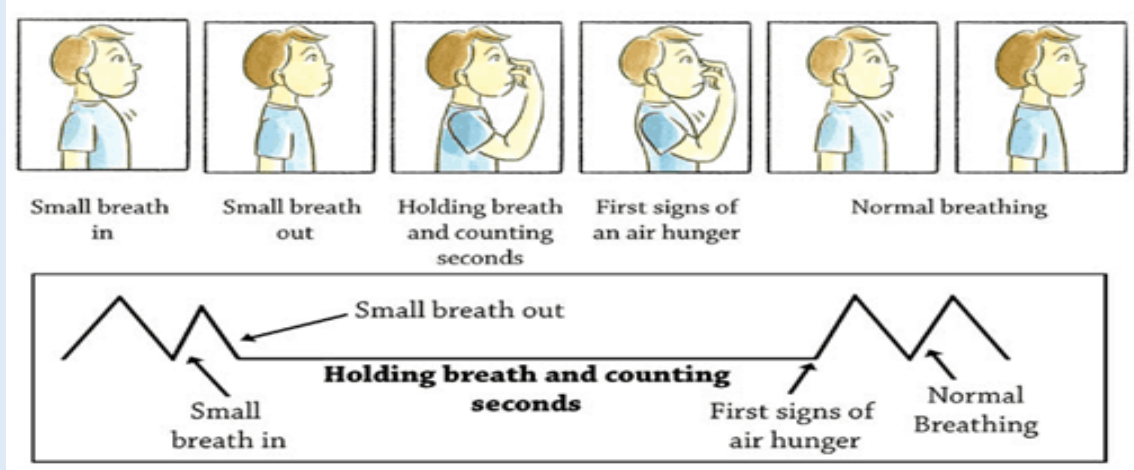
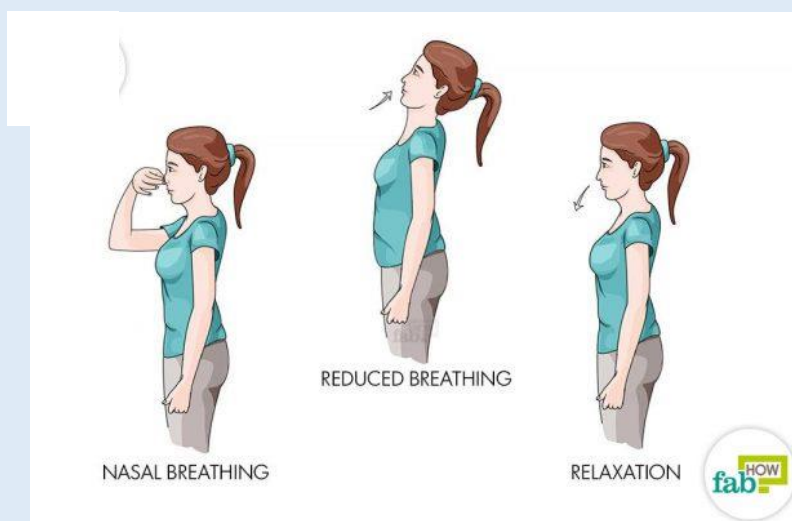


Fig 2. Holding Breathing

https://buteykoclinic.com/wp-content/uploads/2016107/holding_breath.png



<https://www.fabhow.com/wp-content/uploads/2017/03/buteyko-breathing-for-relief-from-asthma-1.jpg>

DIFFICULT ASTHMA

Persistent symptoms and/or frequent exacerbations despite treatment at step 4/5. Check diagnosis and exacerbating factors.

Assess adherence to medication. Find out about family, psychological, or social problems that may be interfering with effective management.

REFERENCE:

1. *GINA Guideline 2022*
2. *Oxford handbook of General Practice 4th edition*

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

DEFINITION

Chronic obstructive pulmonary disease (COPD) is a slowly progressive disorder characterized by airflow obstruction (**FEV1<80% predicted; FEV1/FVC<0.7**) with little or no reversibility. It includes chronic bronchitis and emphysema.

Aetiology, Pathobiology and Pathology of COPD leading to Airflow Limitation and Clinical Manifestation



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PATHWAY TO DIAGNOSIS OF COPD

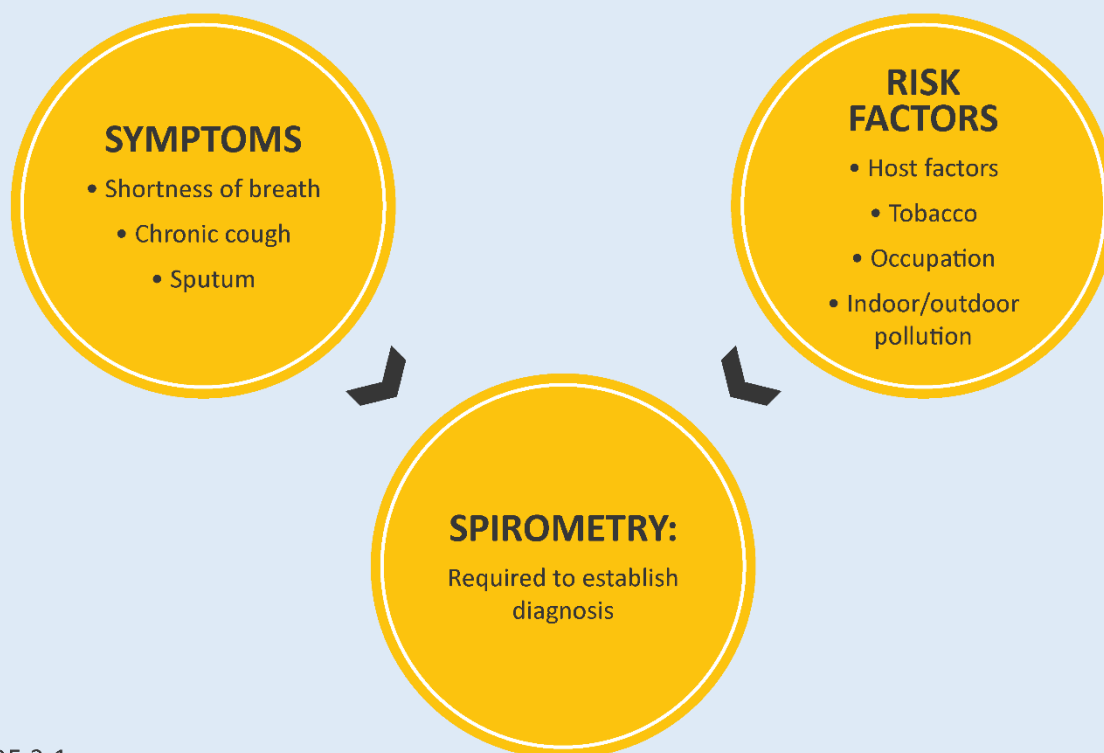


FIGURE 2.1

© 2022 Global Initiative for Chronic Obstructive Lung Disease

▶ KEY INDICATORS FOR CONSIDERING A DIAGNOSIS OF COPD

Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD. Spirometry is required to establish a diagnosis of COPD.

Dyspnea that is:	Progressive over time. Characteristically worse with exercise. Persistent.
Chronic Cough:	May be intermittent and may be unproductive. Recurrent wheeze.
Chronic Sputum Production:	Any pattern of chronic sputum production may indicate COPD.
Recurrent Lower Respiratory Tract Infections	
History of Risk Factors:	Host factors (such as genetic factors, congenital/developmental abnormalities etc.). Tobacco smoke (including popular local preparations). Smoke from home cooking and heating fuels. Occupational dusts, vapors, fumes, gases and other chemicals.
Family History of COPD and/or Childhood Factors:	For example low birthweight, childhood respiratory infections etc.

TABLE 2.1

▶ OTHER CAUSES OF CHRONIC COUGH

INTRATHORACIC

- Asthma
- Lung Cancer
- Tuberculosis
- Bronchiectasis
- Left Heart Failure
- Interstitial Lung Disease
- Cystic Fibrosis
- Idiopathic Cough

EXTRATHORACIC

- Chronic Allergic Rhinitis
- Post Nasal Drip Syndrome (PNDS)
- Upper Airway Cough Syndrome (UACS)
- Gastroesophageal Reflux
- Medication (e.g. ACE Inhibitors)

TABLE 2.2

▶ CONSIDERATIONS IN PERFORMING SPIROMETRY

PREPARATION

- Spirometers need calibration on a regular basis.
- Spirometers should produce hard copy or have a digital display of the expiratory curve to permit detection of technical errors or have an automatic prompt to identify an unsatisfactory test and the reason for it.
- The supervisor of the test needs training in optimal technique and quality performance.
- Maximal patient effort in performing the test is required to avoid underestimation of values and hence errors in diagnosis and management.

BRONCHODILATION

- Possible dosage protocols are 400 mcg short-acting beta₂-agonist, 160 mcg short-acting anticholinergic, or the two combined.^a FEV₁ should be measured 10-15 minutes after a short-acting beta₂-agonist is given, or 30-45 minutes after a short-acting anticholinergic or a combination of both classes of drugs.

PERFORMANCE

- Spirometry should be performed using techniques that meet published standards.^b
- The expiratory volume/time traces should be smooth and free from irregularities. The pause between inspiration and expiration should be < 1 second.
- The recording should go on long enough for a volume plateau to be reached, which may take more than 15 seconds in severe disease.
- Both FVC and FEV₁ should be the largest value obtained from any of three technically satisfactory curves and the FVC and FEV₁ values in these three curves should vary by no more than 5% or 150 ml, whichever is greater.
- The FEV₁/FVC ratio should be taken from the technically acceptable curve with the largest sum of FVC and FEV₁.

EVALUATION

- Spirometry measurements are evaluated by comparison of the results with appropriate reference values based on age, height, sex, and race.
- The presence of a postbronchodilator FEV₁/FVC < 0.70 confirms the presence of airflow limitation.

a Pellegrino et al. Eur Respir J 2005; 26(5): 948-68;

b Miller et al. Eur Respir J 2005; 26(2): 319-38.

The GOLD guidelines classify patients into four different categories: GOLD 1 (mild), GOLD 2

(moderate), GOLD 3 (severe), or GOLD 4 (very severe) based on their level of airflow limitation. This is assessed by evaluating

CLASSIFICATION OF AIRFLOW LIMITATION SEVERITY IN COPD (BASED ON POST-BRONCHODILATOR FEV ₁)		
In patients with FEV ₁ /FVC < 0.70:		
GOLD 1:	Mild	FEV ₁ ≥ 80% predicted
GOLD 2:	Moderate	50% ≤ FEV ₁ < 80% predicted
GOLD 3:	Severe	30% ≤ FEV ₁ < 50% predicted
GOLD 4:	Very Severe	FEV ₁ < 30% predicted

MODIFIED MRC DYSPNEA SCALE ^a		
PLEASE TICK IN THE BOX THAT APPLIES TO YOU ONE BOX ONLY Grades 0 - 4		
mMRC Grade 0.	I only get breathless with strenuous exercise.	<input type="checkbox"/>
mMRC Grade 1.	I get short of breath when hurrying on the level or walking up a slight hill.	<input type="checkbox"/>
mMRC Grade 2.	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level.	<input type="checkbox"/>
mMRC Grade 3.	I stop for breath after walking about 100 meters or after a few minutes on the level.	<input type="checkbox"/>
mMRC Grade 4.	I am too breathless to leave the house or I am breathless when dressing or undressing.	<input type="checkbox"/>
^a Fletcher CM. BMJ 1960; 2: 1662. TABLE 2.5		

CAT™ ASSESSMENT

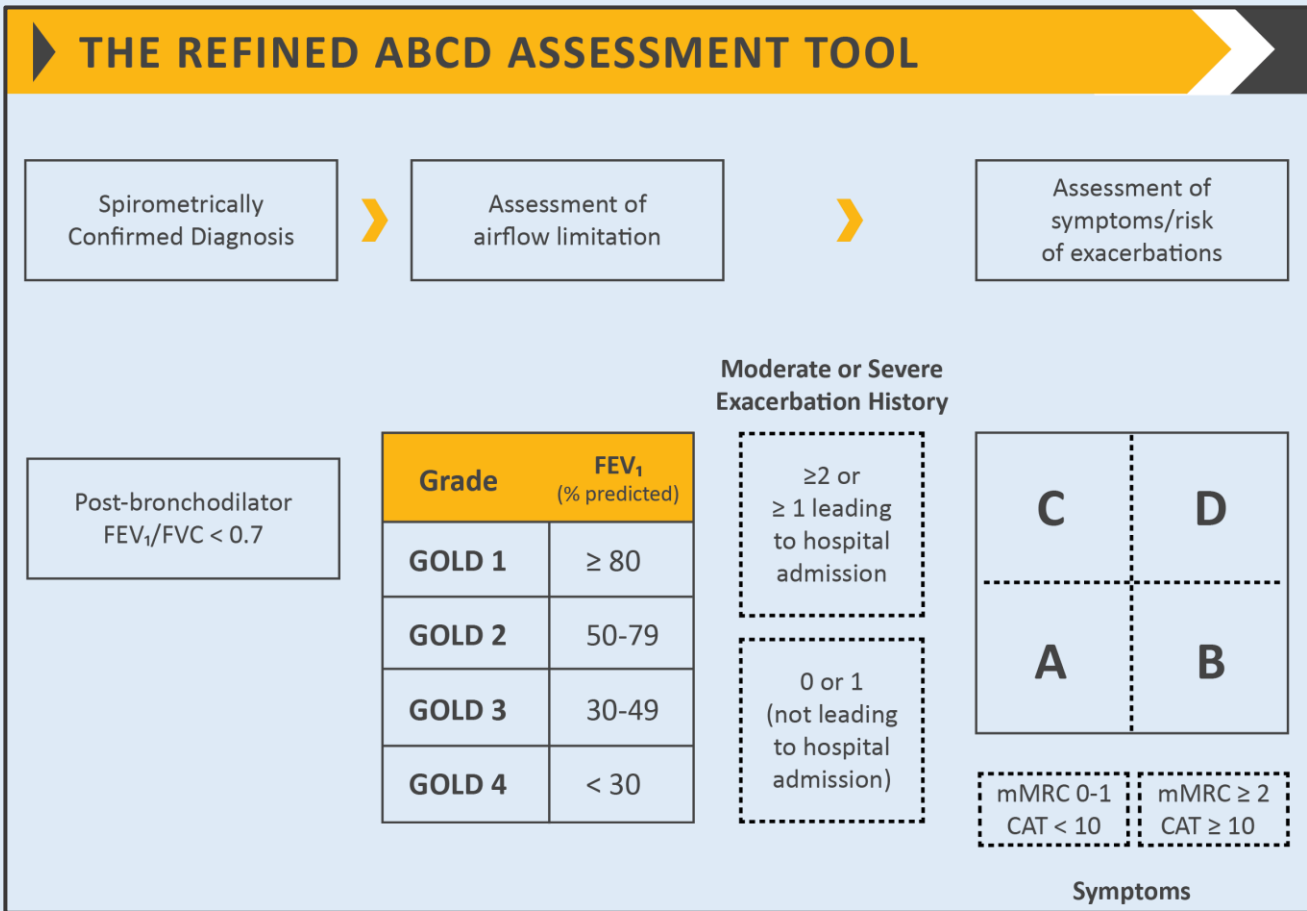
For each item below, place a mark (x) in the box that best describes you currently.
Be sure to only select one response for each question.

EXAMPLE: I am very happy	0	<input checked="" type="radio"/>	2	3	4	5	I am very sad	SCORE
I never cough	0	1	2	3	4	5	I cough all the time	
I have no phlegm (mucus) in my chest at all	0	1	2	3	4	5	My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all	0	1	2	3	4	5	My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless	0	1	2	3	4	5	When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home	0	1	2	3	4	5	I am very limited doing activities at home	
I am confident leaving my home despite my lung condition	0	1	2	3	4	5	I am not at all confident leaving my home because of my lung condition	
I sleep soundly	0	1	2	3	4	5	I don't sleep soundly because of my lung condition	
I have lots of energy	0	1	2	3	4	5	I have no energy at all	
Reference: Jones et al. ERJ 2009; 34 (3); 648-54.								TOTAL SCORE: <input type="text"/>
FIGURE 2.3								

What is CAT score in COPD?

Table

CAT score	Impact level
<10	Low
10-20	Median
20-30	High
>30	Very High



Spirometry as the gold standard for accurate and repeatable measurement of lung function. Evidence is emerging that when spirometry confirms a COPD diagnosis, doctors initiate more appropriate treatment.

Spirometry is the cornerstone of COPD diagnosis. According to GOLD guidelines, persistent airflow limitation is defined as a **post-bronchodilator ratio of FEV1 to Forced Vital Capacity (FEV1/FVC) of less than 0.7**

▶ ROLE OF SPIROMETRY

- **Diagnosis**
- **Assessment of severity of airflow obstruction (for prognosis)**
- **Follow-up assessment**
 - » Therapeutic decisions.
 - Pharmacological in selected circumstances (e.g., discrepancy between spirometry and level of symptoms).
 - Consider alternative diagnoses when symptoms are disproportionate to degree of airflow obstruction.
 - Non-pharmacological (e.g., interventional procedures).
 - » Identification of rapid decline.

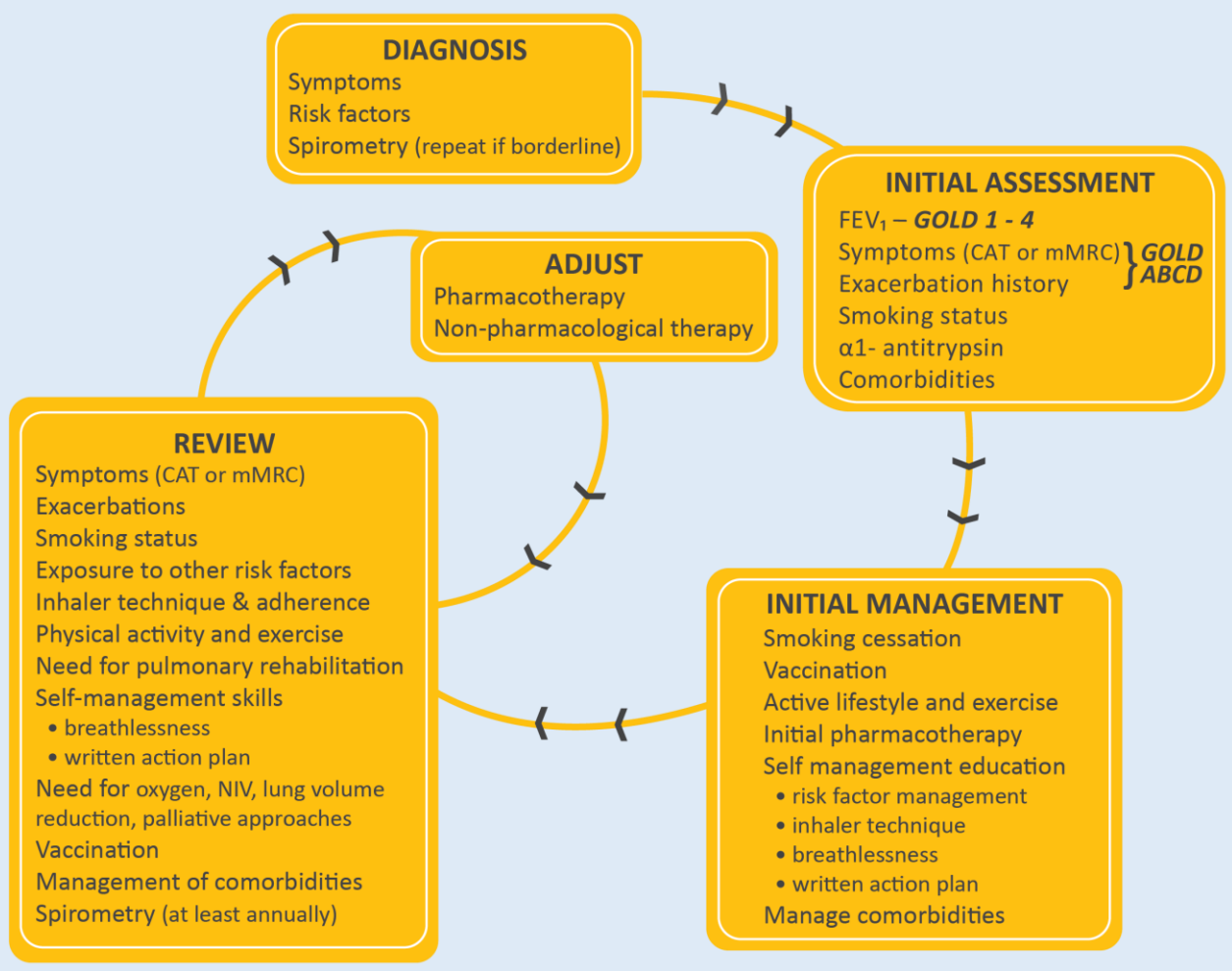
TABLE 2.6

▶ DIFFERENTIAL DIAGNOSIS OF COPD

DIAGNOSIS	SUGGESTIVE FEATURES
COPD	Onset in mid-life. Symptoms slowly progressive. History of tobacco smoking or exposure to other types of smoke.
Asthma	Onset early in life (often childhood). Symptoms vary widely from day to day. Symptoms worse at night/early morning. Allergy, rhinitis, and/or eczema also present. Family history of asthma. Obesity coexistence.
Congestive Heart Failure	Chest X-ray shows dilated heart, pulmonary edema. Pulmonary function tests indicate volume restriction, not airflow limitation.
Bronchiectasis	Large volumes of purulent sputum. Commonly associated with bacterial infection. Chest X-ray/CT shows bronchial dilation, bronchial wall thickening.
Tuberculosis	Onset all ages. Chest X-ray shows lung infiltrate. Microbiological confirmation. High local prevalence of tuberculosis.
Obliterative Bronchiolitis	Onset at younger age, nonsmokers. May have history of rheumatoid arthritis or acute fume exposure. Seen after lung or bone marrow transplantation. CT on expiration shows hypodense areas.
Diffuse Panbronchiolitis	Predominantly seen in patients of Asian descent. Most patients are male and nonsmokers. Almost all have chronic sinusitis. Chest X-ray & HRCT show diffuse small centrilobular nodular opacities & hyperinflation.

These features tend to be characteristic of the respective diseases, but are not mandatory. For example, a person who has never smoked may develop COPD (especially in the developing world where other risk factors may be more important than cigarette smoking); asthma may develop in adult and even in elderly patients.

MANAGEMENT OF COPD



MANAGEMENT CYCLE

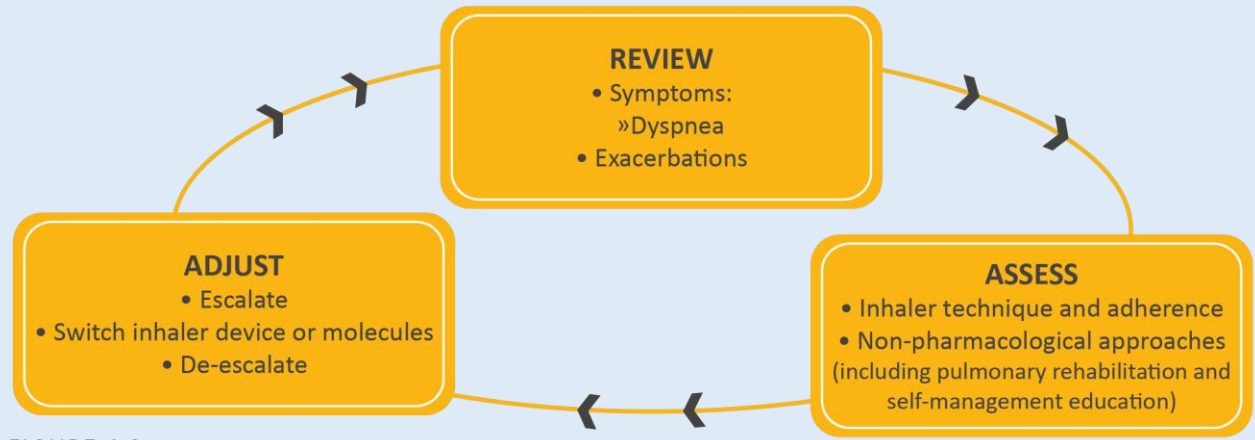


FIGURE 4.3

INITIAL PHARMACOLOGICAL TREATMENT

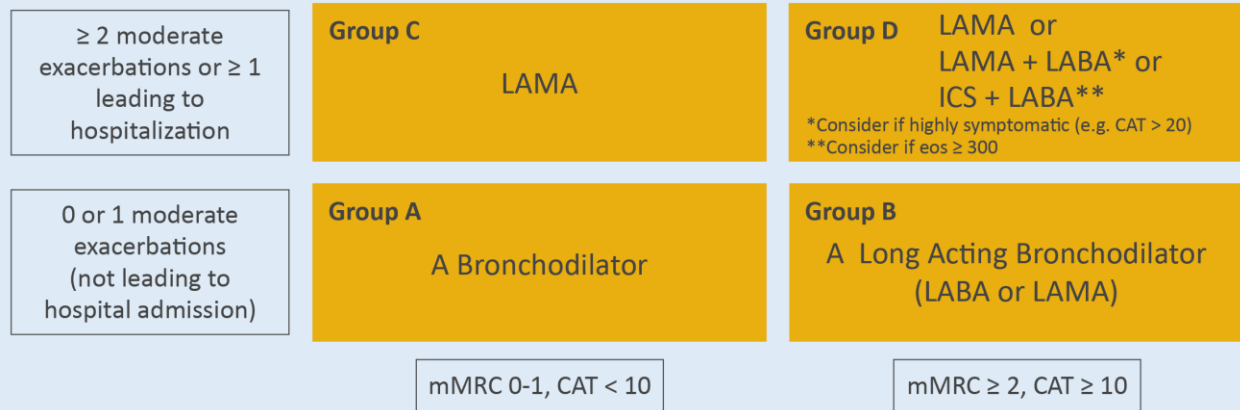
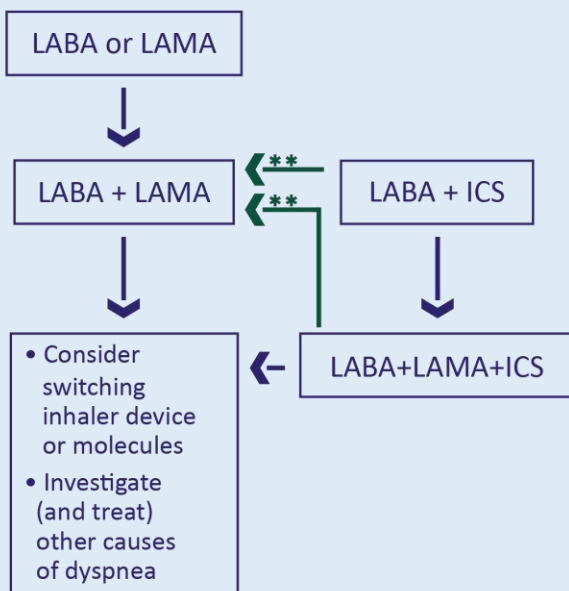


FIGURE 4.2

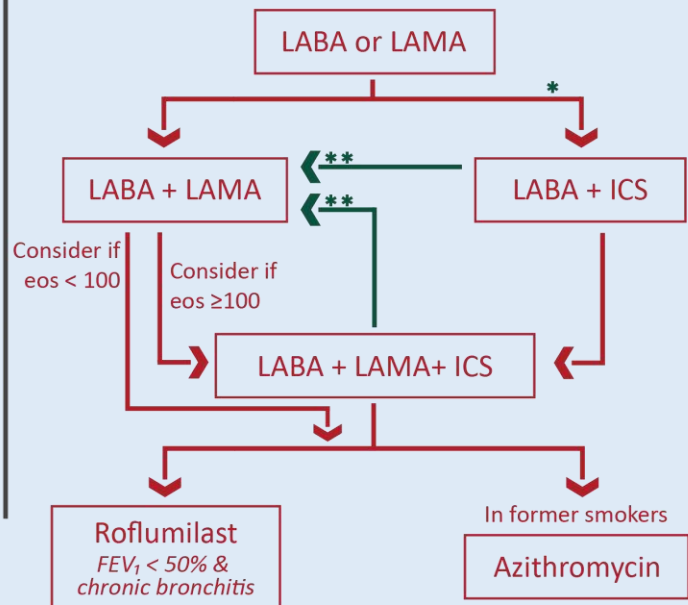
FOLLOW-UP PHARMACOLOGICAL TREATMENT

- IF RESPONSE TO INITIAL TREATMENT IS APPROPRIATE, MAINTAIN IT.
- IF NOT:
 - ✓ Consider the predominant treatable trait to target (dyspnea or exacerbations)
 - Use exacerbation pathway if both exacerbations and dyspnea need to be targeted
 - ✓ Place patient in box corresponding to current treatment & follow indications
 - ✓ Assess response, adjust and review
 - ✓ These recommendations do not depend on the ABCD assessment at diagnosis

• DYSPNEA •



• EXACERBATIONS •



eos = blood eosinophil count (cells/ μ L)

* Consider if eos ≥ 300 or eos ≥ 100 AND ≥ 2 moderate exacerbations / 1 hospitalization

** Consider de-escalation of ICS or switch if pneumonia, inappropriate original indication or lack of response to ICS

FIGURE 4.4

▶ NON-PHARMACOLOGIC MANAGEMENT OF COPD*

PATIENT GROUP	ESSENTIAL	RECOMMENDED	DEPENDING ON LOCAL GUIDELINES
A	Smoking Cessation (can include pharmacologic treatment)	Physical Activity	Flu Vaccination Pneumococcal Vaccination Pertussis Vaccination Covid-19 Vaccination
B, C and D	Smoking Cessation (can include pharmacologic treatment) Pulmonary Rehabilitation	Physical Activity	Flu Vaccination Pneumococcal Vaccination Pertussis Vaccination Covid-19 Vaccination

*Can include pharmacologic treatment.

TABLE 4.8

▶ FOLLOW-UP OF NON-PHARMACOLOGICAL TREATMENT

1. IF RESPONSE TO INITIAL TREATMENT IS APPROPRIATE, MAINTAIN IT AND OFFER:

- Flu vaccination every year and other recommended vaccinations according to guidelines
- Self-management education
- Assessment of behavioral risk factors such as smoking cessation (if applicable) and environmental exposures

Ensure

- Maintenance of exercise program and physical activity
- Adequate sleep and a healthy diet

2. IF NOT, CONSIDER THE PREDOMINANT TREATABLE TRAIT TO TARGET

• DYSPNEA •

- ▶ Self-management education (written action plan) with integrated self-management regarding:
 - Pulmonary rehabilitation (PR) program and/or maintenance exercise program post PR
 - Breathlessness and energy conservation techniques, and stress management strategies

• EXACERBATIONS •

- ▶ Self-management education (written action plan) that is personalized with respect to:
 - Avoidance of aggravating factors
 - How to monitor/manage worsening of symptoms
 - Contact information in the event of an exacerbation

All patients with advanced COPD should be considered for end of life and palliative care support to optimize symptom control and allow patients and their families to make informed choices about future management

OXYGEN THERAPY AND VENTILATORY SUPPORT IN STABLE COPD

OXYGEN THERAPY

- The long-term administration of oxygen increases survival in patients with severe chronic resting arterial hypoxemia (**Evidence A**).
- In patients with stable COPD and moderate resting or exercise-induced arterial desaturation, prescription of long-term oxygen does not lengthen time to death or first hospitalization or provide sustained benefit in health status, lung function and 6-minute walk distance (**Evidence A**).
- Resting oxygenation at sea level does not exclude the development of severe hypoxemia when traveling by air (**Evidence C**).

VENTILATORY SUPPORT

- NPPV may improve hospitalization-free survival in selected patients after recent hospitalization, particularly in those with pronounced daytime persistent hypercapnia ($\text{PaCO}_2 \geq 52$ mmHg) (**Evidence B**).

INTERVENTIONAL THERAPY IN STABLE COPD

LUNG VOLUME REDUCTION SURGERY

- Lung volume reduction surgery improves survival in severe emphysema patients with an upper-lobe emphysema and low post-rehabilitation exercise capacity (**Evidence A**).

BULLECTOMY

- In selected patients, bullectomy is associated with decreased dyspnea, improved lung function and exercise tolerance (**Evidence C**).

TRANSPLANTATION

- In appropriately selected patients with very severe COPD, lung transplantation has been shown to improve quality of life and functional capacity (**Evidence C**).

BRONCHOSCOPIC INTERVENTIONS

- In select patients with advanced emphysema, bronchoscopic interventions reduce end-expiratory lung volume and improve exercise tolerance, health status and lung function at 6-12 months following treatment. Endobronchial valves (**Evidence A**); Lung coils (**Evidence B**); Vapor ablation (**Evidence B**).

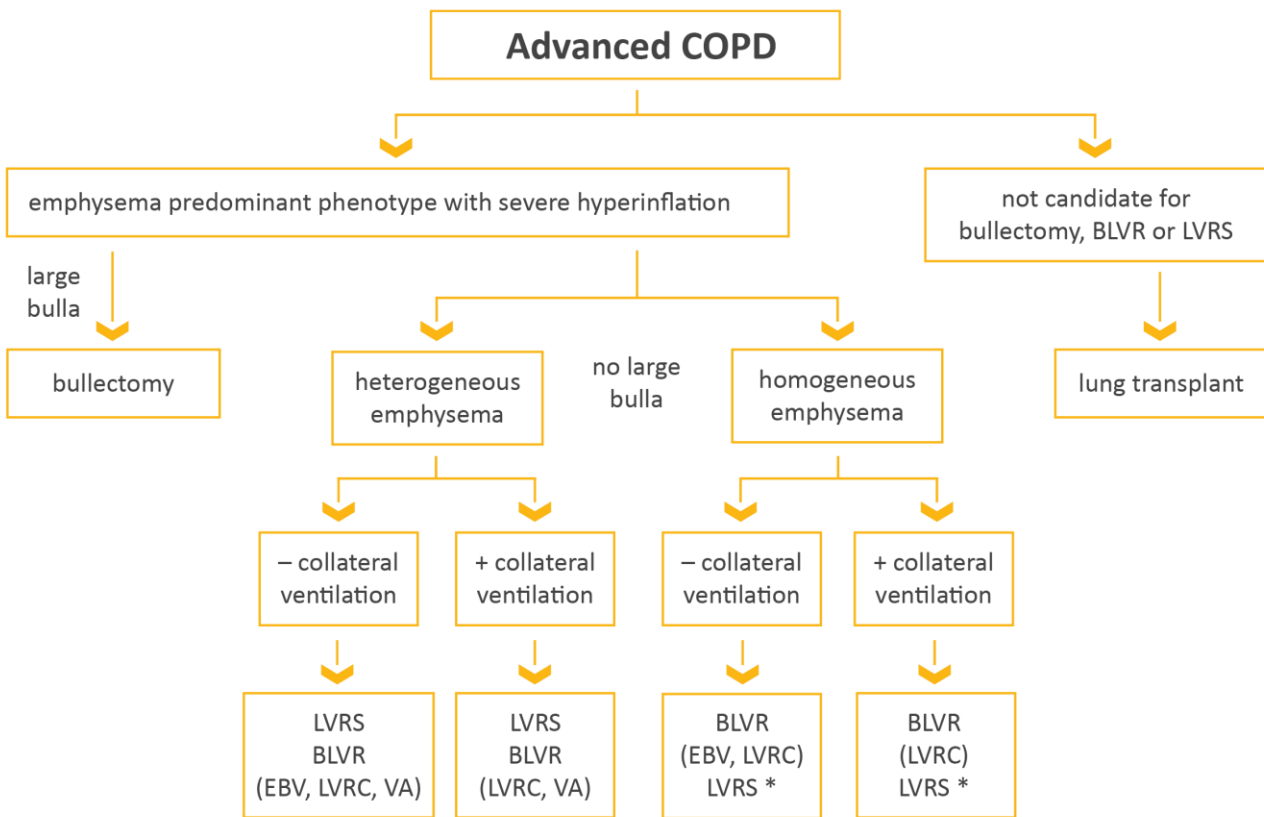
GOALS FOR TREATMENT OF STABLE COPD

- | | | |
|--|---|-----------------|
| <ul style="list-style-type: none"> • Relieve Symptoms • Improve Exercise Tolerance • Improve Health Status | ➤ | REDUCE SYMPTOMS |
| <i>and</i> | | |
| <ul style="list-style-type: none"> • Prevent Disease Progression • Prevent and Treat Exacerbations • Reduce Mortality | ➤ | REDUCE RISK |

TABLE 4.1

INTERVENTIONAL BRONCHOSCOPIC AND SURGICAL TREATMENTS FOR COPD

Overview of various therapies used to treat patients with COPD and emphysema worldwide. Note that all therapies are not approved for clinical care in all countries. Additionally, the effects of BLVR on survival or other long term outcomes or comparison to LVRS are unknown.



Definition of Abbreviations: BLVR, Bronchoscopic Lung Volume Reduction, EBV, endobronchial Valve, LVRS, Lung volume reduction surgery, LVRC, Lung volume reduction coil, VA, Vapor ablation

*at some but not all centers

FIGURE 4.6

KEY POINTS FOR THE USE OF NON-PHARMACOLOGICAL TREATMENTS

EDUCATION, SELF-MANAGEMENT AND PULMONARY REHABILITATION

- Education is needed to change patient's knowledge but there is no evidence that used alone it will change patient behavior .
- Education self-management with the support of a case manager with or without the use of a written action plan is recommended for the prevention of exacerbation complications such as hospital admissions (**Evidence B**).
- Rehabilitation is indicated in all patients with relevant symptoms and/or a high risk for exacerbation (**Evidence A**).
- Physical activity is a strong predictor of mortality (**Evidence A**). Patients should be encouraged to increase the level of physical activity although we still don't know how to best insure the likelihood of success.

VACCINATION

- Influenza vaccination is recommended for all patients with COPD (**Evidence A**).
- Pneumococcal vaccination: the PCV13 and PPSV23 are recommended for all patients > 65 years of age, and in younger patients with significant comorbid conditions including chronic heart or lung disease (**Evidence B**).
- Covid-19 vaccination in line with national recommendations (**Evidence B**).
- Tdap (dTdap/dTPa) vaccination for adults with COPD who were not vaccinated in adolescence to protect against pertussis (whooping cough) (**Evidence B**).

NUTRITION

- Nutritional supplementation should be considered in malnourished patients with COPD (**Evidence B**).

END OF LIFE AND PALLIATIVE CARE

- All clinicians managing patients with COPD should be aware of the effectiveness of palliative approaches to symptom control and use these in their practice (**Evidence D**).
- End of life care should include discussions with patients and their families about their views on resuscitation, advance directives and place of death preferences (**Evidence D**).

TREATMENT OF HYPOXEMIA

- In patients with severe resting hypoxemia long-term oxygen therapy is indicated (**Evidence A**).
- In patients with stable COPD and resting or exercise-induced moderate desaturation, long term oxygen treatment should not be routinely prescribed. However, individual patient factors may be considered when evaluating the patient's needs for supplemental oxygen (**Evidence A**).
- Resting oxygenation at sea level does not exclude the development of severe hypoxemia when travelling by air (**Evidence C**).

TREATMENT OF HYPERCAPNIA

- In patients with severe chronic hypercapnia and a history of hospitalization for acute respiratory failure, long term noninvasive ventilation may be considered (**Evidence B**).

INTERVENTION BRONCHOSCOPY AND SURGERY

- Lung volume reduction surgery should be considered in selected patients with upper-lobe emphysema (**Evidence A**).
- In selected patients with a large bulla surgical bullectomy may be considered (**Evidence C**).
- In select patients with advanced emphysema, bronchoscopic interventions reduce end-expiratory lung volume and improve exercise tolerance, quality of life and lung function at 6-12 months following treatment. Endobronchial valves (**Evidence A**); Lung coils (**Evidence B**); Vapor ablation (**Evidence B**).
- In patients with very severe COPD (progressive disease, BODE score of 7 to 10, and not candidate for lung volume reduction) lung transplantation may be considered for referral with at least one of the following: (1) history of hospitalization for exacerbation associated with acute hypercapnia ($P_{CO_2} > 50$ mm Hg); (2) pulmonary hypertension and/or cor pulmonale, despite oxygen therapy; or (3) $FEV_1 < 20\%$ and either $DLCO < 20\%$ or homogenous distribution of emphysema (**Evidence C**).

TABLE 4.10

▶ IDENTIFY & REDUCE RISK FACTOR EXPOSURE

- Smoking cessation interventions should be actively pursued in all COPD patients (**Evidence A**).
- Efficient ventilation, non-polluting cooking stoves and similar interventions should be recommended (**Evidence B**).
- Clinicians should advise patients to avoid continued exposures to potential irritants, if possible (**Evidence D**).

▶ KEY POINTS FOR THE USE OF BRONCHODILATORS

- LABAs and LAMAs are preferred over short-acting agents except for patients with only occasional dyspnea (**Evidence A**), and for immediate relief of symptoms in patients already on long-acting bronchodilators for maintenance therapy.
- Patients may be started on single long-acting bronchodilator therapy or dual long-acting bronchodilator therapy. In patients with persistent dyspnea on one bronchodilator treatment should be escalated to two (**Evidence A**).
- Inhaled bronchodilators are recommended over oral bronchodilators (**Evidence A**).
- Theophylline is not recommended unless other long-term treatment bronchodilators are unavailable or unaffordable (**Evidence B**).

▶ KEY POINTS FOR INHALATION OF DRUGS

- The choice of inhaler device has to be individually tailored and will depend on access, cost, prescriber, and most importantly, patient's ability and preference.
- It is essential to provide instructions and to demonstrate the proper inhalation technique when prescribing a device, to ensure that inhaler technique is adequate and re-check at each visit that patients continue to use their inhaler correctly.
- Inhaler technique (and adherence to therapy) should be assessed before concluding that the current therapy requires modification.

▶ KEY POINTS FOR THE USE OF ANTI-INFLAMMATORY AGENTS

- Long-term monotherapy with ICS is not recommended (**Evidence A**).
- Long-term treatment with ICS may be considered in association with LABAs for patients with a history of exacerbations despite appropriate treatment with long-acting bronchodilators (**Evidence A**).
- Long-term therapy with oral corticosteroids is not recommended (**Evidence A**).
- In patients with severe to very severe airflow limitation, chronic bronchitis and exacerbations the addition of a PDE4 inhibitor to a treatment with long acting bronchodilators with/without ICS can be considered (**Evidence B**).
- Preferentially, but not only in former smokers with exacerbations despite appropriate therapy, macrolides, in particular azithromycin, can be considered (**Evidence B**).
- Statin therapy is not recommended for prevention of exacerbations (**Evidence A**).
- Antioxidant mucolytics are recommended only in selected patients (**Evidence A**).

▶ KEY POINTS FOR THE USE OF OTHER PHARMACOLOGICAL TREATMENTS

- Patients with severe hereditary alpha-1 antitrypsin deficiency and established emphysema may be candidates for alpha-1 antitrypsin augmentation therapy (**Evidence B**).
- Antitussives cannot be recommended (**Evidence C**).
- Drugs approved for primary pulmonary hypertension are not recommended for patients with a pulmonary hypertension secondary to COPD (**Evidence B**).
- Low-dose long acting oral and parenteral opioids may be considered for treating dyspnea in COPD patients with severe disease (**Evidence B**).

ACUTE EXACERBATIONS OF COPD

PRESENTATION

Worsening of previous stable condition. *Features:* 2:1 of:

- increase dyspnoea- marked dyspnoea, tachypnoea (>25 breaths/min), use of accessory muscles at rest and pursed lip breathing are signs of severe exacerbation
- decrease exercise tolerance-marked decrease in activities of daily living is a sign of severe exacerbation
- increase fatigue
- increase fluid retention-new-onset oedema is a sign of severe exacerbation
- increase wheeze
- Chest tightness
- increase cough
- increase sputum purulence
- increase sputum volume
- Upper airways symptom e.g., colds, sore throats,
- New-onset cyanosis-severe exacerbation
- Acute confusion-severe exacerbation

Fever and chest pain are uncommon presenting features-consider alternative diagnosis.

CAUSES OF EXACERBATIONS

30% have no identifiable cause

- Infections Viral upper and lower respiratory tract infections, e.g., common cold, influenza; bacterial lower respiratory tract infections
- Pollutants, e.g., nitrous oxide, sulphur dioxide, ozone

DIFFERENTIAL DIAGNOSIS

- Pneumonia
- LVF/pulmonary oedema
- Lung cancer
- Pleural effusion
- Recurrent aspiration
- Pneumothorax
- PE
- Upper airway obstruction

INVESTIGATIONS

- **Pulse oximetry:** can be used to assess severity (saturation: S92% breathing after suggests hypoxaemia- consider admission) and to monitor progress
- **CXR:** Consider if diagnostic doubt and/or to exclude other causes of symptoms
- **Sputum culture:** Not recommended routinely in the community.

DECIDING TO TREAT EXACERBATIONS AT HOME OR IN HOSPITAL

The more features in the 'treat in hospital' column, the more likely the need for admission.

	Treat at home	Treat in Hospital*
Ability to cope at home	Yes	No
Breathlessness	Mild	Severe
General condition	Good	Poor-deteriorating
Level of activity	Good	Poor/Confined to bed
Cyanosis	No	Yes
Worsening peripheral oedema	No	Yes
Level of consciousness	Normal	Impaired
Already receiving LTOT	No	Yes
Social circumstances	Good	Living alone/not coping
Acute confusion	No	Yes
Rapid rate of onset	No	Yes
Significant co-morbidity e.g., cardiac disease, DM	No	Yes
Changes on CXR (if available)	No	Present

DIFFERENTIAL DIAGNOSIS OF COPD EXACERBATION

WHEN THERE IS CLINICAL SUSPICION OF THE FOLLOWING ACUTE CONDITIONS, CONSIDER THE FOLLOWING INVESTIGATIONS:

▶ PNEUMONIA

- Chest radiograph
- Assessment of C-reactive protein (CRP) and/or procalcitonin

▶ PNEUMOTHORAX

- Chest radiograph or ultrasound

▶ PLEURAL EFFUSION

- Chest radiograph or ultrasound

▶ PULMONARY EMBOLISM

- D-dimer and/or Doppler sonogram of lower extremities
- Chest tomography – pulmonary embolism protocol

▶ PULMONARY EDEMA DUE TO CARDIAC RELATED CONDITIONS

- Electrocardiogram and cardiac ultrasound
- Cardiac enzymes

▶ CARDIAC ARRHYTHMIAS – ATRIAL FIBRILLATION/FLUTTER

- Electrocardiogram

POTENTIAL INDICATIONS FOR HOSPITALIZATION ASSESSMENT*

- Severe symptoms such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, confusion, drowsiness.
- Acute respiratory failure.
- Onset of new physical signs (e.g., cyanosis, peripheral edema).
- Failure of an exacerbation to respond to initial medical management.
- Presence of serious comorbidities (e.g., heart failure, newly occurring arrhythmias, etc.).
- Insufficient home support.

*Local resources need to be considered.

MANAGEMENT OF SEVERE BUT NOT LIFE-THREATENING EXACERBATIONS*

- Assess severity of symptoms, blood gases, chest radiograph.
- Administer supplemental oxygen therapy, obtain serial arterial blood gas, venous blood gas and pulse oximetry measurements.
- Bronchodilators:
 - » Increase doses and/or frequency of short-acting bronchodilators.
 - » Combine short-acting beta 2-agonists and anticholinergics.
 - » Consider use of long-active bronchodilators when patient becomes stable.
 - » Use spacers or air-driven nebulizers when appropriate.
- Consider oral corticosteroids.
- Consider antibiotics (oral) when signs of bacterial infection are present.
- Consider noninvasive mechanical ventilation (NIV).
- At all times:
 - » Monitor fluid balance.
 - » Consider subcutaneous heparin or low molecular weight heparin for thromboembolism prophylaxis.
 - » Identify and treat associated conditions (e.g., heart failure, arrhythmias, pulmonary embolism etc.).

*Local resources need to be considered.

KEY POINTS FOR THE MANAGEMENT OF EXACERBATIONS

- Short-acting inhaled beta₂-agonists, with or without short-acting anticholinergics, are recommended as the initial bronchodilators to treat an acute exacerbation (**Evidence C**).
- Systemic corticosteroids can improve lung function (FEV₁), oxygenation and shorten recovery time and hospitalization duration. Duration of therapy should not be more than 5-7 days (**Evidence A**).
- Antibiotics, when indicated, can shorten recovery time, reduce the risk of early relapse, treatment failure, and hospitalization duration. Duration of therapy should be 5-7 days (**Evidence B**).
- Methylxanthines are not recommended due to increased side effect profiles (**Evidence B**).
- Non-invasive mechanical ventilation should be the first mode of ventilation used in COPD patients with acute respiratory failure who have no absolute contraindication because it improves gas exchange, reduces work of breathing and the need for intubation, decreases hospitalization duration and improves survival (**Evidence A**).

INDICATIONS FOR RESPIRATORY OR MEDICAL INTENSIVE CARE UNIT ADMISSION*

- Severe dyspnea that responds inadequately to initial emergency therapy.
- Changes in mental status (confusion, lethargy, coma).
- Persistent or worsening hypoxemia ($\text{PaO}_2 < 5.3 \text{ kPa}$ or 40 mmHg) and/or severe/worsening respiratory acidosis ($\text{pH} < 7.25$) despite supplemental oxygen and noninvasive ventilation.
- Need for invasive mechanical ventilation.
- Hemodynamic instability - need for vasopressors.

*Local resources need to be considered.

INTERVENTIONS THAT REDUCE THE FREQUENCY OF COPD EXACERBATIONS

INTERVENTION CLASS	INTERVENTION
Bronchodilators	LABAs LAMAs LABA + LAMA
Corticosteroid-containing regimens	LABA + ICS LABA + LAMA + ICS
Anti-inflammatory (non-steroid)	Roflumilast
Anti-infectives	Vaccines Long Term Macrolides
Mucoregulators	N-acetylcysteine Carbocysteine Erdosteine
Various others	Smoking Cessation Rehabilitation Lung Volume Reduction Vitamin D Shielding measures (e.g., mask wearing, minimizing social contact, frequent hand washing)

KEY POINTS FOR THE MANAGEMENT OF STABLE COPD DURING COVID-19 PANDEMIC

PROTECTIVE STRATEGIES

- Follow basic infection control measures
- Wear a face covering
- Consider shielding/sheltering-in-place
- Have the COVID-19 vaccination in line with national recommendations

INVESTIGATIONS

- Only essential spirometry

PHARMACOTHERAPY

- Ensure adequate supplies of medications
- Continue unchanged including ICS

NON-PHARMACOLOGICAL THERAPY

- Ensure annual influenza vaccination
- Maintain physical activity

Table 1. Comparison of COPD and asthma

	COPD	Asthma
<i>Symptoms <35y</i>	Rare	Common
<i>Smoking history</i>	Nearly all	Maybe
<i>Breathlessness</i>	Persistent and progressive Good response to inhaled therapy is typical	Variable throughout the day, and from day to day. Poor response to inhaled therapy if good reconsider diagnosis
<i>Chronic productive cough</i>	Common	Uncommon
<i>Waking at night with cough/wheeze</i>	Common	Uncommon

REVERSIBILITY TESTING

Can be misleading. Not routinely recommended:

- >400 ml increase in FEV1 following trial of bronchodilator or prednisolone (30 mg od for 2 week) suggests asthma
- Clinically significant COPD is *not* present if FEV1 and FEV1/FVC return to normal after drug therapy

PEAK EXPIRATORY FLOW RATE (PEFR)

- Patients with COPD have little variability in PEFR. Serial home PEFR measurements can help distinguish between asthma and COPD. PEFR may underestimate severity of airflow limitation and a normal PEFR does not exclude airflow obstruction.

OTHER INVESTIGATIONS ORGANIZED IN PRIMARY CARE

- CXR
 - Indicated to exclude other diagnoses, e.g., lung cancer
- FBC
 - To identify polycythaemia or anaemia
- BMI
- al-antitrypsin : if early onset COPD or family history
- ECG/echo: if Cor pulmonale is suspected
- Sputum culture: if purulent sputum is persistent

MANAGEMENT

SMOKES: a consultation checklist for obstructive pulmonary diseases

- S** Smoking cessation
- M** Medication-inhaled bronchodilator, vaccines (influenza, pneumococcus), corticosteroids (if indicated)
- O** Oxygen-is it needed?
- K** Komorbidity-cardiac dysfunction, sleep apnoea, osteoporosis, depression, asthma
- E** Exercises and rehabilitation
- S** Surgery-bullectomy, lung volume reduction surgery, single -lung transplantation

Record values of spirometric tests performed at diagnosis and review. At each review record current

symptoms, problems since last seen, exercise tolerance, and smoking status. Calculate BODE score if possible. Educate the patient/family about COPD, medication, and self-help strategies.

NON-DRUG THERAPY

- Smoking cessation: Most important. Improves outcome
- Vaccination: Offer pneumococcal and annual influenza vaccination
- Exercise: Lack of exercise decrease FEV1. Pulmonary rehabilitation is of proven benefit-refer directly or via respiratory physicians
- Nutrition: Weight decrease in obese patients improves exercise tolerance

SMOKING AND COPD

FEV as % of value aged 25 yr	Age 60 yrs	Age 75 yrs
Non-smoker	85%	80%
Ex-smoker: quit aged 40 yr	60%	45% (symptoms)
Ex-smoker: quit aged 60 yr	33% (severe symptoms)	15% (severe disability)
On-going smoker	33% (severe symptoms)	Dead

REFERENCE:

1. GOLD Guideline 2022
2. Oxford handbook 4th edition

ACUTE RESPIRATORY INFECTIONS (ACUTE VIRAL INFECTIONS)

INTRODUCTION AND RELEVANCE TO GENERAL PRACTICE

- Acute respiratory infections range from self-limited conditions such as uncomplicated upper respiratory infections (URI, common cold) to serious life-threatening conditions like pneumonia. URI and acute bronchitis are among the most common reasons for visits in ambulatory care; they account for significant morbidity and absenteeism from work and school.
- Management of these infections has been complicated by recent evidence indicating a rise in the prevalence of antibiotic-resistant pathogens. The vast majority of antibiotics prescribed in ambulatory settings are for respiratory tract infections. The injudicious use of antimicrobial agents creates an environment for developing resistance, placing the population and individual patient at risk.
- Consequently, appropriate treatment of acute respiratory tract infections has become a challenge to the clinician. Acute Respiratory infection specifically URTI stands for second ranking in USA family practice and third ranking in Australia general practice regarding most frequently managed disorders.

CLASSIFICATION OF ARI

- Infections localized in respiratory structures above the larynx can be conceptualized as URI, and correspondingly those below the larynx as lower respiratory tract infections.
- An advantage to focusing on different primary sites of infection is that different pathogens are more common at certain sites than others thereby providing a guide for treatment.

Sinus And Nasal Problems

- Generally, viruses are the most common causes of respiratory complaints, and the history and physical examination are meant to detecting other reasons for these symptoms. The most common causes of noninfectious nasal discharge include allergic rhinitis and foreign bodies.
- **Key historical findings** that suggest causes other than infection for a runny nose include unilateral nasal discharge (as seen with a foreign body or necrotic tumor) or a clear nasal discharge that has persisted for several weeks (suggesting allergy).
- Differentiating bacterial from viral URI is very difficult. Evidence suggests that many of the symptoms that are typically ascribed to bacterial sinusitis occur just as often with sinus inflammation associated with common colds.
- **Two studies** have been helpful in identifying clinical cues that can be used to differentiate these two conditions. Combined, these studies suggest that patients with sinusitis are more likely to have a constellation of maxillary toothache, purulent nasal discharge by history or examination, poor sinus transillumination, and a lack of response to decongestants, along with a phenomenon termed "double sickening".
- **Double sickening** refers to patients who say they started with the symptoms of a common cold, but a few days later they "got sicker". One or two of these symptoms alone are not predictive of a sinusitis, but the presence of the entire spectrum increases the likelihood of a secondary sinusitis.

Sore Throats

- **Sore throats or hoarseness** may be caused by a limited number of other conditions. These are primarily laryngeal inflammation from acute insults such as smoke inhalation, chronic abuse from smoking, or singing.
- **Vocal cord neoplasm** may also be suspected in patients with chronic hoarseness, especially if risk factors such as cigarette smoking or alcohol use are present.
- Pharyngeal infections are even more difficult to differentiate into bacterial- or viral-based on clinical criteria. **Decision rules (CENTOR SCORE)** using fever, tonsillar exudate, cervical adenopathy, and the lack of either a runny nose or cough have been evaluated to identify patients with streptococcal pharyngitis, but have had mixed success.
- Another difficulty in evaluating decision rules is that a large segment of the population (up to 20%) can be colonized with group A Streptococcus; so, a positive culture may not indicate infection with this agent in a fairly large group of patients.
- However, at this time either rapid or conventional throat cultures remain the best way to differentiate a strep throat from pharyngitis associated with a cold.

Cough And Lower Respiratory Symptoms

- For **lower respiratory symptoms** such as cough, additional noninfectious causes should be considered. Congestive heart failure, aspiration of gastric contents or foreign bodies, lung neoplasms, asthma, and other inflammatory pulmonary conditions can produce a cough and shortness of breath.
- **Congestive heart failure and pulmonary embolism** also can cause pulmonary infiltrates and effusions that can be confused with pneumonia. Medications, most notably angiotensin-converting enzyme inhibitors, may cause a cough as well.
- If **a lower respiratory infection** is suspected, several possible conditions should be considered. First, it is important to differentiate acute bronchitis, a self-limited condition, from pneumonia.
- The presence of **rales and/or a localized decrease in breath sounds** suggest pneumonia, but may not be sensitive. Patients who appear to be ill yet have no abnormal physical findings on lung examination that suggest pneumonia should have a chest radiograph.
- In addition to pneumonia, other infectious conditions can cause rales and productive cough. Pulmonary abscesses may produce undulating fevers, shortness of breath, and cough.
- **Abscesses** are associated with certain types of pneumonia-causing organisms such as Staphylococcus sp. Bronchiectasis is a bronchial wall disease that allows accumulation of large amounts of secretions in the diseased bronchus.
- The **copious secretions** result in a chronic productive cough. Furthermore, these pooled secretions often become infected and can produce a fever, shortness of breath, and purulent sputum production consistent with pneumonia.

DIFFERENTIAL DIAGNOSIS

UNCOMPLICATED URI/COMMON COLD

- URI, or the "**common cold**," are common infectious conditions often seen in the ambulatory care setting. Adults typically have two to four URI annually, and children in day care have as many as six or seven.
- Although **URI are mild, self-limited**, and of short duration, they are a leading cause of acute morbidity, and industrial and school absenteeism. Each year, URI account for 170 million days

of restricted activity, 23 million days of school absence, and 18 million days of work absence. And while colds may be viewed as benign, the impact of URI on quality of life is similar in magnitude to such chronic illnesses as chronic lung disease, depression, or osteoarthritis.

- The **mechanisms of transmission** suggest that URI can be spread through contact with inanimate surfaces as well as direct hand-to-hand contact. URI have a seasonal variation, with an increased prevalence in the United States between September and March. It is unclear why this variation exists although it may be related to increased crowding of indoor populations in the colder months.
- **Temperature** is not the key to seasonal variation without the presence of a pathogen. Evidence from Antarctica showed that spacious well-ventilated rooms reduced transmission of URI compared to crowded poorly-ventilated rooms regardless of temperature.
- **One study** was able to identify the specific virus believed to cause a URI in 69% of all URI cases. Rhinoviruses were the most prevalent virus and were seen in 52% of the patients. Coronaviruses were the second most common group of causative agents, followed by influenza A or B virus. Identified bacterial pathogens were *Chlamydia pneumoniae*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Mycoplasma pneumoniae*. None of the patients had beta-hemolytic group A *Streptococcus*. In terms of bacterial pathogens, infections without evidence of a viral infection occurred in only 0.05% of the cases.
- **Uncomplicated URI** are characterized by rhinorrhea, nasal congestion, sneezing, sore or "scratchy" throat, and cough. The incubation period varies between 48 and 72 hours. In some cases, a low-grade fever is present, but temperature elevation is rare in adults. The early symptoms may be minimal and limited to malaise and nasal symptoms.
- The **nasal discharge** is initially clear and watery. There is a subsequent transition period where the nasal discharge becomes viscous, opaque, and discolored (white, yellow, green). The color of the secretions alone is not predictive of a bacterial infection.
- The clinical presentation is similar in both adults and children. The episode tends to be self-limited. The median duration of a cold is 1 week, with most patients improving by day 10, but lingering symptoms may last up to two weeks.

ACUTE SINUSITIS

- Because **sinusitis** usually is a complication of upper respiratory viral infections, the incidence peaks in the winter. Among children, sinusitis is frequently found as a comorbidity with otitis media.
- **Children** are also more likely to have posterior ethmoidal and sphenoid inflammation, while adults have mainly maxillary and anterior ethmoidal sinusitis.
- Some **medical conditions** may increase the risk for sinusitis. These include cystic fibrosis, asthma, immunosuppression, and allergic rhinitis. Cigarette smoking may also increase the risk of bacterial sinusitis during a cold because of reduced muco-ciliary clearance.
- **Sinus inflammation** can be caused by viral, fungal, and bacterial infections as well as allergies. Most acute sinusitis is caused by viral infection. The inflammation associated with viral infections clears without additional therapy.
- **Cultures from patients with sinusitis** show that the most prevalent organisms are *S. pneumoniae* and, especially in smokers, *H. influenzae*. These two organisms are present in 70% of bacterial acute sinusitis cases. When antibiotics are used to treat bacterial sinusitis, selection criteria should include sufficient coverage of these two organisms.
- **Fungal sinusitis** is very rare and usually occurs in immunosuppressed individuals or those with diabetes mellitus.

- Acute sinusitis has considerable overlap in its constellation of signs and symptoms with URI. One half to two thirds of patients with sinus symptoms seen in primary care are unlikely to have sinusitis. UR is often precursors of sinusitis, and at some point, symptoms from each condition may overlap.
- Sinus inflammation from a URI without bacterial infection is also common. In a series of 60 children undergoing computerized tomography (CT) for non-sinus-related diagnoses, 47% had evidence of sinus inflammation with no clinical signs of sinusitis, and with complete resolution following their viral illness.
- Acute sinusitis tends to start with a URI that leads to sinus ostial obstruction. The signs and symptoms that increase the likelihood that the patient has acute sinusitis are a "double sickening" phenomenon, maxillary toothache, purulent nasal discharge, poor response to decongestants, and a history of discolored nasal discharge (4,23). Other authors have stressed that the symptoms need to persist longer than 1 week to distinguish sinusitis from a URI (24). It should be pointed out that the commonly used sign of facial pain or swelling has low sensitivity for acute sinusitis.

ACUTE BRONCHITIS

- **Acute bronchitis** in the otherwise healthy adult is one of the most common medical problems encountered in primary care. The prevalence of acute bronchitis peaks in the winter and is much less common in the summer.
- **Viral infection** is the primary cause of most episodes of acute bronchitis. A wide variety of viruses have been shown as causes of acute bronchitis including influenza, rhinovirus, adenovirus, coronavirus, parainfluenza, and respiratory syncytial virus. Nonviral pathogens including *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* have also been identified as causes.
- The etiologic role of bacteria like **H. influenzae** and **S. pneumoniae** in acute bronchitis is unclear because these bacteria are common upper respiratory tract flora. Sputum cultures for acute bronchitis are therefore difficult to evaluate since it is unclear whether the sputum has been contaminated by pathogens colonizing the nasopharynx.
- **Acute bronchitis** is an inflammatory condition of the tracheobronchial tree usually associated with a generalized respiratory infection. Cough begins early in the course of the illness and is the most prominent feature of the condition. An initially dry cough may later result in sputum production, which characteristically changes from clear to discolor in the later stages of the illness. The cough may last for a significant time. Although the duration of the condition is variable, one study showed that 50% of patients had a cough for more than 3 weeks, and 25% had a cough for more than 4 weeks.
- **Patients with acute bronchitis** usually have a viral respiratory infection with transient inflammatory changes that produce sputum and symptoms of airway obstruction. Acute bronchitis is essentially a diagnosis of exclusion. The history should include information on cigarette use, exposure to environmental toxins (e.g., dust, beryllium, volatile organic compounds, asbestos), as well as medication history (e.g., use of angiotensin converting enzyme inhibitors). The chronicity of the cough should be established to distinguish acute bronchitis from chronic bronchitis since they have different treatments.
- Both **acute bronchitis and pneumonia** can present with fever, constitutional symptoms, and a productive cough. While patients with pneumonia often have rales, this finding is neither sensitive nor specific for the illness. When pneumonia is suspected because of a high fever, constitutional symptoms, severe dyspnea, and certain physical findings or risk factors, a chest radiograph should be obtained to confirm the diagnosis.
- **Asthma and allergic bronchospastic disorders** can mimic the productive cough of acute bronchitis. When obstructive symptoms are not obvious, mild asthma may be misdiagnosed as

acute bronchitis. Further, since respiratory infections can trigger bronchospasm in asthma, patients with asthma that occurs only in the presence of respiratory infections may present as patients with acute bronchitis.

- **Asthma** should be considered in patients with repetitive episodes of acute bronchitis. Patients who repeatedly present with cough and wheezing can be given pulmonary function testing with and without a bronchodilator. For patients for whom routine pulmonary function testing is equivocal but asthma is highly suspected, further or provocative testing with a methacholine challenge test may help differentiate asthma from recurrent bronchitis.

BRONCHIOLITIS

- **Acute bronchiolitis** is a distinct syndrome occurring in young children with a peak incidence at 6 months of age. It is an acute respiratory illness resulting from inflammation of small airways and characterized by wheezing.
- Children generally acquire the infection from family members or other children in day care who are infected with an upper respiratory tract infection. Bronchiolitis is not uncommon, with approximately 15% of children experiencing this illness during the first 2 years of life.
- **Respiratory syncytial virus (RSV)** is the most common cause of bronchiolitis accounting for between 50% and 90% of cases. The majority of the cases occur during the winter and early spring mirroring the prevalence of the viral pathogens in the community.
- **The diagnosis of** bronchiolitis is based on clinical findings and on the knowledge of the prevalence of viral pathogens prevalent in the community. The classic signs of bronchiolitis are wheezing and hyperexpansion of the lungs.
- **Bronchiolitis begins** as a URI, but soon the patient develops a cough, audible wheezing, irritability, listlessness, dyspnea, and cyanosis. Chest radiographs may reveal atelectasis, hyperinflation, or both. Untreated, infants with bronchiolitis can die from hypoxemia, dehydration, or apnea; less than 1% of affected infants die, however. Most recover but suffer recurrent wheezing episodes, usually precipitated by viral infections

INFLUENZA

- Approximately **10% to 20%** of the population in the United States develops influenza annually, with influenza season usually peaking between late December and early March.
- In Myanmar, influenza season starts from mid-May to end of August, that is, rainy season. In recent influenza seasons, especially when influenza A type **H3N2** predominated, **80% to 90%** of influenza-related deaths occurred in individuals older than 65 years of age. It is the fifth leading cause of death in individuals over 65, and the most common infectious cause of death in this country.
- Rates of disease are increased in individuals 65 years of age or older and in those with underlying health problems, such as diabetes mellitus and coronary artery disease. Influenza is caused by highly infectious RNA viruses of the orthomyxovirus family.
- **Influenza A viruses** are classified into subtypes based on two surface antigens- hemagglutinin (H) and neuraminidase (N). Changes in the H or N antigen account for the epidemiologic success of these viruses. Infection with one subtype however, provides little protection against infection with other subtypes, and infection or vaccination with one strain does not result in immunity to distantly related strains of the same subtype because of antigenic drift. Consequently, major epidemics of respiratory disease are caused by influenza virus strains not represented in that year's vaccine.
- Occasionally, there are **major shifts in the antigenic composition** of the influenza virus. Such

shifts are believed to be associated with the deadly pandemic of influenza in 1917-1918 as well as pandemics in 1957 and 1968. What made the 1918 influenza pandemic so remarkable was the high fatality rate in young to middle-age adults. Concerns about emerging antigen shifts associated with avian flu viruses compare possible consequences to the 1918 pandemic, although there is no evidence that an avian strain was associated with this prior epidemic.

- During the initial evaluation of an influenza-like illness, the following entities must be considered in the differential: respiratory syncytial virus, parainfluenza, adenovirus, enterovirus, mycoplasma, chlamydia, and streptococcal disease. Influenza is extremely contagious and is transmitted from person to person through small particles of virus-laden respiratory secretions that are propelled into the air by infected persons during coughing, sneezing, and talking.
- **The abrupt onset of fever, myalgia, sore throat, and a nonproductive cough** characterize the typical influenza infection. Symptoms usually last 1 to 5 days. Unlike other common respiratory illnesses, influenza viruses cause severe malaise lasting several days. The symptoms vary by age, with children commonly presenting with cough, rhinorrhea, and croup, while adults present with cough, myalgia, sore throat, and headache. The elderly usually complains of cough alone or in combination with headache. A key to diagnosis is being aware of influenza outbreaks in the community at the time of presentation.
- **The availability of rapid tests** to identify influenza has made it possible to evaluate patients quickly for this disorder. However, the cost-benefit of this approach is questionable. Most studies show that during flu season when influenza is highly likely, it is most economical to simply treat the patient. The value of office-based testing is probably during the "shoulders" of flu season when the probability of influenza is lower.

CLINICAL EVALUATION (DIAGNOSTIC WORK UP)

- Patients with acute respiratory infections need a focused history that includes current and recent symptoms, duration of episode, prior episodes and treatment, other family members affected, risk factors, and smoking history.
- In addition, several red flags in the history and physical examination may alert the clinician to noninfectious or life-threatening emergencies

HISTORY AND PHYSICAL EXAMINATION

- **Fever is a nonspecific sign**, but can be used to discriminate mild, **self-limited problems**, such as acute bronchitis, from more significant infections such as pneumonia. Both the height of the temperature and the pattern of its development can give clues to the diagnosis. For example, URI ("colds") generally cause little or no fever in older children and adults.
- However, a child with symptoms of a common cold but who also has a high fever might be suspected of having otitis media, sinusitis, or another bacterial infection. Fever also can be used to discriminate between different types of virally-mediated illnesses such as the common cold and influenza, since influenza generally produces a high fever.
- **Rhinorrhea**, either watery or purulent, indicates inflammation in the nasal cavities from either an infectious or noninfectious source. Although viral illnesses commonly cause runny noses, noninfectious conditions such as allergic rhinitis (hay fever) and a foreign body in the nose can also present with rhinorrhea. **In viral illnesses**, the nose is usually red and swollen with patchy areas of exudates. In contrast, in allergic rhinitis, the mucosa usually is swollen (boggy) and often pale with a clear, glistening surface and little exudate. With foreign bodies, generally only one nostril is affected and the drainage is usually purulent and foul-smelling.

- **Headache** is another symptom that can arise from respiratory and nonrespiratory structures. Frontal headache, particularly if it gets worse by bending over, suggests sinus inflammation from either a cold or sinusitis. Facial pain is another symptom of sinus disease, since portions of the sinuses, the ear, and the skin of the face are all supplied by the trigeminal nerve.
- However, as noted below, facial pain alone is not a good discriminator of sinusitis since many patients with common colds also have sinus inflammation. Sinus inflammation also can cause maxillary toothache because the superior alveolar nerve passes through that sinus.

Red Flags of History and Physical Examination

Red Flag	Other Condition to consider
Unilateral purulent nasal drainage	Occult nasal foreign body
Severe sore throat with deviation of the uvula laterally	Peritonsillar abscess
Difficulty swallowing with stridor	Epiglottitis
Hoarseness persisting greater than 30 days	Laryngeal cancer or nodule
Cough persisting greater than 30 days	Lung cancer
Hemoptysis	Bronchial lung cancer
Cough with unilateral wheezing	Bronchial foreign body
Early morning cough with hoarseness	Reflux esophagitis
Shortness of breath with unilateral decreased breath sounds	Spontaneous pneumothorax

- **Sore throats** can result from direct inflammation of the throat caused by a variety of different pathogens. The type of pain associated with streptococcal pharyngitis is not helpful in differentiating strep throat from virally mediated infections. However, very severe pain with difficulty swallowing may be indicative of a peritonsillar abscess. Severe pain and swallowing difficulty also is common with herpangina, a Coxsackie viral infection of the throat, palate, and posterior tongue.
- **Pharyngeal inflammation** also can cause referred pain to the ear because the middle ear shares innervation from the glossopharyngeal nerve.
- **Hoarseness** generally indicates narrowing of the airway in the region of the larynx. Typically, the cause is inflammation of the vocal cords from laryngitis. In small children, narrowing of the same air passage leads to stridor.
- **Cough** is the most common symptom observed in lower respiratory tract infections, but is common in URI as well; Inflammation of the trachea, bronchi, bronchioles, or alveoli causes cough regardless of the etiology of the inflammation.
- Infections also cause hypersecretion of and production of infectious exudates that are cleared with coughing. Some infections, such as mycoplasma influenza and other viral infections, produce inflammation without a great deal of exudate and are typified by a nonproductive or dry cough. The degree of cough provides little indication of disease severity; many viral respiratory infections cause severe, persistent cough, even when they are largely resolved.
- **Chest pain** can occur with some respiratory infection, but it is a rare symptom in isolation. Usually chest pain is present with coughing, shortness of breath, fever, or some other sign of infection. Chest pain with infection is usually pleuritic in nature and represents pleural inflammation from the infectious process.
- **The pleuritis** can be mild as seen in some cases of acute bronchitis, severe as with some Coxsackie B virus infections, or associated with significant pleural effusions and respiratory compromise in pneumonias. Because other conditions, such as a pulmonary embolism, can cause chest pain and low grade fever, clinicians should consider non-infectious causes as well as respiratory infections when patients complain of chest discomfort.

- Finally, **cough can lead to chest pain** by straining or otherwise injuring the muscles and bones of the chest wall or by irritating an inflamed trachea or bronchi. Red flags in the history that suggest other disorders are shown in Table described above.
- For patients with respiratory tract disease, a thorough physical examination often confirms the diagnosis that was suspected after a careful history. When evaluating a patient with a potential respiratory infection, **a first step** is to assess the overall appearance of the patient, and his or her vital signs. Patients who are comfortable, breathing easily, and afebrile are unlikely to have a life-threatening disease.
- However, tachycardia, tachypnea, and alterations in mental status are ominous signs that are associated with much higher death rates from respiratory infections such as pneumonia.
- **After initial assessment**, careful attention should be paid to the ears, nose, throat, neck, and chest. As noted earlier, the appearance of the nasal mucosa may be helpful in determining if rhinorrhea is caused by infection as opposed to allergy.
- **Palpation of the sinuses** is sometimes performed, but since this maneuver offers little value in differentiating sinusitis from a common cold and is likely to be highly operator-dependent, it is of little value. Transillumination of the sinuses has a much higher value in differentiating a sinusitis from a cold.
- **Pharyngeal findings** of erythema, tonsillar enlargement, and exudate are useful in identifying a likely infection.
- **Exudative tonsillitis** is common with streptococcal pharyngitis, but is seen just as frequently in adenoviral throat infections; exudative tonsillitis also is a feature of mononucleosis. Uvular deviation is another important sign to look for during the pharyngeal examination. Deviation of the uvula is an early sign of a peritonsillar abscess; in these cases, the uvula points away from the side with the abscess.
- **Cervical adenopathy**, which is common with streptococcal pharyngitis and mononucleosis, should be searched for during the neck examination. Palpation of the neck also may be useful in detecting an enlarged thyroid or other mass that may be compressing the trachea and producing stridor.
- A complete lung examination is crucial in patients with suspected lower respiratory infections. Auscultation over all areas of the lungs is important to detect rales or a rub from pleuritis. Percussion of the lower lung fields may be useful in detecting pleural effusions.

DIAGNOSTIC TESTING

- Routine laboratory testing is not indicated for the vast majority of respiratory infections. The most helpful tests for URI are a throat culture and serum testing for mononucleosis.
- Because of delays in the appearance of anti-mononucleosis antibodies, the commonly used mononucleosis tests may be falsely negative in the first week of symptoms, so testing should be delayed until symptoms have been present for a week or longer.
- Sinus radiographs or CT scans offer little benefit over clinical criteria for sinusitis and should be reserved only for patients with very confusing clinical pictures or fever without a clear origin.
- **For lower respiratory infections, the most valuable test is a chest radiograph.** This is useful in differentiating pneumonia from acute bronchitis or other causes of shortness of breath and cough. The appearance of the infiltrate on the chest radiograph is sometimes helpful in predicting the etiologic agent as well.
- However, clinicians should be wary of false negative chest radiographs, which can occur in patients who are dehydrated or neutropenic.
- Sputum cultures are less helpful. Cultures are often unreliable or contaminated. Many studies have found that only about one third of hospitalized patients with pneumonia are able to produce adequate specimens for culturing. Empiric treatment based on the age of the patient, severity of illness, and other risk factors is the most cost-effective strategy.

MANAGEMENT OF UTRI

- **Patient management and treatment is based on the condition.** The following table summarizes the evidence for treatment options for most acute respiratory infections.
- Uncomplicated URI/Common Cold
- **The most effective symptomatic treatments** are over the counter decongestants. Preparations containing pseudoephedrine are effective in treating the nasal congestion associated with the common cold. However, several states have begun regulating the purchase of pseudoephedrine because of its integral role in the illicit manufacture of methamphetamine.
- Despite the viral etiology of common colds, several studies have shown that the majority of common cold cases seen by physicians are treated with antibiotics. It should be noted that many individuals do not seek care from physicians for colds and therefore are limited in their ability to use antibiotics.
- **Controlled trials of antibiotic treatment of URI have consistently demonstrated no benefit.**
- In seven trials of antimicrobial treatment of URI, six found no difference in improvement or further complications between the groups. Complications tend to be minimal and occur at a rate of 1%-15%. One trial found a slight benefit in decreasing purulent rhinitis. Similarly, an additional trial attempted to isolate "bacterial colds," for which antibiotics might be effective treatments. Although there was some indication of patient improvement at day 5, the differences were gone by day 10. It should be noted that the normal presentation of a URI is 1 to 2 weeks.
- In addition, it is important to emphasize that the use of antibiotics for URI does not prevent bacterial complications such as otitis media.
- Many alternatives to antibiotics for URI have been investigated and have their advocates, if not strong evidence of effectiveness. Zinc gluconate lozenges are available without a prescription and have been suggested as effective in decreasing the duration of the common cold. However, one meta-analysis of eight randomized trials and another of seven trials.
- It is concluded that **zinc lozenges were not effective** in reducing the duration of cold symptoms.
- Echinacea has shown mixed results as a treatment for URI in both children and adults. Although several smaller studies have observed some benefit, larger studies have tended to find no significant benefit for using Echinacea. Several recent studies have found echinacea beneficial when compounds are used that mix echinacea with other treatments such as vitamin C or tea.
- **Vitamin C** also has been advocated for URI; systematic reviews of the literature, however, provide **only weak support for its effectiveness**. Antihistamines, with a few exceptions, have not been shown to be effective treatments.
- Other treatments also are being evaluated for use in URI. Ipratropium bromide has demonstrated use in controlling congestion and rhinorrhea, but its cost has limited its usefulness to date.
- Other treatments that are being investigated for URI include acupuncture, nitric oxide, vitamin E, and North American ginseng. Some appear promising, but it is premature to recommend them as treatments because of limited evidence of their benefits.

ACUTE SINUSITIS

- Antibiotics are commonly prescribed for adult patients who present with complaints consistent with acute sinusitis. The effectiveness of antibiotics is unclear, although some evidence supports their use. Four recent placebo-controlled, double-blind, randomized trials of antibiotics for acute sinusitis encountered in general practice settings have yielded mixed results.
- **Two of these trials showed no beneficial effect of antibiotics**, but two demonstrated significant effects of penicillin and amoxicillin. The trials demonstrating effects suggested that patients with more severe signs and symptoms, as well as radiographic or CT confirmation, may benefit from an antibiotic.
- A meta-analysis of 32 randomized trials of antibiotics versus placebo and antibiotics of different

classes captured in computerized databases such as MEDLINE and studies from pharmaceutical companies indicated that acute maxillary sinusitis may benefit from treatment with penicillin or amoxicillin for 7 to 14 days.

- If an antibiotic is used, evidence using trimethoprim/sulfamethoxazole suggests that short duration treatment (e.g., 3 days) is as effective as longer treatment (76). Further, a meta-analysis indicates that narrow spectrum agents are as effective as broad spectrum agents.

ACUTE BRONCHITIS

- Antibiotic treatment for acute bronchitis is quite common, with evidence indicating that 60%-75% of adults visiting a doctor for acute bronchitis receive an antibiotic. Clinical trials of the effectiveness of antibiotics in treating acute bronchitis have had mixed results.
- One reason for the lack of consensus is that in each of the nine trials, different antibiotics were used as well as different outcomes. In an effort to quantitatively review the data, three different meta-analyses were recently conducted. In one meta-analysis, neither resolution of cough nor clinical improvement at reexamination was affected by antibiotic treatment. Importantly, the side effects were more common in the antibiotic groups compared to placebo.
- The other **two meta-analyses** concluded that antibiotics may be modestly effective for acute bronchitis. All of the meta-analyses agreed that the benefits of antibiotics in a general population are marginal and should be weighed against the impact of excessive use of antibiotics on the development of antibiotic resistance.
- Data from clinical trials suggests that bronchodilators may provide effective symptomatic relief to patients with acute bronchitis. Treatment with bronchodilators demonstrated significant relief of symptoms including faster resolution of cough, as well as return to work.
- However, one study evaluated the effect of albuterol in a population of patients with undifferentiated cough and found no beneficial effect. Since a variety of conditions present with cough, there may have been some misclassification in generalizing this to acute bronchitis.
- **The mainstay of therapy consists of respiratory support**, nutrition, and hydration. Bronchodilators have been suggested as treatments for bronchiolitis, but a meta-analysis of eight trials of bronchodilators versus placebo indicated that bronchodilators produce only limited short-term improvement in clinical scores. This small benefit must be weighed against the costs of these agents.
- **The use of antivirals in the treatment of RSV infections remains controversial.** Ribavirin is the only antiviral agent licensed for the treatment of RSV infections and should be considered in severely affected children. Patients undergoing therapy should be placed in negative pressure rooms with frequent air exchanges and scavenging systems to decrease ribavirin exposure of health care providers and to minimize release into the surrounding environment.
- Uncontrolled trials show that early therapy with ribavirin aerosol may be beneficial. In a systematic review of 10 trials of ribavirin for RSV, it was concluded that the trials lack sufficient power to provide reliable estimates of the effects. The cumulative results of three small trials show that ribavirin reduces length of mechanical ventilator support, and may reduce days of hospitalization.
- Vaccines against RSV are under development but not currently available. Hand washing is currently the most effective preventive measure.
- Cough nor clinical improvement at reexamination was affected by antibiotic treatment. Importantly, the side effects were more common in the antibiotic groups compared to placebo. The other two meta-analyses concluded that antibiotics may be modestly effective for acute bronchitis.
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INFLUENZA

- **Treatment of influenza infection** is targeted toward symptoms, with spontaneous recovery within 5 to 7 days. The typical therapy includes bed rest, oral hydration, acetaminophen or NSAIDs to reduce fever, headache, and myalgias, over the counter throat lozenges, intranasal anticholinergics, and systemic antihistamines and anticholinergics. Preventative measures along with antiviral drugs are used to shorten the disease course and decrease secondary complications.
- **In addition to symptomatic medications, influenza may be treated with antiviral agents.** First generation antiviral agents effective against influenza A are amantadine hydrochloride and rimantadine hydrochloride.
- These drugs have no effect on influenza B viruses, however. A meta-analysis of the randomized and quasi-randomized trials indicate that both amantadine or rimantadine are effective in reducing the severity and duration of symptoms, particularly if given within 48 hours of illness onset. Unfortunately, these drugs have shown substantial rates of adverse events.
- **Two neuraminidase inhibitors, zanamivir and oseltamivir,** have been successful against both influenza A and B. An analysis of six randomized placebo-controlled trials of zanamivir indicated a decrease in illness duration ranging from 1 to 3 days in different populations.
- The greatest benefit was found in patients >50 years of age. Randomized trials of oseltamivir have also demonstrated reduced duration of illness. The treatments must be given within 36 hours, and preferably within 24 hours, of onset for maximum effectiveness.

PREVENTION

- **Vaccination for influenza** is the **most common** and effective way to prevent influenza. Each year's vaccine contains three virus strains representing the viruses that are predicted to circulate in the United States during the upcoming influenza season.
- Unfortunately, sometimes the supply of influenza vaccine is delayed or insufficient to cover all patients recommended for immunization.
- It is recommended that the following individuals receive the influenza vaccine:
 - persons aged 65 years and older
 - residents at least 6 months of age in nursing homes or chronic care facilities
 - individuals 6 months or older who have underlying medical conditions
 - individuals 6 months to 18 years of age who receive long-term aspirin therapy and have an increased risk for developing Reye's syndrome after being infected with influenza virus
 - women who will be at or beyond 14 weeks gestation during the influenza season or at any stage, if underlying medical conditions may result in secondary complications
 - Employees of hospitals, outpatient settings, nursing homes, and other chronic care facilities that care for high-risk patients.
 - Because of the decline of immunity during the year and antigen variation from year to year within the influenza virus, individuals should receive the vaccine every year. The optimal time for vaccination is May to June.

Drug	Dosage	Adverse effects	Cost per course (\$)*	Comments
Amantadine	100 mg BID for 3-5 days Reduce dose in elderly (100 mg/D) Dose in children 5 mg/kg/D in 2 divided doses	Confusion, insomnia, depression, nervousness, nausea, anorexia, dry mouth	\$	Start within 48 hrs: Type A only shortens symptoms by 1 day
Rinantidine	100 mg BID for 3-5 days Reduced dose in elderly (100 mg/D) Dose in children under 10 is 5 mg/kg/D once a day	Nausea, dizziness	\$	Start within 48 hrs: Type A only shortens symptoms by 1 day
Zanamivir	2 inhalations Q12 hrs for 5 days Approved in children 0.5- <u>7</u> yrs of age	Cough	NA	Start within 30 hrs: Type A & B shortens symptoms by 1.5 days
Oseltamivir	75 mg BID for 5 days Approved in children 0.5-1 yr of age, >15 kg-30 kg, 45 mg BID; >24-40 kg, 60 mg BID; >40 kg, 75 mg BID	Nausea and vomiting; Insomnia; vertigo; bronchitis	\$\$\$	Initiate treatment within 2 days of symptoms; Type A & B shortens symptoms by 1.5 days

\$ = <\$ 33, \$\$ = \$ 34-66, \$\$\$ = > \$ 67

Evidence supporting management of common respiratory tract infections

Treatment strategy	Strength of recommendation (SOR)	Recommendation/conclusion
Antibiotic therapy	A	No benefits seen and complications higher in treated groups
Use of decongestants	A	Assist in symptom control, no effect on duration of illness
Echinacea products	B	Early trials demonstrated modest benefit, better controlled trials show no benefit
Zinc lozenges	A	Effectiveness in adults not shown; one randomized trial shown no benefit in children
Antihistamine therapy	A	No benefit at relieving symptoms or altering duration of disease
Acute bronchitis		
1. Use of routine Antibiotics	A	Meta-analyses show weak/modest benefit ; no evidence of effectiveness in single trials of individual drugs
Use of short acting bronchodilators	A	Two randomized trials showed benefit in cough duration
Influenza		
Use of amantadine/rimantadine	A	Useful in influenza A only if started in first 48 hours
Use of neuraminidase inhibitors	A	Useful in influenza A and B but only if started in first 30-36 hours

		Beneficial in preventing complications in high-risk patients
Sinusitis		
Antibiotics	A	Useful, but benefit limited to small number of patients; highly dependent on accurate diagnosis
http://www.aafp.org/lafpsort.xml		

REFERENCE:

1. *Essentials of Family Medicine* by D.Sloane et.al, fifth edition, Lippincott William & Walkins publishing.

PNEUMONIA IN ADULTS

Pneumonia

An acute lower respiratory tract infection associated with fever, symptoms and signs in the chest, and abnormalities on the chest x-ray.

CLASSIFICATION AND CAUSES

1. Community-acquired pneumonia
2. Hospital-acquired.
3. Aspiration:
4. Immunocompromised patient:

COMMUNITY ACQUIRED PNEUMONIA

DEFINITION

A syndrome of infection that is usually bacterial, with symptoms and signs of consolidation of parts of the lung parenchyma. Community-acquired pneumonia is the most common type of pneumonia. It occurs outside of hospitals or other health care facilities. It may be caused by:

- Bacteria.
- Bacteria-like organisms
- Fungi.
- Viruses, including COVID-19.

COMMON CAUSATIVE ORGANISMS

- Pneumococcus (*S. pneumoniae* -36%)
- *H. influenza* (10%) - more common amongst the elderly
- *Influenza A and B* (8%) – annual epidemics during the winter months - 73% develop pneumonia
- *Mycoplasma pneumonia* (1.3%) - less common in the elderly;
- Gram (-)ive enteric bacteria (1.3%)
- *C. psittaci* (1.3%) - 20% have history of bird contact
- *S. aureus* (0.8%) - more common in the winter months; may be associated with viral infection, e.g., flu
- *Legionella* spp. (0.4%) - most common in September/October; >50% related to travel

RISK FACTORS FOR CAP

- Aspiration
- Alcoholism and diabetes
- Oral steroids/immunosuppression
- Cigarette smoking
- COPD
- Nursing home residents

CLINICAL FEATURES

1. Fever
2. Cough
3. Sputum
4. SOB
5. Pleuritic chest pain
6. Non-specific features in elderly - confusion, hypothermia

EXAMINATION

1. Raised RR (may be the only sign in the elderly)
2. Tachycardia
3. Localizing signs on chest examination. Reduced chest expansion on the affected side, with signs consistent with consolidation (reduced air entry, with bronchial breathing, reduced percussion note, increased vocal resonance) crackles. A normal chest examination makes the unlikely.

DIAGNOSIS

- Symptoms and signs of an acute lower respiratory tract infection
- New focal chest signs
- New radiographic shadowing
- At least one systemic feature
- No other explanation for illness

SEVERITY

'CURB-65' is a simple, validated severity scoring system one point for each of:

- **C**onfusion (abbreviated mental test ≤ 8)
- **U**rea $> \text{mmol/L}$
- **R**espiratory rate $\geq 30/\text{min}$
- **B**P < 90 systolic and/or 60 mmHg diastolic
- **A**ge ≥ 65
 - 0-1, po antibiotic/home treatment
 - 2- hospital therapy
 - ≥ 3 severe pneumonia indicates mortality 15-40% -consider ITU

COMPLICATIONS

- Respiratory failure
- Hypotension
- Atrial fibrillation
- Pleural effusion
- Empyema
- Lung abscess
- Septicaemia
- Pericarditis and myocarditis
- Jaundice –cholestatic and may be due to sepsis

MANAGEMENT

- Consider the need for admission
- Have a low threshold for admission if ill but afebrile, concomitant illness (e.g., heart failure, chronic lung, renal or liver disease, DM, cancer), or poor social situation. If life-threatening

infection or considerable delay (>2h) consider administering antibiotics before admission

If a decision is made to treat at home

- Advise not to smoke, to rest, and drink plenty of fluids
- Start antibiotics, e.g., amoxicillin 500 mg- 1 g tds, or doxycycline 100-200 mg od, or clarithromycin 500 mg bd for 5 days course
- Treat pleuritic pain with simple analgesia, e.g., paracetamol 1 g qid
- Review within 48 hr. Reassess clinical state. If deteriorating or not improving consider CXR or admission
- Most antibiotics are used empirically at diagnosis of CAP in the absence of microbiological information
- Early antibiotic administration is associated with an improved outcome

It is vital there is no delay in the administration of the first antibiotic dose in patients with confirmed CAP. Confirmation of pneumonia with CXR and antibiotic administration should occur within 4 hours of admission.

CAP: ANTIBIOTICS

Suggested empirical antibiotics for CAP treatment

	Preferred treatment	alternative
Community treatment	Amoxicillin 500mg-1 g tds PO	Doxycycline 100mg od (after 200mg loading dose) po OR Clarithromycin 500mg bd PO
Hospital treatment: low severity (CURB-65=0-1)	Amoxicillin 500mg tds PO (or same dose IV if oral treatment impossible)	Doxycycline 100mg od (after 200mg loading dose) po OR Clarithromycin 500mg bd PO
Hospital treatment: moderate severity (CURB-65= 2)	Amoxicillin 500mg-1 g tds PO Clarithromycin 500mg bd PO If oral treatment. If oral treatment is impossible, amoxicillin 500mg tds IV or benzylpenicillin 1.2g qds IV and clarithromycin 500mg bd IV	Doxycycline 100mg od (after 200mg loading dose) PO or levofloxacin 500mg od PO or moxifloxacin 400mg od PO
Hospital treatment: high severity (CURB-65= 3-5)	Co-amoxiclav 1.2g tds IV and clarithromycin 500mg bd IV (add levofloxacin if Legionella strongly suspected)	Benzylpenicillin 1.2g qds IV and either levofloxacin 500mg bd IV or Ciprofloxacin 400mg bd IV or Cefuroxime 1.5mg tds IV cefotaxime 1g tds IV /ceftriaxone 2g od IV (add levofloxacin if Legionella strongly suspected)

Length of treatment

There is no evidence to guide treatment length, but consensus suggests

- 7 days --- non-severe, uncomplicated pneumonia
- 7-10 days --- severe, microbiologically undefined pneumonia
- 14-21 days --- if Legionella, staphylococcal disease, or Gram-negative enteric bacteria suspected

PREVENTION

- Influenza vaccination:

- Pneumococcal vaccination

VACCINATION

Influenza vaccination

This reduces hospital deaths from pneumonia and influenza by about 65% and respiratory deaths by 45%

Recommended for high-risk individuals

- Chronic lung disease
- Cardiac, renal, and liver disease
- Diabetes
- Immunosuppression
- Those aged over 65
- Long- stay residential care
- Health care workers

Pneumococcal vaccination

Recommended for;

- Those aged over 65
- Chronic Cardiac, renal, and liver disease
- Diabetes
- Immunodeficiency or immunosuppression (due to disease including HIV infection or drugs)

REFERENCE

1. *Oxford Handbook of General Practice, 4th Edition,*
2. *John M URTA GH 's Handbook of General Practice, 6th Edition*
3. *Oxford Handbook of Clinical Medicine*

BRONCHIECTASIS

Chronic inflammation of the bronchi and bronchioles leading to permanent dilatation and thinning of these airways.

Main organisms; *H. influenzae*; *Strep. pneumoniae*; *Staph. aureus*; *Pseudomonas aeruginosa*.

CAUSES

- Congenital
 - Cystic fibrosis
 - Kartagener syndrome
- Post-infection
 - TB
 - pertussis
 - measles
 - pneumonia
- Other Bronchial obstruction
- Aspergillosis
- Hypogammaglobulinaemia
- Gastric aspiration

CLINICAL FEATURES

Symptoms; Persistent cough, copious purulent sputum, intermittent haemoptysis

Signs; finger clubbing, coarse inspiratory crepitations, wheeze

Complications; pneumonia, pleural effusion, pneumothorax, haemoptysis, cerebral abscess, amyloidosis

INVESTIGATIONS

- CXR
- Sputum-Microscopic, C&S
- Spirometry- often shows an obstructive pattern
- Bronchoscopy to locate site of haemoptysis; exclude obstruction and obtain samples for culture.

MANAGEMENT

- **Airway clearance techniques and mucolytics.** physiotherapy (to aid the sputum expectoration and mucus drain.
- Antibiotics
- Bronchodilators
- Corticosteroids (e.g., prednisolone) and itraconazole for ABPA
- vaccination (influenza and pneumococcal)
- Surgery.

PLEURAL EFFUSION

DEFINITION

A pleural effusion is fluid in the pleural space. Effusion can be divided by their protein concentration into transudates (25g/L) and exudates (35g/L). The accumulation of frank pus is termed empyema, that of blood is haemothorax and that of chyle is chylothorax. Both blood and air in the pleural space is called a haemopneumothorax.

In general, pleural fluid accumulates as a result of either increased hydrostatic pressure or decreased osmotic pressure ('transudative effusion', as seen in cardiac, liver or renal failure) or from increased microvascular pressure due to disease of the pleural space itself or injury in the adjacent lung (exudative effusion).

KEY POINTS

- normal pleural space has 10-20ml fluid
- can be detected on X-ray if > 300ml fluid in pleural space.
- can be detected clinically if > 500 ml fluid
- can be sub pulmonary-simulates a raised diaphragm.
- may be asymptomatic
- dyspnea common with large effusion
- chest pain in setting of pleuritis, infection or trauma
- Signs
 - Pleural effusion >500ml
 - Trachea - towards opposite side (if massive)
 - Chest wall movement -decreased (unilateral)
 - Percussion note - stony dull
 - Breath sounds - absent or decreased
 - Vocal fremitus - absent or decreased
 - Adventitious sounds (crackles, wheeze, pleural rubs and stridor) -none
 - The fluid may be transudate or exudates (diagnosed by aspirate)
 - If blood stained - malignancy, pulmonary infarction, TB

TRANSUDATE

- May be due to increase venous pressure (cardiac failure; Constrictive pericarditis, fluid overload) or Hypoproteinaemia (cirrhosis, nephrotic syndrome malabsorption),
- Also occur in hypothyroidism and Meigs' syndrome, Ovarian tumour- right-sided effusion (Meig's syndrome (right pleural effusion and ovarian fibroma)

EXUDATE

- Are mostly due to increased leakiness of pleural capillaries secondary to infection, inflammation, or malignancy.

CAUSES

- Infection
 - bacterial pneumonia
 - Pleurisy
 - Empyema
 - TB
 - viral
- Malignancy
 - Bronchogenic carcinoma
- Pulmonary infarct
- Connective tissue diseases
 - SLE
 - RA
- Acute pancreatitis
- Lymphoma
- Sarcoidosis
- HIV with parasitic pneumonia

TESTS

- CXR:
- Ultrasound
- Diagnostic aspiration:
- Pleural biopsy

MANAGEMENT

Treat the underlying cause.

- Drainage
- Pleurodesis
- Intra-pleural alteplase and dornase alfa
- Surgery

REFER

- For drainage if symptomatic. Repeated drainage ± pleurodesis may be necessary

PNEUMOTHORAX

DEFINITION

Pneumothorax is the present of air in the pleural space which can occur spontaneously or result from iatrogenic injury or trauma to the lung or chest wall.

Primary spontaneous pneumothorax occurs in patients with no history of lung disease in whom smoking, tall stature and the present of apical subpleural blebs are additional risk factors.

CLASSIFICATION OF PNEUMOTHORAX

SPONTANEOUS

PRIMARY

- No evidence of overt lung disease. Air escape from the lung into the pleural space through rupture of a small subpleural emphysematous bullae or pleural bleb, or the pulmonary end of a pleural adhesion.

SECONDARY

- Underlying lung disease, most commonly COPD and TB; also seen in asthma, lung abscess, pulmonary infarcts, bronchogenic carcinoma, all forms of fibrotic and cystic lung disease.

TRAUMATIC

- Iatrogenic (e.g., following thoracic surgery or biopsy) or chest wall injury.

TYPES OF SPONTANEOUS PNEUMOTHORAX

- Closed type
- Open type
- Tension (valvular) type

CLINICAL FEATURE

- sudden onset unilateral pleuritic chest pain
- breathlessness
- pallor ± or tachycardia

PHYSICAL EXAMINATION

- normal in small pneumothorax,
- tracheal shift to opposite side
- hyper-resonance on percussion and obliteration of cardiac dullness
- decreased or absent breath sound in large pneumothorax

INVESTIGATION

- Chest X-ray

MANAGEMENT

- Primary pneumothorax in which lung edge is less than 2 cm from the chest wall and the patient is not breathless normally resolves without intervention. 50% collapse takes 40 days to be resolved.
- In young patients presenting with a moderate or large spontaneous pneumothorax, acute respiratory distress, chest pain, percutaneous (16-l 8G cannula through the 2nd intercostal space just above the 3rd rib at mid clavicular line) aspiration of air is a simple and well-tolerated alternative to intercostal tube drainage, with a 60-80% chance of avoiding the needle for a chest drain.
- When needed, intercostal drains are inserted in the 4th' 5th or 6th intercostal space in the mid-axillary line connected to an underwater seal or one-way Heimlich valve and secured firmly to the chest wall or refer depending upon clinical judgement.
- Smoking cessation reduces risk of recurrence.

REFER

- Tension pneumothorax is the medical emergency. It was indicated/categoraized as urgent referral.

REFERENCE

1. *Davison's Principles and Practice of Medicine, 22nd Edition*

PULMONARY EMBOLISM

DEFINITION

Venous thrombi, usually from a deep vein thrombosis in the leg pass into the pulmonary circulation and block blood flow to the lungs. The source is often occult. Always suspect pulmonary embolism in sudden collapse 1-2 weeks after surgery. Without treatment, 20% with proximal deep vein thrombosis develop pulmonary embolus (PE).

RISK FACTORS

- Immobility: long flight or bus journey, post-op, plaster cast, bed-ridden
- Smoking
- Surgery –especially pelvic and lower limb
- Pregnancy or puerperium, OCP, HRT.
- Malignancy, Myeloproliferative disorder
- Past history or family history of DVT, PE, or clotting tendency

SYMPTOMS

- Acute dyspnoea
- Pleuritic chest pain
- Haemoptysis
- Syncope
- Large clots can be rapidly fatal

SIGNS

- Hypotension
- Tachycardia, gallop rhythm
- Cyanosis, loud P2, right ventricular heave
- Increased JVP
- Tachypnoea
- Pleural rub

Look for a source of emboli - although DVT may not be clinically obvious, have a high level of suspicion. Patients may have minimal symptoms/ signs apart from some pleuritic pain and dyspnoea. PE in the community can be associated with surgical procedures done 2-3wk previously.

DIFFERENTIAL DIAGNOSIS

- Pneumonia and pleurisy
- Acute coronary syndrome
- Other causes of acute breathlessness- acute LVF, asthma, exacerbation of COPD, pneumothorax, shock (e.g., due to anaphylaxis), arrhythmia, hyperventilation
- Other causes of acute chest pain - aortic dissection, rib fracture, musculoskeletal chest pain, pericarditis, oesophageal spasm, shingles

INVESTIGATION

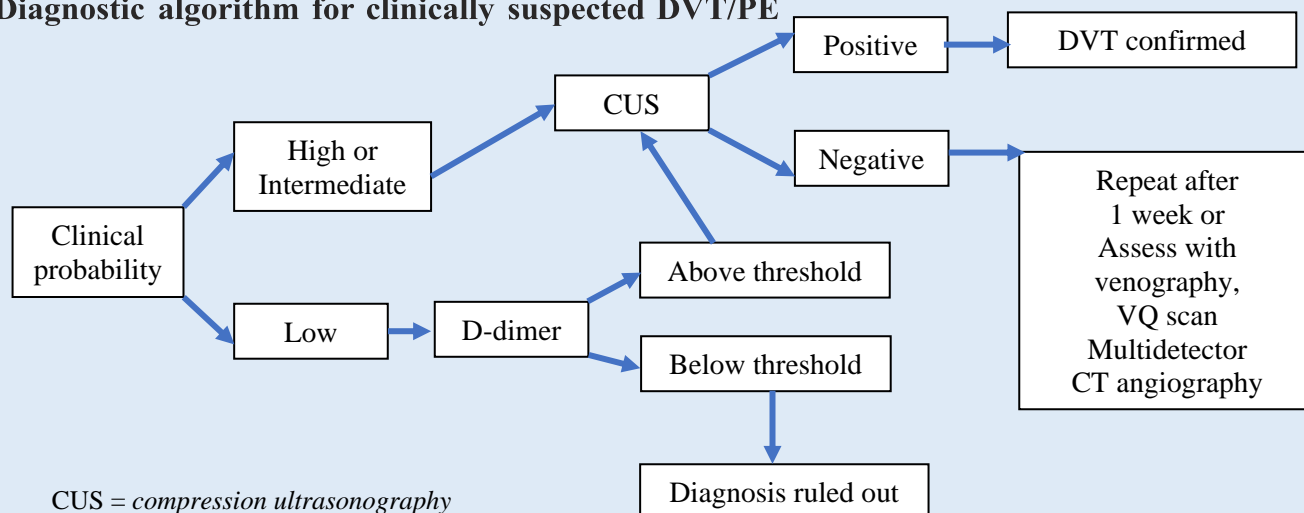
- Chest X-ray and ECG
- U&E FBC, baseline clotting
- ABG
- Serum D –dimer
- CT Pulmonary angiogram.

Well Score for DVT

Cancer	+1
Paralysis or recent plaster cast	+1
Bed rest >3 days or surgery <4 weeks	+1
Pain on palpation of deep veins	+1
Swelling of entire leg	+1
Diameter difference on affected calf >3cm	+1
Pitting oedema (affected side only)	+1
Dilated superficial vein (affected side)	+1
Alternative diagnosis at least as probable as DVT	-2

(0) Low risk, (1-2) Intermediate risk, (3) High risk

Diagnostic algorithm for clinically suspected DVT/PE



MANAGEMENT

IMMEDIATE ACTION

Most of the pulmonary embolism deaths occur within 1 hour.

- It is an acute medical emergency.
- Give i.v. access for haemodynamic instability patient before refer to hospital.
- If suspected, give oxygen as soon as possible (aim to keep SpO₂ at 94-98%).
- Needs supportive medical care and anti-coagulation
- Initial anticoagulation with LMWH and warfarin, then followed by oral anticoagulant
- LMWH should be continued for at least 4 day and anti INR is in therapeutic range for 2:2 day. Target INR 2.5 (Range 2-3)

- Oral anticoagulants reduce risk of further thromboembolism and should be continued for 3-6 months after a single DVT
- If a patient has a DVT and there is no obvious cause
 - If <45 years, consider thrombophilia
 - If >45 years, consider undiagnosed cancer

FURTHER MANAGEMENT

- In all cases of proven PE, anticoagulation is started in hospital before discharge to general practice.
- Warfarin should be continued for 2:3 months. Aim to keep the INR 2.5 (range 2-3)
- Malignancy: continue treatment with LMWH for 6 months or until cure of cancer
- Pregnancy: LMWH is continued until delivery or end of pregnancy

REFER

- All suspected DVT/PE cases must be referred to hospital (urgently).

REFERENCE:

1. *Oxford Handbook of General Practice, 4th Edition*
2. *John MURTAGH's Handbook of General Practice, 5th Edition*

RESPIRATORY FAILURE

The term respiratory failure is used when pulmonary gas exchange fails to maintain normal arterial oxygen and carbon dioxide levels. Respiratory failure occurs when gas exchange is inadequate, result in hypoxia. It is defined as a $\text{PaO}_2 < 8\text{kPa}$ and subdivided into two types according to PaCO_2 LEVEL. It is classified into Type I and Type II depending on the absence or presence of hypercapnia (raised PaCO_2)

TYPE I RESPIRATORY FAILURE

Defined as hypoxia ($\text{PaO}_2 < 8\text{kPa}$) with normal or **low PaCO_2** . It is caused primarily by ventilation/perfusion mismatch, hypoventilation, abnormal diffusion, right to left cardiac shunt. Examples of ventilation/perfusion mismatch;

- Pneumonia
- Pulmonary oedema
- PE
- Asthma
- Emphysema
- Pulmonary fibrosis
- ARDS

TYPE II RESPIRATORY FAILURE

Defined as hypoxia ($\text{PaO}_2 < 8\text{kPa}$) with hypercapnia (**$\text{PaCO}_2 > 6.0\text{kPa}$**) This is caused by alveolar hypoventilation, with or without ventilation/perfusion mismatch.

Cause includes;

- Pulmonary disease: Asthma, COPD, pneumonia, end-stage pulmonary fibrosis, obstructive sleep apnoea
- Reduced respiratory drive: Sedative drugs. CNS tumour or trauma
- Neuromuscular disease: cervical cord lesion, diaphragmatic paralysis, poliomyelitis, myasthenia gravis, Gullian-Barre' syndrome
- Thoracic wall disease: Flail chest, kyphoscoliosis

BLOOD GAS ABNORMALITIES IN RESPIRATORY FAILURE

Type I			Type II	
Hypoxia [$\text{PaO}_2 < 8.0 \text{ kPa}$ (60mmHg), Normal or low $\text{PaCO}_2 (< 6.6 \text{ kPa}$ (50mmHg)]			Hypoxia [$\text{PaO}_2 < 8.0 \text{ kPa}$ (60mmHg) Raised $\text{PaCO}_2 (> 6.6 \text{ kPa}$ (50mmHg)]	
	Acute	Chronic	Acute	Chronic
H+	-7 or j	-7	i	-7 or j
Bicarbonate	-7	-7	-7	i
Causes	<ul style="list-style-type: none"> • Acute asthma • Pulmonary oedema • Pneumonia • Lobar collapse • Pneumothorax • Pulmonary embolus • ARDS 	<ul style="list-style-type: none"> • Emphysema • Lung fibrosis • Lymphangitis carcinomatosa • Right-to-left shunts • Brain-stems lesion 	<ul style="list-style-type: none"> • Acute severe asthma • Acute exacerbation COPD • Upper airway obstruction • Acute neuropathies/paralysis • Narcotic drugs • Primary alveolar hypoventilation • Flail chest injury 	<ul style="list-style-type: none"> • COPD • Sleep apnoea • Kyphoscoliosis • Myopathies/muscular dystrophy • Ankylosing spondylitis

CLINICAL FEATURES

Are those of underlying cause together with symptoms and signs of hypoxia, with or without hypercapnia

Hypoxia – dyspnea, restlessness, agitation, confusion, central cyanosis. If long standing hypoxia; polycythemia, pulmonary hypertension, cor-pulmonale

Hypercapnaea –headache, peripheral vasodilatation, tachycardia, bounding pulse, tremor/flap, papilloedema, confusion drowsiness, coma

INVESTIGATIONS

are aim at determining the underlying cause

- Blood test –FBC, U&E, CRP, ABG
- Radiology –CXR
- Microbiology - sputum and blood culture
- Spirometry (COPD, neuromuscular disease, Guillian-Barre' syndrome)

MANAGEMENT – DEPEND ON CAUSES.

MANAGEMENT OF ACUTE RESPIRATORY FAILURE

Prompt diagnosis and management of the underlying cause is crucial to the management.

In type I respiratory failure, high concentrations of oxygen (40-60% by mask) will usually relieved

hypoxia.

Acute type II respiratory failure is an emergency, which requires immediate interventions. The most common cause of chronic type II respiratory failure is severe COPD.

Assessment and management of 'acute on chronic' type II respiratory failure
Initial assessment
Patient may not appear distressed in spite of being critically ill <ul style="list-style-type: none">• Conscious level (respond to commands, ability to cough)• CO₂ retention (warm periphery, bounding pulses, flapping tremor)• Airways obstruction (wheeze, prolong expiration, hyperinflation, intercostal indrawing, pursed lips)• Cor-pulmonale (peripheral oedema, raised JVP, hepatomegaly, ascites)• Background functional status and quality of life• Signs of precipitating causes.
Investigations
<ul style="list-style-type: none">• Arterial blood gas (severity of hypoxaemia, hypercapnia, acidaemia, bicarbonate)• Chest X-ray
Management
<ul style="list-style-type: none">• Maintenance of airway• Treatment of specific precipitating cause• Frequent physiotherapy+ /- pharyngeal suction• Nebulized bronchodilators• Controlled oxygen therapy Start with 24% Venturi mask <p>Aim for a PaO₂ >7kPa (52mmHg) (a PaO₂ <5 (37mmHg) is dangerous)</p> <ul style="list-style-type: none">• Antibiotics• diuretics
Progress
<ul style="list-style-type: none">• If PaCO₂ contributes to rise or patient cannot achieve a safe PaO₂ without severe hypercapnia and acidaemia, mechanical ventilator support may be required.

LUNG CANCER

Lung cancer is the most common cancer.

Incidence increased with age-85% are aged >65 years and 1% <40 years at presentation.

M : F = 2:1 but incidence is increasing in women.

RISK FACTORS

- Cigarette smoking (causes 90% of lung cancer)
- Other passive smoking
- Asbestos, chromium, arsenic, iron oxides and radiation

HISTOLOGY

Clinically the most important division is small cell and non-small cell lung cancer.

Squamous cell (35%); adenocarcinoma (27%) large cell (10%); adenocarcinoma in situ (rare 1%);

Small cell (oat cell) (20%).

Most 70% of small cell lung cancer are disseminated at the time of presentation.

CLINICAL FEATURES

SYMPTOMS:

- Cough (80%)
- Haemoptysis (70%)
- Dyspnea (60%)
- Chest pain (40%)
- Recurrent or slowing resolving pneumonia, lethargy, anorexia, weight loss.

SIGNS

- Cachexia, anaemia, clubbing, HOPA (causing wrist pain), supraclavicular or axillary nodes
- Chest signs: none or consolidation, collapse and pleural effusion
- Metastases: bone tenderness, hepatomegaly, confusion, fits, focal CNS signs, cerebellar syndrome, proximal myopathy, peripheral neuropathy.

COMPLICATIONS

- Local: recurrent laryngeal nerve palsy, phrenic nerve palsy, SVC obstruction, Horner's syndrome, (Pancoast's tumour), rib erosion. Pericarditis, AF.
- Metastatic: brain, bone, liver, adrenal (Adison)
- Non-metastatic neurological: confusion, fits, cerebellar syndrome, proximal myopathy, neuropathy, polymyositis
- Most present between 50 and 70 years (mean 67 years)
- Only 10-25% asymptomatic at time of diagnosis
- If symptomatic - usually advanced and not resectable

TESTS

- CXR;
- Cytology
- Fine needle aspiration or biopsy
- Bronchoscopy
- Radio-nuclide bone scan

PRESENTATION

>90% have symptoms at the time of diagnosis. Common presenting features:

- Cough (56%)
- Chest/shoulder pain (37%)
- Haemoptysis (7%)
- Dyspnoea
- Hoarseness
- Weight decrease
- Finger clubbing
- General malaise
- Distant metastases
- Incidental finding on CXR

REFERRAL FOR SUSPECTED LUNG CANCER IMMEDIATE REFERRAL/ ACUTE ADMISSION

- Stridor
- Superior vena cava obstruction (swelling & congestion of face/neck with fixed increase JVP)

URGENT REFERRAL

- Persistent haemoptysis (in smokers/ex-smokers aged 2:40 years)
- CXR suggestive of lung cancer (including pleural effusion and slowly resolving consolidation)
- May be normal CXR where there is high suspicion of lung cancer
- History of asbestos exposure and recent onset of chest pain, shortness of breath, or unexplained systemic symptoms where a CXR indicates pleural effusion, pleural mass, or any suspicious lung pathology

URGENT REFERRAL FOR CXR

- Haemoptysis
- Any of the following if unexplained or present for >3 weeks,
 - Cough
 - Chest/shoulder pain
 - Weight loss
 - Hoarseness of voice
 - Cervical or supraclavicular lymphadenopathy
 - Features suggestive of metastases from a lung cancer, e.g., secondaries in the brains, bone, liver and skin
 - Dyspnoea
 - Chest signs
 - Finger clubbing

- But do not delay for 3 weeks if high suspicious of lung cancer
 - smoker/ex-smoker
 - COPD
 - History of asbestos exposure
 - Previous history of cancer (especially head and neck cancer)

PREVENTION

- **Smoking cessation:** 90% of lung cancer patients are smokers or ex-smokers. The younger a person is when he/she starts smoking the greater the risk of developing lung cancer. Risk also increased with amount smoked (duration of smoking and number of cigarettes smoked/day)
- **Diet:** increase consumption of fruit, carrots, and green vegetables may decrease incidence, but there is no evidence that vitamin supplements are beneficial and they might be harmful.

PANCOAST SYNDROME

- Apical lung cancer + ipsilateral Homer's syndrome.
- *Cause:* invasion of the cervical sympathetic plexus.
- *Other features:* shoulder and arm pain (brachial plexus invasion C8-T2) ± hoarse voice/bovine cough (unilateral recurrent laryngeal nerve palsy and vocal cord paralysis).

PARANEOPLASTIC SYNDROMES

- (E.g., ectopic ACTH production, SIADH, hypercalcaemia, hypercoagulability) Affect 10- 20% of patients with lung cancer-particularly small cell. Have a high index of suspicion and refer for specialist management if suspected.

MANAGEMENT

- Once the diagnosis has been confirmed, or suspected, refer to chest physician. Active treatment options depend on type and extent of tumour and include surgery, radiotherapy, and/or chemotherapy.
- Follow-up regularly. 80% die in < 1yr.

PALLIATIVE RADIOTHERAPY

Radiotherapy is a key component of symptomatic treatment for:

- Haemoptysis
- Chest pain
- Breathlessness due to bronchial occlusion
- Pain from bone metastasis
- Symptoms from brain metastasis

Radiotherapy may be combined with palliative chemotherapy, particularly for patients with non-small cell lung cancer.

REFERENCE

1. *Oxford Handbook of General Practice 4th Edition*
2. *Oxford Handbook of Clinical Medicine*