Drugs of cough:

Antitussive drugs:

Drugs Alter mucoceliary factors

*Ipecachuanha and squill: cause emesis
*volatile oils:direct effect on bronchi
*iodinated glycerol:excreted thrugh bronchial glands,contraindicated in pregnancy and lactation
*aromatic chest rub
*brohmexine :increase lysosome activity
*carbocisteine:irritation
*H1histamine antagonist +decongestant
*ammonium chloride
*Hydration
*Ipratropium bromide.
*Beta adrenergic agonists.
* Theophylline.
*Sodium chromoglycate.
* Beclomethasone.

Drugs act on afferent limb:

Local anesthetics:lodocaine

Opioids

Drugs act on cough center:

Narcotic:

Codiene
Morphine
Dimorphine

Non narcotic:

Dextromethorphan
Glaucine
Diphenhydramine
Pholcodine

Drugs act on efferent limb:

Ipratrpium promide (+alter mucoceliary factor),effective for asthma ,chronic bronchitis,persistent cough

Drugs act on respiratory muscles:

Nondepolarizing blockers like pancuronium for pateints can't be mechanically ventilated

Proteusive drugs:

Hypertonic saline aerosol(increase efficacy of cough)
Amiloride aerosol(cystic fibrosis)
Bronchodilator:cause decrease flow rate

New treatments:

Opioids : New opioid peptides such as the endomorphins bind to the opioid receptor- like 1 receptor (ORL1)

Drugs treat inflammatory process that cause cough:

Anti inflammatory drugs

Drugs of asthma and COPD

novel proton pump inhibitors as treatment for gastro- oesophageal reflux

compounds that are targeted to inhibit sensory nerve activity directly which should, in theory, inhibit cough of any aetiology

Drugs of tuberculosis

1st line therapy:

Izoniazid

Most active,related to pyrodoxine,prodrug activated by KATG and peroxidase,bactericidal effect by inhibition of mycolic acid synthesis

Ready absorbed ,widely distributed, metabolised by strong and weak acytalators

Adverse effects:

*hepatitis1%

*neuropathy10-20%: due to DM,Aids,alcoholism,uraemia,malnutrition,pyrdoxine deficiency ex: psychosis,seizures,memory loss,memory loss

*GIT,tinnitis mhematologic interactions

Rifampin:

Bactericidal (Mycobacteria,enterococci, chlamydia)

Inhibit RNA synthesis (by binding to B subunit of bacterial RNA polymerase

Hepatic metabolism

Using in Tb,leprosy,,meningococcal carrier states,staph osteomyelitis (inflammation of the bone arm and leg in children,spine ,hip, feet in adults), valve endocarditis(inflammation in the inner layer of the heart and valves

Side effects:

Orange Color to secretions,hepatitis,Flue like syndrome,lowering serum levels of other drugs

Streptomycin:

Usings: Tuleremia ,plaque, Brucellosis ,endocarditis

Side effects:vestibular toxicity (irreversible),rash,fever,pain (IM injection),

2nd line therapy:(we use them in case of resistance or toxicity and side effects from 1st line)

Ethionamide:

Moa:like isoniazid,block mycolic acid synthesis,poorly tolerated,orally,good destribution

Side effects:neurotoxicity,nephrotoxicity,GIT irritation

Capreomycin:

Moa:inhibit peptide protein synthesis

Side effects:nephrotoxicity,ototoxicity,local pain,sterile abscesses

Cycloserine :

Moa:inhibit cell wall synthesis
Side effects:neuropathy,CNs toxicity
Para Amino- Salicylic-Acid (PAS):
Moa:inhibit folate synthesis,good distribution except CNS and absorption
Side effects:hypersensitivity, Crystalluria,GI toxicity
Amikacin :
Multidrug resistant strains,atypical mycobacteria
Fluroquinolones :
Resistance develops rapidly if used alone
Linezolid:last resort
Side effects: Bone marrow suppression,Irreversible peripheral and optic neuropathy

Rifabutin,Rifapentine
Like rifampin inhibit bacterial RNA polymerase
Rifabutin less potent and use in HIV patients recieiving protease inhibitors or non nucleoside reverse transcriptase inhibitor (e.g. efavirenz)
Nontuberculosis mycobacteria:
10% of laboratory isolates
Desinictive laboratory characteristics.
Less susceptible to drugs
M.tuberculosis:
Sulfonamide
Erythromycin
Tetracycline
M.avium:common cause of TB in late stage of AIDS
Azithromycin,Clarithromycin, Ethambutal, Ciprofloxacin

TB depend on resistance:
Mono resistant TB:resistant to any drug
Poly resistant TB:resistant to at least 2drugs but not isoniazid and rifampin
Multi drug resistant TB:at least resistant to isoniazid and rifampin
Extensively drug resistant TB: resistant to isoniazid and rifampin and flouroquinilone,and at least one of 2nd line drugs

**Number of days to treat resistant TB 730 days

Antiviral agents

Viruses:intacellular organisms use host cell mechanisms to produce viral particles composed of nuclei acidDNa orRNA (core)and protein coat (capsid)

DNA viruses
adenoviruses (colds, conjunctivitis)
hepadnaviruses (hepatitis B);
herpesviruses (cytomegalovirus chickenpox)
papillomaviruses (warts)

RNA viruses
arborviruses (yellow fever)
arenaviruses (meningitis);
orthomyxoviruses(influenza);
paramyxoviruses (measles, mumps);
picornaviruses (meningitis, colds);
rubella virus (German measles)
retroviruses (AIDS)

ANTIHERPESVIRUS AGENTS:

Acyclovir:

Available as oral tablets, IV injections, eye drops and ointment, or as a cream.
Restricted in varicella (chicken pox)in immunocompramised pateints
Side effects:N,V,rash
MOA:conversion to acyclovir triphosphate leading to DNA chain termination and formation of inactive complex with viral DNA polymerase

Anti influenza Agents:

Amantadine:symmmetril ,
synthetic tricyclic amine
Rimantadine:flumadine;
methyl derivative
Both inhibit M2protein (membrane acts as H channel) prevent acid dissociation of the ribonucleoprotein complex ,the pH changes ,inhibit viral assembly

Anti HIV Agents:

Zidovudine:expensive
MOA:inhibit DNA production
Side effects:N,V,muscle pain,BM suppression
Indinavir:expensive
MOA:protease inhibitor: HIV-1 protease is an enzyme required for the proteolytic cleavage of the viral polyproteint precursors into the individual functional proteins found in infectious HIV-1. Indinavir binds to the protease active site and inhibits the activity of the enzyme
Side effects:N,V,diarrhea,renal stones
Short half life ,dosing each 8 hours to prevent HIV from making drug mutations and resistance
Interferons :
antiviral, immunomodulating, and antiproliferative activities
Produced normally from viral infected cell,and from donor WBCs,by recombinant DNA technology now
Usings:hepatitis c,leukemias
Side effects:N,fever,malaise(flue like symptoms)
MOA: activate the JAK STAT signal transduction pathway leads to synthesis of over two dozen proteins that contribute to viral resistance mediated at different stages of viral penetration

Drugs of Asthma

Asthma:inflammation with intermittent narrowing of airways,resistance to flow,chronic condition,

Treatment; individualized,goals of therapy:no acute episodes,no need to B agonist inhaler,no limitation of activities,no adverse effects of drugs
Risk of Not Treating Asthma:increase mortality,decrease lung function,increase number of asthma attacks
Early asthma: prevented by bronchodilator
Allergen provoke IGE,in the next epoxure produce Ag-Ab interaction leading to release mediators
Late asthma:prevented by corticosteroid
4-5hours later,more bronchoconstriction,influx of inflammatory cells, secrete interleukins5,9,13 by TH2 lymphocytes,IgE ,and mucus production

Asthma triggers:

Exercise , cold air!Cigarette smoke,Stress,anxiety situations!Animal dander's Allergens (grass, trees, molds, cockroach) Pollutants (sulfur dioxide, ozone) ,Fumes/toxic substances
Medications (ASA, NSAID's)

Diagnosis:

Cough after exposure to air cold,upper respiratory infection,exercise,allergic
Past history of bronchiolitis
Family history of asthma
Decrease PEFR (peak expiratory flow rate)
Decrease FEFR(forced expiratory flow rate)
Reversible with bronchodilator
Highly responsive to methylcholine
Increase in expired NO
Increase in inflammatory mediators

Myths and Misconceptions:

Asthma is an emotional illness.
Asthma is an acute disease.
Asthma medications are addictive.
Asthma medications become ineffective if they are used regularly.
Asthma is not a fatal illness.

Drugs:

Quick:
Short acting B2 agonists
Systemic Corticosteroids
Anticholenergics
Long term:
Inhaled Typical Corticosteroids
Inhaled Cromolyn Na,and nedocromil
Oral Methylxanthines (Theophyllines)
LABA:long acting B2 agonist
Oral leukotreine modifiers