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Content

From the Chairperson and Secretary of the Chapter

Dear Fellow Academicians,

Best wishes from the new executive committee of IAP Respiratory Chapter 2009-2010 and Greetings from Kolkata.

For the first time the Eastern Zone has been given the responsibility of running the central IAP Respiratory Chapter activities. We all need your blessings, advice patronage and active support for these two years (2009-2010).

Website – We have already launched the website **"iaprespiratory.org".** Please go through it and send your comments and suggestions regarding ita upgradation.

Scientific programs – We have already organised these scientific programs in this year 2009, namely, CME in Karnataka (8 March; 2-3 May), RTI GEMS (13 March, Kolkata and 15 March, Karnataka), Workshop in Bronchoscopy (25-26 April, Bangalore), Asthma Day celebration (2 May, New Delhi and 17 May, Chennai) and many others. The RESPICON 2009 will be held at Kottayam, Kerela on 11 to 13 September 2009. Dr T U Sukumaran is its Organising Chairperson. The First program of AWESOME RESPIROLOGY will be held at Kolkata on 31 May. We hope to have newer modules in different aspects of respirology (ATM, RTI) in near future.

Membership strength – The membership strength has been close to 1300 uptil now. We request our members to go through the membership list and check their addresses, phone nos etc. and to definitely send the IAP Membership No and email id. If the name of any member does not appear in the list he/she is requested to inform us with all documents.

The journal – We are going to publish the first issue (Resumed Publication) of *"Pediatric Pulmonology Update"* , the journal of IAP Respiratory Chapter on pneumonia. The format is question answer type. We are grateful to have articles contributed by academicians from all over India. Our team of editors with Dr Gautam Ghosh and Dr Atul Kr Gupta as chief editors; Dr K K Ghosh and Dr Jaydeep Choudhury as the executive editors; Dr Subhasis Roy as managing editor along with experienced Dr Tapan Kr Ghosh as editor emeritus have put their efforts to give the resumed edition a new shape. We must thank Mr Somnath Mukherjee for kindly making it to see the light of the day. Please feel free to comment, criticize and bless this inaugural issue.

We are proud to have this first issue during the AWESOME RESPIROLOGY CME in Kolkata on 31 May 2009. This publication of the Scientific Journal will continue longer.

Greetings from the team of Kolkata.

Gautam Ghosh, *Chairperson*
K K Ghosh, *Secretary*

Editorial

Pneumonia in Under-5 Children

Pneumonia is the leading single cause of mortality in under-5 children (0.29 episode per child year in developing and 0.05 episode per child year in developed countries). Of the total 156 million new episodes each year worldwide 151 million episodes occur in the developing countries. Most number of cases (43 million) take place in India. Of all community cases 7 to 13% are severe enough to be life threatening and require hospitalization. Pneumonia is responsible for about 19% of all deaths in children aged less than 5 years[1].

All these data give enough evidence about the severity of this problem in under 5 child population. The renewal interest in reduction of childhood mortality came from Millennium Declaration and the Millennium Development Goal 4 (MDG4), which states and stresses that two-third reduction of under 5 mortality rate is to be achieved between 1990 and 2015[1].

About 2 million pneumonia deaths occur in each year in under 5 children all over the world, the major incidences are in South-East Asian and African Regions. Estimates of clinical pneumonia is highest in South East Asian Region (0.36 episodes per child year). It is already informed that highest number of cases are in India (43 million new cases with estimated incidence 0.37) followed by, China (21.1 million new cases with estimated incidence 0.22) and Pakistan (9.8 million new cases with estimated incidence 0.41)[2].

The main bacterial causes of childhood clinical pneumonia in under-5 children in the developing countries are *S pneumonie* and *H influenze* type b and the main viral cause is respiratory syncitial virus (RSV). Effective RSV vaccine is still not available. All RSV vaccines are in stage of candidates and we have to wait, may be another decade to have an effective vaccine against RSV. Two bacterial vaccines are important – Hib conjugate vaccine and 7-valent pneumococcal conjugate vaccine to prevent the incidence of pneumonia in under 5 children. The 2nd vaccine is very costly and to make it available to majority of children the cost is to be subsidized with the help of GAVI. Already it is a news that GAVI will come forward to extend their helpful hands for pneumococcal vaccine in India. This will solve a great problem in this developing country to reduce the number of under 5 mortality to a significant level. At the same time we have to get more valent pneumococcal vaccine like 9-valent, 10-valent, 13-valent to have more effective role.

IAP Respiratory Chapter welcomes this decision of GAVI support, which is a real step forward to reduce under 5 mortality rate in India.

Tapan Kr Ghosh, Gautam Ghosh

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Community Acquired Pneumonia

Varinder Singh*, Susil Kabra**

**Professor, Pedaitric Medicine, Lady Hardinge Medical College and Kalavati Saran Hospital for Children, New Delhi*
*** Professor, Pediatric Medicine, All India Institute of Medical Sciences, New Delhi*

How should one define CAP in a clinician's office ?

Community acquired pneumonia is an acute infection of the pulmonary parenchyma in a previously healthy child, acquired outside of a hospital setting. The patient should not have been hospitalized within 14 days prior to the onset of symptoms or has been hospitalized less than 4 days prior to onset of symptoms[1].

What is the magnitude of the problem in India ? How frequently ARI may lead to CAP ?

Recent estimate from WHO[2] suggest the median incidence of clinical pneumonia is 0.28 episodes per child-year. This equates to an annual incidence of 150.7 million new cases, 11-20 million (7-13%) of which are severe enough to warrant hospitalization.

Recent estimates from India suggest that ARI in children under 5 years of age constitutes 24% of the National Burden of disease and 13% of deaths[3]. Community based studies from different parts of the country have reported the annual incidence of ARI as 2.5-6.42 episodes per child per year[4,5]. 0.23-12.5% of children with ARI are estimated to have LRTI. Hospital based studies have reported that 20-30% of admissions in children below 5 years of age are due to ALRTI[6,7].

What are the risk factors for CAP in our settings ?

Mortality estimates suggest that 2.3 million children less than 5 years of age die every year in India and 20% of these deaths are due to ARI.

Review of ALRTI epidemiology in developing countries have identified the following as the key host and environmental and socioeconomic factors that affect the incidence and severity of LRTI[8,9].

Host :

(1) Young age
(2) Low birth weight
(3) Malnutrition
(4) Lack of breastfeeding/ breastfeeding less than 4 months
(5) Inappropriate immunization for age
(6) Subclinical vitamin D deficiency in first 4 months

Environmental/socioeconomic factor :

(1) Crowding
(2) Family size
(3) Advanced birth order
(4) Indoor air pollution – biomass fuels (wood, agriculture waste, animal dung, manure), kerosene, coal.
(5) URTI in the family (mother, siblings)
(6) Non-allopathic treatment in early stages of illness
(7) High nitrate ingestion in drinking water

What is the incidence of viral and bacterial pneumonia ?

Evidence of a potential causative agent has

been identified in 24-85% of the cases. In a recent study from India etiological agents could be identified in 94% of the patients. Bacterial etiology was demonstrated in 16%, viruses in 38%, mycoplasma in 24% and *Chlamydia pneumonie* in 11% of the cases. Mixed infection was present in 8%[10].

Other studies from India have demonstrated the following as the proportion of the organisms in pneumonia in children 2-60m age[10-12] viruses- 38%, bacteria-62.6%, mycoplasma– 24-30%, chlamydia- 6-11%, mixed infections- 9%.

Bacterial causes:

S pneumonie–33-40%,*H influenze* b–22 %

How can one clinically suspect and differentiate a bacterial pneumonia from a viral one ?

Viral – Acute onset, other system involved (conjunctivitis, rhinorrhea, cough and wheeze), vesicles. Self-limiting (< 4-7 days) unless complicated.

Bacterial – Explosive onset, pain throat, rapid progress, headache, abdominal pain and vomiting, general malaise. Usually little coryza or mild cough rarely wheeze. May get complicated (effusion) early, needs drugs to improve.

Radiology :

Lobar/segmental, pleural involvement, no volume loss, unilateral – bacterial. Bilateral, interstitial, over-inflation (occasional), no pleural involvement - viral.

Is the IMNCI guideline of age specific RR is fool proof for diagnosis pneumonia ?

The WHO algorithm for children presenting with cough and/or difficulty in breathing proposes fast breathing as the most critical sign to identify pneumonia. The age related cutt offs to define fast breathing are:

Table 1. *Age related cut offs to define fast breathing*

Age	Respiratory rate (breath/minute)
< 2 months	60 or more
2 months up to 12 months	50 or more
12 months up to 5 years	40 or more

In addition, age is a good predictor of the likely pathogen of pneumonia and can help to narrow the list of etiological agents.

This definition identifies children who have a very high probability of having pneumonia and are therefore candidates for antibiotic therapy. Respiratory rate should ideally be counted for full 60 seconds when the child is awake and not crying. Inconsistencies in respiratory rate measurement require repeat observation. The presence of lower chest indrawing indicates pneumonia of greater severity.

A comparison of sensitivity and specificity of different clinical findings in patients with radiographically confirmed pneumonia is presented in Table 2. (Ref Ped Pneumonia *CMAJ* 1997;156(5):S704-11).

Is there any age specific guidelines for suspecting etiology of CAP ?

The age specific guideline is shown in

Table 2. *Sensitivity and specificity of various respiratory symptoms*

Study	Age range	Tachypnea		Retractions		Crackles	
		Sens	Spec	Sens	Spec	Sens	Spec
Berman *et al*	<4 month	62	63				
Leventhal *et al*	3 month-15 year	81	60	35	82	44	80
Taylor	<2 year	75	70				
Grossman *et al*	< 19 year	64	54			43	77

Sens = Sensitivity, Spec=Specificity

Table 3.

Table 3. *Age specific guidelines for suspecting etiology of community acquired pneumonia*

0-2 months	Gram negative *S pyogenes* Chlamydia Viruses
2months-5years	*S pneumonie* *H Influenze* *S pyogenes* Viruses *Mycopl pneumonie*
>5 years	*S pneumonie* Staphylococcus Viruses *Mycopl pneumonie* *S pyogenes* *H Influenzae*

Can we clinically diagnose CAP in OPD? Do we always need to do a xray to confirm CAP ?

Signs that suggest a high probability of pneumonia and need for antibiotic treatment are the following :

Children with suspected pneumonia can present with symptoms including

(i) Fever

(ii) Cough, may or may not be productive

(iii) Chest pain and/or abdominal pain

(iv) Difficulty in breathing/ Rapid breathing

(v) Constitutional symptoms: Malaise, lethargy, headache, nausea/vomiting

In this context it is also important to mention that a lot of other clinical syndromes like bronchiolitis-VAW syndromes, LTB-croup complex can also present similarly with fever, cough and tachypnea. These can probably be excluded clinically if the patient fits into any of the following definitions.

Consider bronchiolitis-VAW if :

(i) Age – 1 month to 1 year

(ii) Presence of upper respiratory catarrh

(iii) Progressive increase in respiratory distress (tachypnea, retractions)

(iv) Wheeze + crackles

(v) Clinical and radiological evidence of hyperinflation

Consider LTB-croup *if:*

(i) Hoarseness of voice and barking/ brassy cough

(ii) Stridor

(iii) Mild to marked respiratory distress

(iv) Sonorous rhonchi

(v) Fever usually mild or spiking (tracheitis, rare)

Consider asthma if:

(i) Recurrent episode, 3 or more

(ii) Wheeze

(iii) Good response to bronchodilator

(iv) Hyperinflation

(v) Family/personal history of atopy

Though there are few clinical features to suggest probable etiology yet some clues may be useful eg, presence of skin boils, rapid progression/deterioration, empyema or pneumothorax or radiological evidence of pneumatocele strongly suggests staphylococci as the cause. It is some what difficult to differentiate the viral pneumonias from bacterial pneumonias clinically.

Which children need xray as a must ?

(i) Those needing domiciliary care usually do not benefit from this.

(ii) Those sick enough to need hospitalization may benefit.

Indications for chest xray in either primary

care or hospital is shown in Table 4.

Table 4. *Indications for chest xray in either primary care or hospital*

(i)	For diagnosis of child under 5 years with fever of 39°C of unknown origin
(ii)	If complication (eg, pleural effusion) suspected
(iii)	Ambiguous features unresponsive to treatment after 48 hours of treatment / deteriorates
(iv)	For follow up of children with lobar collapse or ongoing symptoms

Do we need to repeat Xray at end treatment of all CAP ?

End of treatment chest xray not needed in any case except when the response is delayed or incomplete or there were any ambiguous signs in initial film or there are any associated complications.

Role of blood test and serology etc, in CAP ?

Routine microbiological tests are of no use. Like wise acute phase reactants like TLC, DLC, CRP are not diagnostic but may be useful to monitor the response to treatment.

Some times small effusions are picked with pneumonias. Pleural tap is indicated only if the collection is over 10 mm on USG.

Pulse oximetry is a good tool for assessing the severity and for monitoring response in those with severe disease.

Any antibiotic protocol for age related CAP ?

The antibiotic protocol for age related CAP is shown in Fig 1.

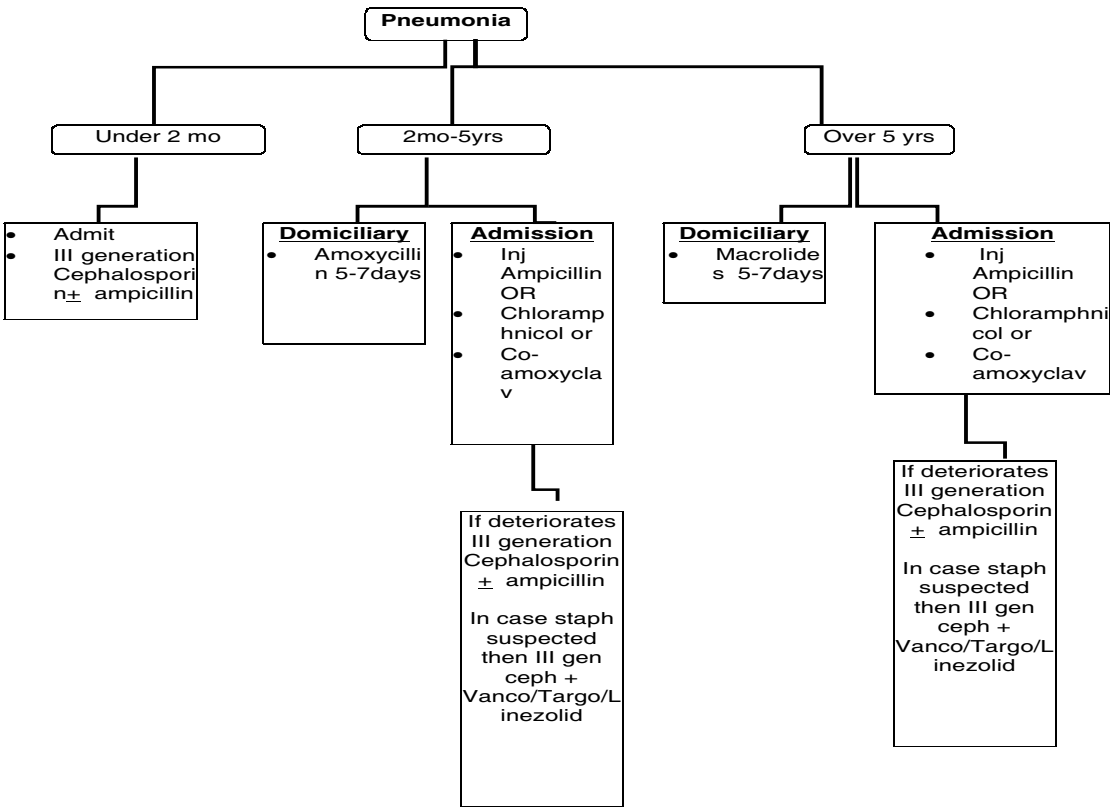

Fig 1. Antibiotic protocol for age related CAP

WHO criteria for severe CAP in infants and older children ?

WHO criteria for severe CAP in infants and older children is shown in Table 5.

Table 5. *Severity assessment of CAP in infants and older children*

	Mild	Severe
Infants	Temperature < 38.5^{0}C RR < 50 breaths/minute Mild recession Taking full feeds	Temperature > 38.5^{0}C RR > 70 breaths/minute Moderate to severe recession Nasal flaring Cyanosis Intermittent apnea Grunting respiration Not feeding
Older children	Temperature < 38.5^{0}C RR < 50 breaths/minute Mild breathlessness No vomiting	Temperature > 38.5^{0}C RR > 50 breaths/minute Severe difficulty in breathing Nasal flaring Cyanosis Grunting respiration Signs of dehydration

CAP needing admission in hospital ?

Indications for admission to hospital in infants:

(i) SaO2 < 92%, cyanosis;

(ii) Respiratory rate > 70 beats /minute;

(iii) Difficulty in breathing;

(iv) Intermittent apnea, grunting;

(v) Not feeding;

(vi) Family not able to provide appropriate observation or supervision.

Indications for admission to hospital in older children:

(i) SaO2 < 92%, cyanosis;

(ii) Respiratory rate > 50 breaths / minute;

(iii) Difficulty in breathing;

(iv) Grunting;

(v) Signs of dehydration;

(vi) Family not able to provide appropriate observation or supervision.

CAP needing PICU care in hospital?

Indications for transfer to Pediatric Intensitve care Unit (PICU)

Hypoxemia is a good indicator of the severity of pneumonia, and pulse oximetry should therefore be performed on every child deemed ill enough to be admitted. Transfer to PICU should be considered when:

(i) There is failure to maintain SaO2 >92% in FIO2 >0.6

(ii) The patient is shocked

(iii) There are rising respiratory and pulse rates with clinical symptoms

(iv) Evidence of severe respiratory distress and exhaustion with or without raised PaCO2

(v) There is recurrent apnea or slow irregular breathing.

Duration of antibiotic therapy in CAP?

(i) Domiciliary 5-7 days

(ii) Admitted, switch to oral after 48-72 hours or earlier if can accept orally. Total 5-7 days

(iii) If on second line then IV for 7-10 days

(iv) If staph 2 weeks atleast if no complication else 4-6 weeks.

Role of supportive therapy in CAP ?

1. Oxygen, use pulse oxymetry.
2. IVF if dehydrated, tachypneic , unable to drink, impending respiratory failure.
3. Fever management.
4. Bronchodilator where indicated.
5. Physiotherapy.
6. Steroids : No role.

Can clinical finding and radiology be used to point to a specific etiology in CAP ?

Clinical and radiographical clues to the etiological diagnosis of pneumonia is shown in Table 6.

Table 6. *Summary of clinical and radiographical clues to the etiological diagnosis of pneumonia*

Radiographical findings	Clinical circumstance	Organism
Segmental consolidation	Community-acquired	*S pneumonie, M pneumonie* – Lobar
consolidation	Community-acquired	*S pneumonie* (2/3 of community acquired pneumonias)
Rounded pneumonia	Community-acquired	*S pneumonie*
Bronchopneumonia	Hospital-acquired	*P aeruginosa, S aureus*, streptococci, Gram negative bacilli, anerobes, *M pneumonie*, *L pneumophila*
Interstitial pneumonia	Community-acquired (winter)	Virus, *M pneumonie*
Cavitation/necrosis	Aspiration	*S aureus*, Gram negative bacilli, anerobes, actinomycosis
Multiple cavitary nodules	Post measles, malnourished	*S aureus*
Pneumatoceles		
Empyema	Complication of pneumonia	*S pneumonie* *S aureus* Gram negative bacilli
Lymphadenopathy		*M pneumonie* *M tuberculosis*

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Book Review

Neonatal and Pediatric Respiratory Diseases

Edited by ; Dr. Praveen Khilnani
Published by : Ik International Publishing House, New Delhi.
Price : Rs 2495 /- Pages : 717.

There are only a few Indian textbooks on Pediatric Respirology. This is an unique attempt by Dr. P. Khilnani , an eminent and very senior pediatric intensivist and pulmonologist, trained and worked in India and USA. To bring out a textbook exclusively on pediatric and neonatal respirology is a tough job. His list of contributors constitute the most elite and experienced pediatric respirologists in India and abroad.

There are 53 chapters in total encompassing the common problems encountered in respiratory system in both children and neonates. There are 5 chapters exclusive on neonatal respiratory problems. Though all the problems are not met with, the commoner ones as encountered in day to day practice have been selectively chosen with proper importance. The chapters are selected as specific diagnostic topics rather than general ones.

Specific and detailed emphasis on some topics as, viral pneumonias, recent advances on rapid diagnosis of respiratory tract infection, Wegener's granuloma, sarcoidosis, ventilator graphics and clinical implications, bronchodilator therapy in mechanically ventilated, pediatric thoracoscopy, Indian experience in flexible fiberoptic bronchoscopy and pediatric heart-lung transplantation are worth mentioning as these are rarely available even in standard textbooks. Dr. Khilnanai's exhaustive experience in p ediatric intensive care has added a rich contribution in these and related segments.

Some topics as clinical evaluation of respiratory symptoms, some IMNCI topics as ARI etc. could be considered to fulfil the interest of the postgraduates and practitioners. Some algorithmic approach in management sections, wherever possible, could have been very much useful for the readers.

Overall, it is a good attempt to bring out a textbook in pediatric and neonatal respiratory system in Indian context and post graduates, residents, pediatricians and pulmonologists will be greatly benefited by this book.

Dr. Gautam Ghosh
Chairperson, IAP Respiratory Chapter
India

Nosocomial Pneumonia

Krishan Chug

Senior Consultant, Pediatric Intensive Care, Sir Gangaram Hospital, New Delhi

How do we define nosocomial infections ?

Nosocomical infections are defined as infections that develop in hospitalised patients and were neither present nor incubating at the time of their admission in hospital.

It includes infections that occur as a consequence of healthcare, regardless of whether they arise during hospitalisation

How do one define VAP, HAP and HCAP? Does these definitions help in treatment and outcome ?

(i) **HAP** (Hospital associated pneumonia) is defined as pneumonia that occurs 48 hours or more after admission, which was not incubating at the time of admission .HAP may be managed in a hospital ward or in the intensive care unit (ICU) when the illness is more severe.

(ii) **VAP** (Ventilator associated pneumonia) refers to pneumonia that arises more than 48–72 hours after endotracheal intubation . Although not included in this definition, some patients may require intubation after developing severe HAP and should be managed similar to patients with VAP.

(iii) **HCAP** (Health care associated pneumonia) includes any patient who was hospitalized in an acute care hospital for two or more days within 90 days of the infection; resided in a nursing home or long-term care facility; received recent intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days of the current infection; or attended a hospital or hemodialysis clinic.

These definitions afford us the opportunity to better understanding the mechanisms leading to the acquisitions of these infections and to design and evaluate interventions to prevent their occurrence.

Proper understanding and correct approach to these infections may help to get a favourable outcome.

What are the risk factors of multidrug resistant organisms in these pneumonias ?

The important risk factors are :

(i) Antimicrobial therapy in preceding 90 days.

(ii) Current hospitalization of 5 days or more.

(iii) High frequency of antibiotic resistance in the community or in the specific hospital unit.

(iv) Presence of risk factors for HCAP:

Hospitalization for 2 days or more in the preceding 90 days.

Residence in a nursing home or extended care facility.

Home infusion therapy (including antibiotics).

Chronic dialysis within 30 days.

Home wound care.

Family member with multidrug-resistant pathogen.

(v) Immunosuppressive disease and/or therapy suggesting a variable outcome impact, according to the severity of underlying medical conditions

What is the incidence of HAP, VAP in hospitalised children ?

(i) It is often difficult to define the exact incidence of HAP and VAP, because there may be an overlap with other lower respiratory tract infections, such as tracheobronchitis, especially in mechanically ventilated patients.

(ii) The exact incidence of HAP is usually between 5 and 15 cases per 1,000 hospital admissions depending on the case definition and study population; the exact incidence of VAPis 6- to 20-fold greater than in nonventilated patients

Is there a difference between early onset and late onset VAP/HAP regarding outcome and bacterial identification ?

(i) Early onset HAP and VAP – Occur within first 4 days of hospitalisation.

(ii) Late onset HAP and VAP – Occur 5 days or more after hospitalisation.

(iii) Early onset HAP and VAP are more likely to be caused by antibiotic sensitive bacteria and carry a better prognosis.

(iv) Late onset HAP and VAP are more likely to be caused by multidrug resistant bacteria and are associated with more mortality and morbidity

(v) Patients with early onset HAP who have received prior antibiotics or who have prior history of hospitalisation within the past 90 days are at greater risk for colonisation and infection with MDR pathogens and should be treated similar to patients with late onset HAP and VAP.

What are common nosocomial pathogens as reported in the nurseries in national surveillance study ?

Commonly reported pathogens by site of nosocomial infection in high risk nurseries participating in the national nosocomial infection surveillance system shown in Table 1.

Table 1. *Common nosocomial pathology*

Pathogen	Pneumonia (%)
Coagulase negative staphylococci	16.5
Staphylococcus aureus	16.7
Group B streptococci	5.7
Enterococci	4.6
Candida spp	0
Esch coli	5.8
Other streptococcus spp	3.3
Enterobacter spp	8.2
Klebsiella pneumonie	5.8
Pseudomonas aeruginosa	11.7
Hemophilus influenze	1.4
Viruses	0
Gram positive anerobes	0
Other enteric bacilli	0
Others	21.7

Data are from 1986 to 1993

What are the common modes of transmission of nosocomial infections?

The common modes of transmission is shown in Table 2.

What is the commonest mode of contact for nosocomial infections ?

A. Contact :

Direct contact – Commonest mode of transmission of organism to patients is the hands of medical and nursing staff.

Table 2. *Common models of transmission of nosocronial infections*

Mode of transmission	Nosocomial infection	Reservoir	Source
Airborne	Measles,varicella,pulmonary tuberculosis	Infected persons	Airborne droplet nuclei
Contact			
(i) Direct	Neonatal staphylococcal skin infection	Infected/colonized caregiver	Drainage from infected wound on hand of caregiver
(ii) Indirect	Respiratory syncytial virus infection	Infected persons	Hands of caregivers, fomites
	Infection with antimicrobial resistant bacteria	Infected/colonized persons	Hands of caregivers, fomites
(iii) Droplet	Pertussis, invasive menigococcal disease, group A streptococcal infection	Infected /colonized persons	Large respiratory droplets
Equipment			
(i) Oxygen, nebulizer, ventilator	Gram negative bacilli	Infected / colonized caregivers or equipment	Hands of caregivers, fomites
(ii) Humidifier, tracheal	Gram negative bacilli	Infected / colonized caregivers or equipment	Hands of caregivers, fomites
(iii) Suction, catheter	Gram negative bacilli	Infected / colonized caregivers or equipment	Hands of caregivers ,fomites

Indirect contact – Contact of susceptible host with a contaminated usually inanimate object, ex-rectal thermometer, resuscitation equipments.

Droplet contact – transmission of micro-organisms from conjuctiva, nose and / or month of an infection person to the patient (as a result of couhing and sneezing). Infected dooplet travel less than three feet, and transmission by this route thus requires close contact.

B. Airborne route :

Transmission occur by dissemination either of droplet nuclei from patients or personnel or of dust particle containing the infectious agents.

Common airbone infections are :

(i) *Varicella zoster*
(ii) Influenza A,B
(iii) Parainfleunza
(iv) Measls virus/mumps virus
(v) *Mycobacterium tuberculosis*
(vi) *Neissenia meningitides*
(vii) *Yarsinia pestis*
(viii) *Bordetella pertussis*

What are the recommendation for modifiable risk factors during intubation ?

1. Intubation and reintubation should be avoided, if possible, as it increases the

risk of VAP.

2. Noninvasive ventilation should be used whenever possible in selected patients with respiratory failure.
3. Orotracheal intubation and orogastric tubes are preferred over nasotracheal intubation and nasogastric tubes to prevent nosocomial sinusitis and to reduce the risk of VAP, although direct causality has not been proved .
4. Continuous aspiration of subglottic secretions can reduce the risk of early-onset VAP, and should be used, if available .

Role of oral antiseptics and antibiotics to prevent colonization in hospital ?

1. Routine prophylaxis of HAP with oral antibiotics (selective decontamination of the digestive tract or SDD), with or without systemic antibiotics, reduces the incidence of ICU-acquired VAP, has helped contain outbreaks of MDR bacteria, but is not recommended for routine use, especially in patients who may be colonized with MDR pathogens.
2. Prior administration of systemic antibiotics has reduced the risk of nosocomial pneumonia in some patient groups,but if a history of prior administration is present at the time of onset of infection, there should be increased suspicion of infection with MDR pathogens.
3. Prophylactic administration of systemic antibiotics for 24 hours at the time of emergent intubation has been demonstrated to prevent ICU-acquired HAP in patients with closed head injury in one study, but its routine use is not recommended until more data become available.
4. Modulation of oropharyngeal colonization by the use of oral chlorhexidine has prevented ICU-acquired HAP in selected patient populations such as those undergoing coronary bypass grafting, but its routine use is not recommended until more data become available.
5. Use daily interruption or lightening of sedation to avoid constant heavy sedation and try to avoid paralytic agents, both of which can depress cough and thereby increase the risk of HAP.

What is the diagnostic strategy and approach to HAP, VAP and HCAP?

The diagnostic strategy and approach is shown in Fig 1.

Major principles in management of nosocomial pneumonia

1. Avoid untreated or inadequately treated HAP, VAP, or HCAP, because the failure to initiate prompt appropriate and adequate therapy has been a consistent factor associated with increased mortality.
2. Recognize the variability of bacteriology from one hospital to another, specific sites within the hospital, and from one time period to another, and use this information to alter the selection of an appropriate antibiotic treatment regimen for any specific clinical setting.
3. Avoid the overuse of antibiotics by focusing on accurate diagnosis, tailoring therapy to the results of lower respiratory tract cultures, and shortening duration of therapy to the minimal effective period.
4. Apply prevention strategies aimed at modifiable risk factors This guideline

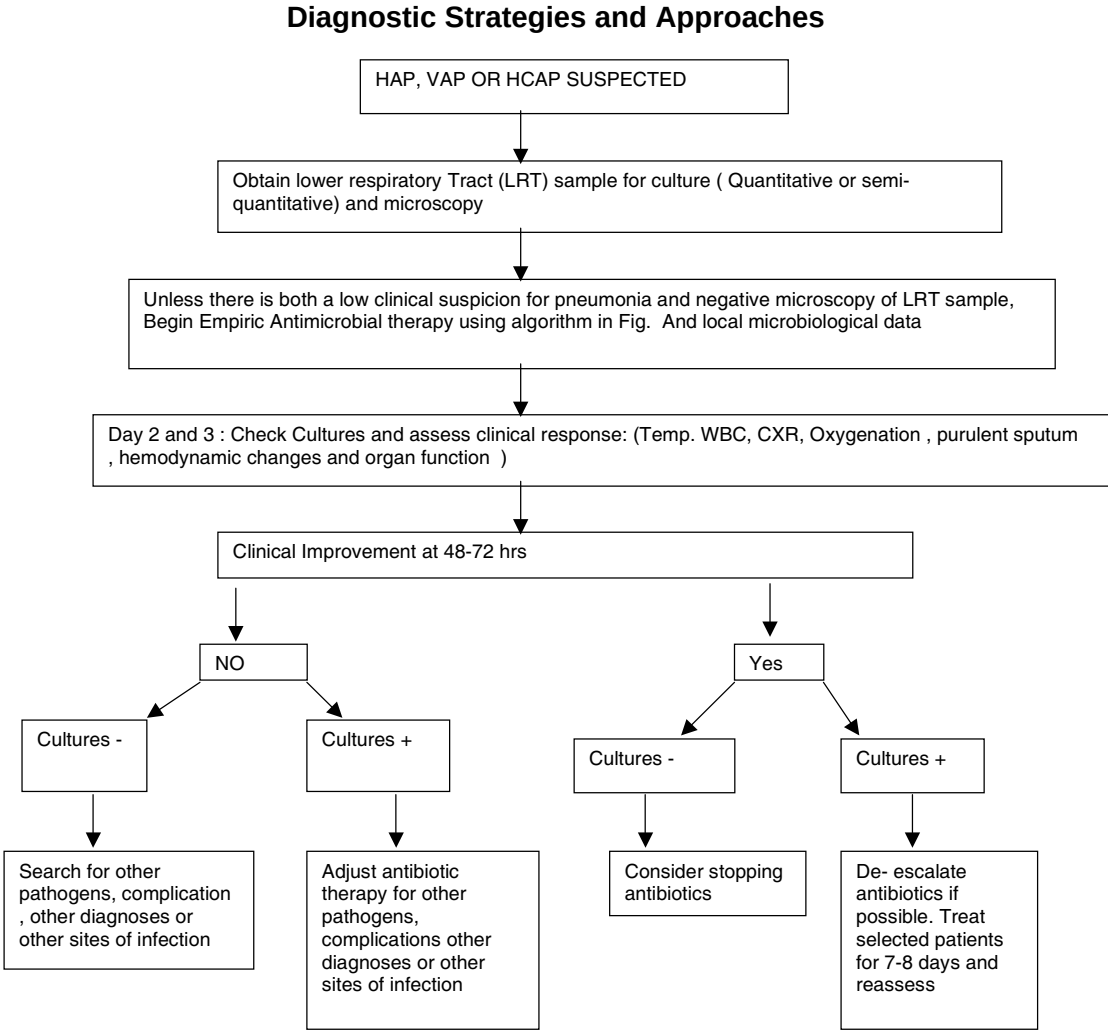


Fig 1. Dagnostic strategy and approach to HAP, VAP and HCAP

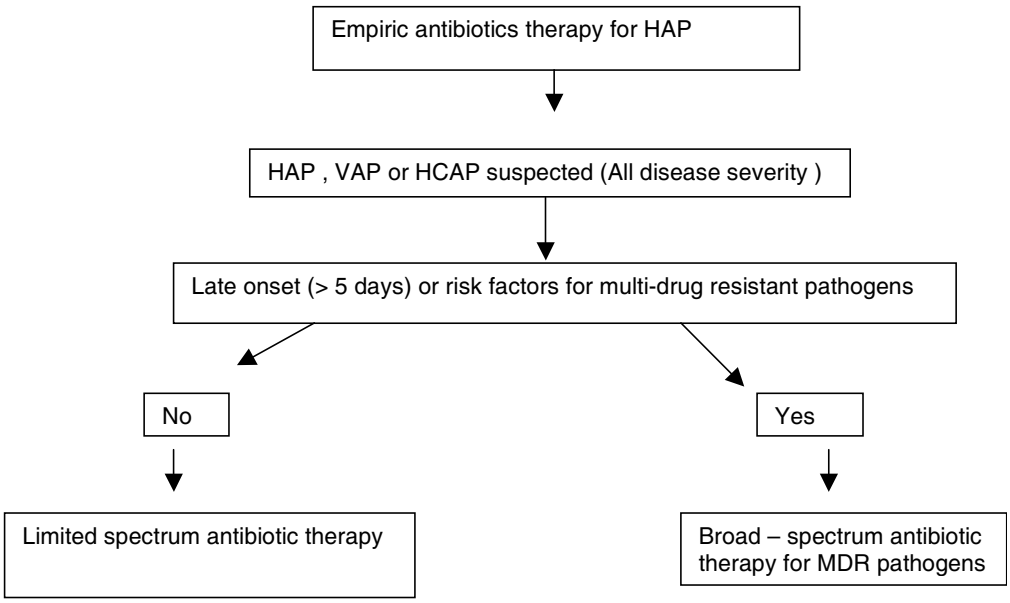
is not meant to replace clinical judgment, but rather to give an organizational framework to patient management. Individual clinical situations can be highly complex and the judgment of a knowledgeable physician with all available information about a specific patient is essential for optimal clinical management.

Algorithm for starting empiric antibiotic therapy HAP, VAP ?

The algorithm is shown in Fig 2.

Initial empiric antibiotic therapy for VAP and HAP ?

Initial empiric antibiotic therapy for hospital-acquired pneumonia or ventilator-associated pneumonia in patients with no



Algorthim for initiating empiric antibiotic therapy for hospital acquired pneumonia (HAP) ventilator asoociated pneumonia (VAP)), and health care associated pneumonia (HCAP).

Fig 2. Algothm for starting emperic antibiotic therapy in HAP and VAP

known risk factors for multidrug-resistant pathogens, early onset, and any disease severity.

(i) *Streptococcus pneumonie*† – Ceftriaxone.

(ii) *Hemophilus influenze* b – Ceftriaxone.

(iii) Methicillin-sensitive *Staphylococcus aureus* (MSSA) – Cloxacillin or clindamycin.

(iv) Antibiotic-sensitive enteric Gram-negative bacilli or *Esch coli* – Ampicillin/ sulbactam.

(v) *Klebsiella pneumonie* or enterobacter species – Imepenem or Meropenem or Aztreonam.

(vi) Proteus species – Imepenem or Meropenem or Azteonem.

† The frequency of penicillin-resistant *S pneumonie* and multidrug-resistant *S pneumonie* is increasing; vancomycin is the drug of choice for MDR pneumococcus.

(vii) In patients with late-onset disease or risk factors for multidrug-resistant pathogens and all disease severity :

Pseudomonas aeruginosa or

Klebsiella pneumonie (ESBL*) – Antipseudomonal carbepenem

Acinetobacter species* (Imipenem or meropenem)

Or

Lactam/lactamase inhibitor (piperacillin–tazobactam)

plus

Antipseudomonal fluoroquinolone* (ciprofloxacin or levofloxacin)

or

[Continued on page 23]

Nosocomial Pneumonia

[Continued from page 18]

Aminoglycoside (amikacin, gentamicin, or tobramycin)

(viii)Methicillin-resistant *Staphylococcus aureus* (MRSA) – (Linezolid or vancomycin**)

If *L pneumophila* is suspected, the combination antibiotic regimen should include a macrolide (eg, azithromycin) or a fluoroquinolone (eg, ciprofloxacin or levofloxacin) should be used rather than an aminoglycoside.

**If MRSA risk factors are present or there is a high incidence locally.

*If an ESBL strain, such as *Kl pneumonie*, or an acinetobacter species is suspected – A carbapenem is a reliable choice.

Recommendations for MDR pathogens?

Major points and recommendations for optimal antibiotic therapy (Level of evidence)

1. Empiric therapy of patients with severe HAP or VAP requires the use of antibiotics at optimal doses, to ensure maximum efficacy (Level I). Initial therapy should be administered to all patients intravenously, with a switch to oral/enteral therapy in selected patients with a good clinical response and a functioning intestinal tract. Highly bioavailable agents, such as the quinolones and linezolid, may be easily switched to oral therapy in such patients (Level II) .
2. Aerosolized antibiotics have not been proven to have value in the therapy of VAP (Level I). However, they may be considered as adjunctive therapy in patients with MDR Gram-negatives who are not responding to systemic therapy (Level III).
3. Combination therapy should be used if patients are likely to be infected with MDR pathogens (Level II).

   No data have documented the superiority of this approach compared with monotherapy, except to enhance the likelihood of initially appropriate empiric therapy (Level I).
4. If patients receive combination therapy with an aminoglycoside containing regimen, the aminoglycoside can be stopped after 5–7 days in responding patients (Level III).
5. Monotherapy with selected agents can be used for patients with severe HAP and VAP in the absence of resistant pathogens (Level I). Patients in this risk group should initially receive combination therapy until the results of lower respiratory tract cultures are known and confirm that a single agent can be used (Level II).
6. If patients receive an initially appropriate antibiotic regimen, efforts should be made to shorten the duration of therapy from the traditional 14 to 21 days to periods as short as 7 days, provided that the etiologic pathogen is not *P aeruginosa*, and that the patient has a good clinical response with resolution of clinical features of infection

How to assess response to therapy ? What to do for non-responders ?

1. A serial assessment of clinical parameters should be used to define the response to initial empiric therapy

(Level II). Modifications of empiric therapy should be made on the basis of this information, in conjunction with microbiologic data (Level III).

2. Clinical improvement usually takes 48–72 hours, and thus therapy should not be changed during this time unless there is rapid clinical decline (Level III). Nonresponse to therapy is usually evident by day 3, using an assessment of clinical parameters (Level II) .
3. The responding patient should have de-escalation of antibiotics, narrowing therapy to the most focused regimen possible on the basis of culture data (Level II) .
4. The nonresponding patient should be evaluated for noninfectious mimics of pneumonia, unsuspected or drug-resistant organisms, extrapulmonary sites of infection, and complications of pneumonia and its therapy. Diagnostic testing should be directed to whichever of these causes is likely (Level III) .

Pediatric Pulmonology Update

Resumed Publication

P*ediatric* P*ulmonology* U*pdate,* is the official scientific journal of IAP Respiratory Chapter. The publication is resumed from January 2009. For the present 2 years 2009 and 2010 this journal will be published from Kolkata. Three issues will come out every year. The 1st issue will be **Winter** issue, which will be published during National PEDICON. The 2nd issue will be published as **Summer** issue – this will be published as a mid-term issue and the 3rd issue the **Spring** issue will be published during the National PEDRESPICON.

We need your whole hearted support and we request you to contribute original paper; review article, current topic, interesting case report, journal and website searches. We will accept articles from our members working in peripheries specially interesting case reports and letter to the editors.

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Recurrent Pneumonia

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How do one differentiate recurrent pneumonia from persistent pneumonia?

(i) Recurrent pneumonia is defined as "at least two episodes of pneumonia occurring in one year or three episodes over any period of time".

(ii) Between two different episodes of recurrent pneumonia the child improve completely and there is radiological improvement also.

(iii) On persistent pneumonia, the clinical and radiological improvements are only partial, or not at all.

Is radiology essential to confirm a recurrent pneumonia?

(i) One may not take radiograph in the first episode, but once a patient has experienced an apparent second episode, follow up films are essential to document radiographic resolution.

(ii) Reviewing all available x-rays are imperative to identify the problem.

How can one exclude masqueraders in persistent / recurrent pneumonia ?

The common "masquerades" are

(i) Sternoclavicular joint – see the rotation

(ii) Breast shadow – see the opposite side

(iii) Plait of hairs

(iv) Bifid ribs / ricket / hemolytic disorder— Density of the opacity.

(v) Segmental collapse.

(vi) Pulmonary edema, tropical esinoplilia.

(vii)Pulmonary hemosiderosis.

What are the possibilities if recurrent pneumonia appear in the same lobe ?

Recurrent pneumonia in the same lobe– The possibilities are :

(i) Congenital anomaly

(ii) Tumor

(iii) Foreign body

(iv) Tuberculosis

What are the possibilities if recurrent pneumonia appear in different lobes ?

Recurrent pneumonia in different lobes– The possibilities are :

(i) Aspiration syndrome

(ii) Asthma

(iii) Cardiovascular shunt

(iv) Cystic fibrosis

(v) Immunodeficiency

What are the common clinical signs one must examine to clinch etiology in recurrent pneumonia ?

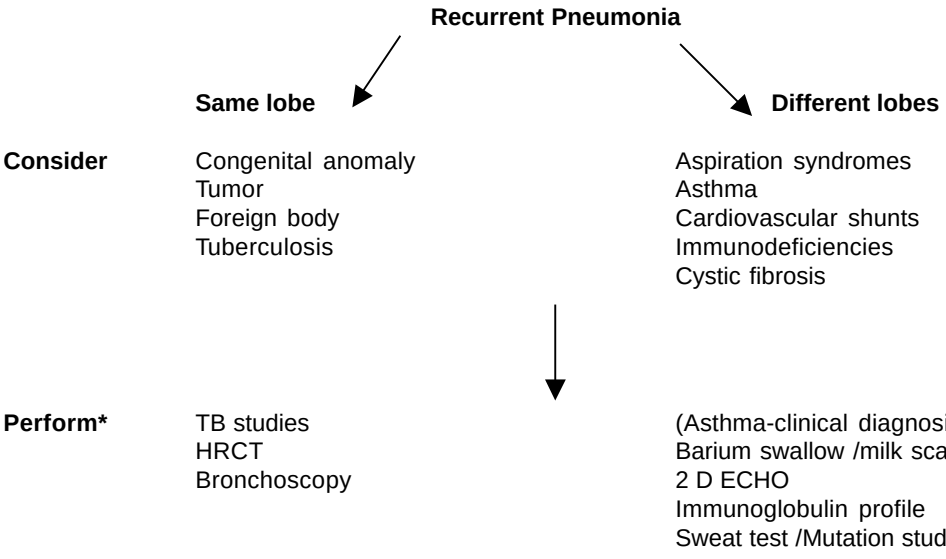
The common clinical signs are shown in Table 1.

Table 1. *Common clinical signs that are (Must) to be examined*

What	Why
Oropharyngeal examination	Anatomical causes of aspiration , eg submucous cleft palate, thrush (immunodeficiency)
Clubbing	Cystic fibrosis, immunodeficiencies, primary ciliary dyskinesias
Other features of atopy eg, flexural dermatitis	Asthma
Failure to thrive	Aspiration syndromes, mucociliary defects,immunodeficiencies
BCG scar, tonsil size	To assess immune status
Pallor, generalized adenopathy	HIV infection
Cardiovascular system	Dextrocardia and situs inversus in Kartageners, Murmurs in shunts
Respiratory system	Localizing signs in tumors, foreign body (unilateral wheeze), generalized wheeze (asthma)

Set an algorithmic approach for investigations while approaching a recurrent pneumonia.

The algorithmic approach is shown in Fig 1.



*Selection of the tests is based upon the clinical evaluation described earlier.

Fig 1. Algorithmic approach to recurrent pneumonia

Pitfalls In Pulmonary Imaging

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Sensitivity of radiologic findings in diagnoses of pneumonia.

(i) Pneumonia can be diagnosed clinically and it is not mandatory to have an x-ray before initiation of empirical treatment

(ii) The sensitivity of an x-ray to diagnose pneumonia is approximately 75%. In early bacterial pneumonia, quite commonly we may get a normal x-ray.

(iii) Specificity ranges from 42% to 100%, in different studies, because of varying definitions of pneumonia.

Technique for getting a good film in suspected pneumonia.

(i) Correctly exposed chest radiograph made with short exposure time to minimize respiratory motion are essential.

(ii) Images should be made as close as possible to the full inspiration to avoid errors of over interpretation resulting from physiologic underareation.

(iii) Radiograph made in expiration or patient rotated may lead to erroneous diagnoses of pneumonia.

When we should take views other than frontal view of chest x-ray, in a case of pneumonia ?

(i) In most cases of pneumonia a frontal view (PA or AP) is good enough.

(ii) In doubtful cases (like pneumonia in retrocardiac region) lateral view helps may be required in 5 – 10% of cases only.

(iii) In equivocal cases oblique or decubitus view may help to clarify, a suspected thoracic abnormality. With decubitus view the "upside" is inspiratory side and the "downside" is expiratory side.

When HRCT should be considered in a case of pneumonia ?

(i) High resolutation, thin section CT is of value in complicated cases of :

(a) Diffuse disease.

(b) When brochiectasis is suspected, despite of the presence of a normal or near normal chest x-ray.

(ii) HRCT gives the best detail of lung parenclyma.

In case of CAP with good clinical responses with treatment, when we should do a follow-up radiograph ?

(i) No follow-up radiograph is necessary in a case of CAP with good clinical response. Control radiograph is necessary only in cases.

(ii) Doubt in diagnosis

(iii) No clinical improvement or deterioration

(iv) Complication (empyema, pneumo-thorax)

(v) Round pneumonia.

(vi) Lobar atelectasis associated with CAP.

Good clinical response with treatment, but persistent radiographic shadow – what to do ?

(i) No action necessary, complete radiologic resolution may take several months.

(ii) Radiologic clearance after resolution of pneumococcal pneumonia may takes as long as 3 months.

(iii) *Staph aureus* – May take even longer.

(iv) Viral pneumonia – Radiologic clearance may take many months.

Can we make an etiologic diagnosis on the basis of chest x-ray in cases of pneumonia ?

(i) Typical bacterial infections, produce alveolar involvement, whereas interstitial involvements are more common in viral pneumonia and after mycoplasma and chlamydia infections.

(ii) Bacterial infections to start with may have interstitial shadows, whereas mycoplasma infections may have alveolar opacities. It is impossible to make an etiologic diagnosis solely on the basis of chest radiograph, because of many overlapping features.

What are the radiographic appearance of pneumo-nia in newborn and infants?

Lobar consolidation is very unusual in infants.

Many anomalies which are commonly found in chest x-rays in newborn or infants with pneumonia are :

(i) Generalized hypereration.

(ii) Irregular aeration (alternating areas of air trapping and atelectasis).

(iii) Atelectasis.

(iv) Bronchial wall thickening. (ring shadow and tramline)

(v) Less frequently bronchopneumonia– Interstitial shadow.

What are the radiographic findings of viral pneumonia ?

(i) Predominant alveolar involvement, effusion, empyema are very uncommon.

(ii) Hypereration, prominent lung markings due to bronchial wall thickening and focal areas of atelectasis, are common radiologic findings. Hili may be somewhat prominent, but major hilar adenopathy is uncommon.

(iii) Radiographic features are very close to and often indistinguishable from bronchiolitis or reactive airway disease.

What are the radiographic features of *Staphylococcus aureus ?*

(i) In contrast to pneumococcal pneumonia, staphycoccal pneumonia is lobular or bronchopneumonia that begins in the airways rather than in alveoli.

(ii) Severe hemorrhagic pulmonary edema may develop rapidly.

(iii) Pneumatocoeles develop in 40 – 60% cases . Usually appear in the 1st week of the disease and may take 3 weeks for resolution.

(iv) Pleural effusion and empyema may develop in 90% cases.

What are the radiographic features in *Hemophilus influenze* type B infection of chest ?

(i) Radiographic pattern is nonspecific and shows opacities that often begin as a segmental, interstitial – appearing process but progress to air space consolidation.

(ii) Approximately 2/3rd of cases have

unilateral involvement, but more than one lobe is involved in 25% of the time.

(iii) Empyema is a common complication occurring in about 40% of cases.

Radiographic mimics of primary pneumonia :

(i) Thymus
(ii) Hair braids / breast bud.
(iii) CCAM / sequestration.
(iv) Bronchogenic cyst.
(v) Asthma with atelectasis.
(vi) Pulmonary edema, ARDS.

Radiographic appearance of specific lobar consolidations :

Right upper lobe (RUL) :

(i) When consolidation extends caudally as far as the minor fissure, they are sharply defined by a horizontal margin. Consolidations along the medial margin may resemble a large right lobe of the thymus or be obscured by the thymus.

Right middle lobe (RML) :

(i) Even complete consolidation of RML produces widely different appearances in different individuals, because of significant anatomic variation in sizes shape of the middle lobe.
(ii) On frontal projection it usually obliterates the right heart border.
(iii) Lateral view may be helpful.

Right lower lobe (RLL):

(i) Lower lobe only rarely undergoes complete consolidation.
(ii) Usually either the superior or the basilar segments are involved.
(iii) Radiographic feature can appear as opacity in retrocardiac area or in the posterior portion of the lower lobe projected through the diaphragm on frontal view.
(iv) Lateral view may be helpful.

Left upper lobe (LUL) :

(i) Left upper and lower lobes overlap in the frontal plane through the entire course of interlobar fissure, except at the apex. So lateral projections are essential for localization.
(ii) As in RUL, consolidation is usually incomplete.
(iii) LUL pneumonia can simulate a large thymic lobe.
(iv) Consolidation of the lingular segment is similar radiographically to right middle lobe pneumonia and is recognized by indistinctness of the left cardiac margin.

Left lower lobe (LLL) :

(i) Even massive consolidation of left lower lobe (LLL) may be obscured by the cardiac shadow in an underpenetrated frontal film.
(ii) Common clues provided by the silhouette – Signs are obscuration of descending aorta margin and the contour of left hemidiaphragm.
(iii) In normal lateral chest film the more caudal thoracic vertebral bodies appear darken than the cranial ones.

Answers to Radiology Forum
(Cases mentioned in page 19-22 of this issue)

Case 1 : Hyaline membrane disease.
Case 2 : Sequestration of lung left lower zone.
Case 3 : Plaits of hair mimicking upper lobe pneumonia.
Case 4 : Retropharyngeal abscess.
Case 5 : Pneumococcal pneumonia.
Cace 6 : Staphylococcal bronchopneumonia.
Case 7 : Pneumococcal pneumonia.
Case 8 : Esophageal duplication cyst.
Case 9 : Cystic fibrosis

Recent Developments in Pneumococcal Disease Prevention

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Health indicators are required to understand the health status of a given community and also to compare the health status of one country with that of another. Indicators help to measure the extent to which the objectives and targets of a programme are being attained. The following mortality indicators are important health indicators. At present the Under Five Mortality Rate (UFMR) in India stands at 74.3, Infant Mortality Rate (IMR) at 57 (72% of UFMR) and Neonatal Mortality Rate (NMR) at 39 (48% of UFMR and 65% of IMR) [1, 2]. In 1980 the UFMR was 142, IMR was 122 and NMR was 78. These figures show that there is statistically significant improvement in the health indices over the last three decades. But mere statistical improvement does not reflect the whole scenario. India has the dubious distinction of leading the list of under-five deaths with 2.07 million deaths annually[3]. The whole world is concentrating on Millennium Development Goals 4 (MDG4) and MDG4 focuses on reduction in child mortality. Even with the present drop in figures in India it will not be possible for India to achieve the MDG4 for child survival and child health and something drastic needs to be done[4].

What is the burden of pneumonia in children in our country?

When we see the world map with burden of any disease, it is depressing to see that India is smeared with red dots all over. When we discuss the specific causes of under-five deaths, we see that pneumonia contributes 19% of the under-5 deaths per year in the world as per estimates in 2004 and again India tops the list of contributors to this disease burden[5]. Approximately 2 million children in India died before their 5th birthday in the year 2006 and 20% or 400,000 of these deaths were due to pneumonia[5,6].

Pneumococcal disease:

It has been estimated that 15-50% of community acquired pneumonia in children below the age of 5 are due to *S. pneumonie*[5,7]. Approximately 50% of all episodes of severe pneumonia and therefore pneumonia related deaths are probably due to *S. pneumonie* [5]. From the above figures it can be reasonably concluded that *S. pneumonie* is responsible for 6.6-22 million episodes of pneumonia and 2,00,000 deaths in children annually in India. The absence of microbiologic confirmation of pneumococcus in most episodes of childhood pneumonia in India and world wide is because of poor infrastructure, insensitivity of blood cultures in diagnosis of pneumonia, lack of blood culture facilities and administration of antibiotics before cultures are sent. Apart from pneumonia, *S. pneumonie* also causes bacterial meningitis, otitis media and

septicemia.

The data about the serotype distribution of *S. pneumonie* in India is scanty. The published results of the IBIS study in patients with invasive pneumococcal disease (IPD) indicate that serotypes 6, 1, 19, 14, 4, 5, 45, 12, 7, 23 are the most prevalent with serotypes 1 and 5 accounting for 30% of invasive pneumococcal disease [8]. Another factor that has to be considered is that the serotypes causing pneumonia and otitis media differ from that causing invasive pneumococcal disease and usually reflect those serotypes present in the nasopharyngeal carriage. It is also to be kept in mind that this data is one decade old.

What is the impact of national programs on morbidity and mortality of pneumonia in India?

Several child health programs including the National ARI control program, the Child Survival and Safe Motherhood Program (CSSM), the Reproductive and Child Health Program (RCH) and most recently the Integrated Management of Neonatal and Childhood Illnesses (IMNCI) have since the past few decades attempted to reduce morbidity and mortality due to Acute Respiratory Infections (ARI) in children. Despite all these measures over the years ARI continues to cause significant childhood morbidity and mortality in India. The NFHS III survey revealed that overall only 69% of children with symptoms and signs of ARI seek health care and only 12.5% are prescribed antibiotics with wide regional and rural urban variations[8] .

In order to limit ARI in children there is an urgent need to strengthen the existing child health care programs and institute new strategies to reduce ARI related morbidity and mortality in India. Immunization against *H influenze* and *S pneumonie* which are the major etiologic agents of ARI in children is an important strategy in this regard.

Since bacterial pneumonia is a treatable disease, why should one consider vaccination?

Though bacterial pneumonia is treatable but the associated morbidity is a cause of concern. The mainstay of treatment of pneumococcal pneumonia is penicillin. Fortunately till date penicillin resistance is not alarming in India. But over the years resistance to penicillin is gradually increasing in India[7]. Further, penicillin resistance is high is many neighbouring Asian countries like China, Thailand, Malaysia, Korea and very high in Sri Lanka[3,7]. So India is surrounded by highly penicillin resistant countries. Even if a child suffers from pneumococcal pneumonia it does not give protection against all strains of pneumococci. There are 90 strains of Pneumococci so a child is always vulnerable of other strains of Pneumococci.

Since the incidence of bacterial pneumonia is far less compared to viral pneumonia how can a bacterial pneumonia vaccine reduce pneumonia morbidity?

Morbidity and mortality associated with bacterial pneumonia is high. Vaccine against two important bacteria which causes pneumonia, *H influenze* and pneumococci are available, whereas there is no available vaccine against viral pneumonia. In order to achieve MDG4, the overall pneumonia mortality has to be reduced.

Is there a vaccine covering all types of bacterial pneumonia in Indian population ?

The two most important pathogens of bacterial pneumonia are *H influenze* and pneumococci. There are very effective vaccines against these two pathogens.

What is the present status of Hib vaccine coverage in India? Has it altered the incidences of Hib pneumonia in vaccinated infants?

Hib vaccine coverage is mainly limited to urban population as it is not yet included in he National Immunisation Program (NIP) and its schedule. Efforts are on to include Hib vaccine in NIP as a combined vaccine along with DTP. In spite of low coverage it has cast a great impact on Hib infection in children. Hib vaccine has got good herd effect too.

What are the preventive measures available against pneumococci?

The only preventive tool currently available to control the menace of pneumococcal disease in children is 7-valent conjugate pneumococcal vaccine (PCV7). The PCV7 containing polysaccharide antigen of serotypes 4, 6B, 9V, 14, 18C, 19F and 23 has been licensed for universal immunization in the US since the year 2000. It covers 85% of the serotypes causing invasive disease in the US. Efficacy trials prior to introduction of the vaccine showed excellent safety, more than 95% reduction in IPD in those vaccinated and 30% reduction in radiologically proven pneumonia[9,10]. Apart from the direct benefits a significant decline in pneumococcal disease in unvaccinated contacts of the vaccinees was noticed following introduction of the vaccine in the immunization program due to herd effect due to reduced nasopharyngeal carriage[11].

GAVI initiative:

Based on the data from various efficacy studies, particularly the data from the developing countries and the Gambian study[12], WHO in its position paper 2007, recommended inclusion of PCV7 with high priority in developing countries with UFMR more than 50/1000 live births or burden of under-five deaths of more than 50, 000[13]. The Global Alliance of Vaccine and Immunization (GAVI) through advanced market commitments to industry have made available PCV7 at affordable price to developing countries including India[14]. There are 72 such GAVI eligible countries who qualify to include PCV in their National Immunization Program urgently (NIP)[15,16]. India with UFMR of 72 and under-five death burden of 2.07 million easily qualifies by these definitions to include PCV7 in the NIP urgently[14]. The price at which the PCV could be offered to India is as low as 0.15-0.3 USD per dose [15]. A Sinha *et al* conducted an economic analysis of pneumococcal vaccination of infants in countries eligible for financial support from GAVI and showed that introduction of PCV7 in National Immunization Schedule will be highly cost effective in 68 of the 72 GAVI eligible countries including India [16].

Initiative in India:

A high level expert committee meeting, the National Technical Advisory Group on Immunization (NTAGI), headed by the Secretary of Department of Biotechnology (DBT), Government of India, Dr MK Bhan, has recommended the use of PCV7 in the National Immunization Program. This will be effective from 2010. Indian Academy of Pediatrics Committee on Immunization

(IAP COI) had recommended the use of PCV 7 in 2007 on one-to-one named child basis after discussion with parents due limited serotype coverage and high cost[17]. The inclusion of PCV7 in the National Immunization Schedule is a landmark decision.

Does PCV7 vaccine cover all strains important in Indian children? PCV7 or 23 valent unconjugated vaccine: which is relevant in our setting?

Two vaccines are available; the 23-valent unconjugated pneumococcal polysaccharide vaccine and the 7 valent conjugate vaccines.

The unconjugated polysaccharide vaccine is a 23 valent vaccine (PPV 23), being a T cell independent vaccine it is poorly immunogenic below the age of 2 years, has low immune memory, does not reduce nasopharyngeal carriage and does not provide herd immunity. It has at best 70% efficacy against prevention of invasive pneumococcal disease in the high-risk population but offers no protection against non-bacteremic pneumonia/otitis media[18]. It is a safe vaccine with occasional local side effects. Not more than two life time doses are recommended.

The 7 valent pneumococcal conjugate vaccine (PCV 7) containing polysaccharide antigen of serotypes 4, 6B, 9V, 14, 18C, 19F and 23 linked to a protein carrier covers 85% of the serotypes causing invasive disease in the US. Efficacy trials showed excellent safety, more than 95% reduction in IPD in those vaccinated and 30% reduction in radiologically proven pneumonia[19]. The efficacy of PCV7 against acute otitis media was 8 %. Apart from the direct benefits a significant decline in pneumococcal disease in unvaccinated contacts of the vaccinees was noticed following introduction of the vaccine in the immunization program due to herd effect as mentioned earlier resulting from reduced nasopharyngeal carriage[20].

Can measles and influenza vaccine efficiently cut down pneumonia?

Measles is an integral part of EPI vaccines. It is a very important vaccine considering the huge burden of the disease. But bacterial pneumonia is a known complication of measles infection in children. Prevention of pneumococcal pneumonia has to be approached in tandem with measles vaccination.

Influenza vaccine is indicated for high risk children only. Though influenza is a common disease but there are limitations of influenza vaccination. It has to be administered every year as the influenza virus changes due to shift and drift. It may not be cost effective, logistically pertinent and effective vaccine in the long run for widespread coverage.

How a general IAP member face these situations regarding pneumococcal vaccine?

IAPCOI recommendations[21]:

For EPI – The burden of pneumococcal disease is the greatest among the underprivileged children in India. The conjugate pneumococcal vaccines are thus of public health importance and ideally should be available to all children. IAPCOI feels that Government of India should avail of this opportunity for GAVI support, establish a pneumococcal disease surveillance system and set into motion a process for inclusion of PCV in EPI.

High risk children – The IAP COI recommends administration of both PCV and PPV 23 in all high-risk children who can afford the vaccine. The PCV vaccines provide robust immune response and immune memory while PPV 23 provides expanded serotype coverage. If PCV is not affordable, at least PPV 23 should be given to high-risk children above 2 years of age.

Healthy children – IAPCOI recommends the use of the currently available conjugate pneumococcal vaccine (PCV 7) after one to one discussion with parents in healthy children aged less than 2 years (Category 3). There is no data to support pneumococcal vaccination in healthy children aged 5 years and above and is not recommended.

Dose and schedule:

Healthy children (PCV vaccine) –

(i) Dose is 0.5 ml IM

(ii) Routine vaccination: Three doses at 6,10,14 weeks and 1 booster at 15-18 months.

(iii) Catch up vaccination.

  (a) 6-12 months: Two doses 4-8 weeks apart and 1 booster at 15 - 18 months.

  (b) 12-23 months: Two doses 8 weeks apart.

  (c) 24-59 months: Single dose.

High risk children (PCV and PPV 23) –

(i) If affordable, PCV should be given first. For children aged less than 5 years follow the schedule mentioned above. For children older than 5 years a single dose of PCV is recommended (Currently available PCV 7 though licensed up to age 9 years, has been shown to be safe and immunogenic in children older than 9 years as well).

(ii) In children aged 2 years or more, PPV 23 should also be given as a single dose of 0.5 ml IM. If PCV has been given earlier, a gap of 2 months must be maintained between PCV and subsequent PPV 23.

(iii) A high-risk child who has received PPV 23 in the past but not PCV vaccine may be offered a single dose of PCV vaccine at the time of presentation if 2 months have elapsed since receipt of PPV 23.

(iv) Only one repeat dose of PPV 23 is recommended only for children who have sickle cell disease, hyposplenia, asplenia, congenital/ acquired immunodeficiency, those on immunosuppressive therapy, renal failure and nephrotic syndrome. The repeat dose of PPV 23 may be given after 3-5 years if the child is less than 10 years of age and after 5 years if child is aged more than 10 years.

The prevalence of penicillin resistance is low in India at present. But it is quite high in many Asian countries. There are evidences that the prevalence of penicillin resistance among pneumococcus are gradually increasing, thereby highlighting the need for an effective vaccine[22].

Is there any more cost effective and economic vaccine for pneumococcus in the pipeline ?

A 13 valent pneumococcal vaccine is to be introduced shortly; it adds protection to 6 additional serotypes to the 7 already present[23]. This vaccine when available will be an able replacement of PCV7. A ray of hope for the future. It has been argued that in the long run, it may even become necessary to use a combination of conjugate and a protein vaccine in order

to get the best protection and the widest coverage[24].

In our resource limited country what is more important: (a) Pulse polio campaign, (b) IPV, (c) measles or (d) pneumococcal vaccine?

Each one is a separate entity and has its own place in national health programs. Pulse polio campaign is there over the past 14 years and has been very successful. It is supported by WHO. Measles vaccine is in National Immunization Schedule and efforts are on to increase the measles vaccine coverage through WHO supported measles prevention program. IPV has gained much importance in national polio control strategy and is being introduced in a phased manner in the polio prevalent districts. IPV has been supported by Bill and Melinda Gates Foundation. Conjugate pneumococcal vaccine in developing countries like India is in GAVI initiative and focussed to control pneumococcal diseases. So when limited resource in India is considered, each vaccine has its own place.

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From "Metro" Page, "The Telegraph" Calcutta, Saturday 23 May 2009

One Child in 10 Prey to Asthma

One in every 10 children in Calcutta of age five to eight years is suffering from asthma, a study has revealed.

The ailment, genetic in most cases, is aggravated by the city's foul air which is rich in pollutants. Among the other aggravating factors are smoking and the presence of allergens in the atmosphere.

The study, conducted by *the respiratory chapter of the Indian Academy of Pediatrics,* covered more than 1,000 pre-school and school-going children and was conducted over a year.

"The findings, to be published soon, show that 10 per cent of the children in the age group of five-eight years are suffering from mild to acute asthma," said **Goutam Ghosh**, a consultant pediatrician and the national chairperson of the academy's respiratory chapter.

The study reveals a lack of awareness about prevention and treatment of asthma among the school authorities and parents. "The study came across asthma-triggering factors in more than 90 per cent of the schools, an indication of the .lack of awareness about the ailment," said Ghosh.

Academy secretary **K K Ghosh** said most schools in Calcutta and the districts did not follow asthma-management guidelines. "Schools must maintain a report card of students suffering from asthma. The card should be filled up by the doctors of asthmatic children".

The academy has written to All Bengal Teachers' Association requesting it to organise interactive sessions between doctors and school authorities.

What's New: Journal Scan

Arun K Manglik

Senior Consultant, Shishu Sanjivani Hospital for Children, Salt Lake, Kolkata

Features predicting failure of pathogen identification in patients with community acquired pneumonia.

When and how much to investigate, this is the question in the current era of laboratory/ radiology driven diagnoses and therapy. Endeman, *et al* (*Scand J Infect Dis.* 2008; **40(9)**:715-20) tried to assess the extent of diagnostic procedures to be performed in CAP. Two hundred and one patients were hospitalized for CAP, clinical and laboratory variables were collected. Pathogen identification was performed by culture of sputum and blood, urine antigen tests, polymerase chain reaction of sputum, serological testing and viral culture of the pharynx. In 128 patients (64%) a respiratory microorganism was identified. Analysis revealed that patients with history of pre-hospital antibiotic therapy, a medical history of hypertension and a low C-reactive protein, pathogen identification was difficult. Thus it was recommended that in these patients invasive testing in high-risk patients with CAP in the presence of these predictors should be considered at an early phase of hospitalization.

This data could provide situations where invasive investigations would be rather avoided. But, still very sorry, where does clinical examination stand? Are we moving further away from clinical methods? Lord Hutchison must be moving in his grave.

Oxygen therapy for lower respiratory tract infections in children between 3 months and 15 years of age.

It is very usual practice in lower respiratory tract infections (LRTIs) to administer oxygen. The effectiveness of oxygen therapy and different methods of delivery is unknown. This very effective Cochrane Database Systemic Review 2009:21(1), by Rojas, *et al* contributes to the rational use of oxygen in the treatment of LRTIs. The aim was to assess the indications of, effectiveness of, oxygen delivery methods and their safety. The Cochrane Central Register, MEDLINE, EMBASE and LILACS were searched, 551 titles were assessed. Obviously not without reason no RCT comparing oxygen versus no oxygen therapy could be found. Simply nobody had the guts. Four RCTs comparing delivery methods and 12 observational studies assessing the accuracy of clinical signs indicating hypoxemia were eligible. A meta-analysis of the RCTs comparing oxygen delivery methods was performed.

Main results :

Three studies assessed the effectiveness of nasal prongs (NP) versus nasopharyngeal catheters (NPC). The pooled estimate effect showed no differences (OR 0.96; 95% CI 0.48 to 1.93) in treatment failure (number of children failing to achieve adequate SaO_2). One study compared the effectiveness of NP versus nasal catheter (NC). No differences were found in treatment failure

(the mean number of episodes of desaturation/child: NC group 2.75, SD +/- 2.18 episodes/child; NP group 3, SD +/- 2.5 episodes/child, p = 0.64). Another study compared face mask (FM) and head box (HB) versus NPC. Use of FM showed lower risk of treatment failure (failure to achieve PaO_2 > 60 mmHg) than the NPC (OR 0.20; 95% CI 0.55 to 0.88), as also did the use of HB compared with NPC (OR 0.40; 95% CI 0.13 to 1.12). Studies assessing the accuracy of signs and/or symptoms indicating hypoxemia showed that cyanosis, grunting, difficulty in feeding and mental alertness have better specificity in predicting hypoxemia and its results were consistent among studies.

HB and FM proved rather better than NPC, but NP, NC or NPC all are more cost-effective considering oxygen is expensive in resource scarce situations.

Cefuroxime for empiric treatment of community-acquired pneumococcal pneumonia: is there a generation gap?

S pneumonie is very much in the news these days, with an estimated over 2million annual deaths in India, increasing drug resistance and costly vaccines being pushed into the market. The growing prevalence of penicillin-resistant pneumococci provoked the attempt to use second generation cephalosporins for empirical treatment of community-acquired pneumonia. Weiner-Well, *et al* in a recent retrospective study (*Chemotherapy,* 2009;**55(2)**:97-104) involving 31 adult patients with pneumococcal pneumonia and bacteremia caused by *S pneumonie* that was intermediately resistant to penicillin, and were compared with 31 control patients with similar infection caused by penicillin-susceptible pneumococci. Morbidity and mortality were studied.

Results :

All unsusceptible pneumococci isolates were intermediately resistant to penicillin. No cases of fully resistant pneumococci were isolated from blood cultures in our hospital. Presence of hematologic malignancy and immunosuppression were significantly associated with penicillin resistance. No significant difference in morbidity or mortality was detected between the 2 groups, and penicillin minimum inhibitory concentration was not found to be a factor associated with mortality. The authors concluded: patients with pneumococcal pneumonia caused by intermediately resistant pneumococci can be empirically treated with cefuroxime.

With drug resistance on the rise, though Indian scene still seems to be better off with percentage resistance figures being still quoted at <10% in most studies, we still have to be looking out for alternatives, be it beta-lactams, macrolides, cephalosporins etc. In regions where fully resistant pneumococci are rare, the use of a second generation cephalosporin for empiric treatment of community-acquired pneumonia may be appropriate.

Zinc modifies the association between nasopharyngeal *Streptococcus pneumoniae* carriage and risk of acute lower respiratory infection among young children in rural Nepal.

Again *Streptococcus pneumonie,* but now prevention and not by vaccines. This was a study by Coles, *et al* (*J Nutrition* 2008; **138(12)**:2462-7) in our neighbourhood, Nepal. The effect of Zinc prophylaxis on nasopharyngeal (NP) carriage of *Streptococcus pneumonie* and acute lower respiratory infection (ALRI) in children

aged 1-35 month was studied in a randomised, prospective rural based trial during an 18 month period. *Streptococcus pneumonie* carriage prevalence was compared in 550 ALRI cases with that of healthy age and season-matched controls. They were randomized to receive either 10 mg tablets of zinc or placebo daily. Approximately 75% of cases and controls were *Streptococcus pneumonie* carriers. There was an interaction between zinc and *Streptococcus pneumonie* carriage (P = 0.091). *Streptococcus pneumonie* carriage increased the risk of ALRI in the placebo group [adjusted matched odds ratio (AMOR) = 2.57; P = 0.025] but not in the zinc group (AMOR = 0.95; P = 0.890). Among the subset of symptomatic cases and their controls, the odds of ALRI for *Streptococcus pneumonie* carriers in the placebo group was 30 times greater (AMOR = 78.09; P = 0.006) than in the zinc group (AMOR = 2.77; P = 0.288). These findings suggest that zinc prophylaxis may protect children against ALRI associated with carriage of *Streptococcus pneumonie*.

Further evidence of the multiutility value of zinc. But several questions regarding how to administer zinc, what dose, when to start, at the start of any respiratory illness or anytime prophylactically at any contact with medical services, at some particular age?

Some surveillance data on pneumococcal disease in Asia and Africa
(*Clinical Inf Dis Journal*, 2009, Mar 1, **48**: Supplement 2)

Country	Author (et al)	Page no	Period of study	Methods	Age	Results
Bangladesh	Naheed	82-89	2004-07	Blood cultures	2-59 m	4155 samples, 161 culture +, 10 *S pneumonie*, isolation low, 68% got antibiotics
Bangladesh	Saha	75-81	2004-07	Blood and CSF cultures	<5 year	17969 blood cultures 3765 CSF culture sent, 139 *S pneumonie*+, 13 in pneumonia, 94 in meningitis and 32 in sepsis cases. 90% culture +ve <2year age. Resistance to penicillin-0%,chloramphenicol-6%,cotrimoxazol-36% Vaccine cover of serotype : 7valent -20%, 10val-43%, 13valent-50%
Thailand	Bagett	65-74	2005-07	Blood cultures, Pneumo Ag test	<5year	7319 blood cultures sent, 72 culture+ *S pneumonie* further 44 +ve on Ag test Incidence: Confirmed *S pneumonie* bacteremia needing hospital treatment = 10-28/lac population

[Table continued on page 40]

[Table continued from page 39]

Country	Author (et al)	Page no	Period of study	Methods	Age	Results
Vietnam	Anh	57-64	2005-06	Blood & CSF cultures, Pneumo Ag test, PCR	<5year	987 kids with suspicion of invasive bacterial disease, 17 proved pneumococcal disease Incidence of invasive pneumococcal disease: 48/lac population
Sri Lanka	Batuwanthu-dawe	136-40	2005-07	Blood, CSF culture, Ag test	2m-5year	23 *S pneumonie* isolates, common types =19F, 14,23F and 6B 7 valent vaccine covers 60% of isolates. 90% penicillin resistant
Nepal	Shah	123-8		Blood, CSF culture, latex agglutination Ag test	2mo-5yr	2461 blood cultures sent, 54 proved pneumococcal by various tests including cultures common serotypes= 1,5,2,7F. Resistance – penicillin-6%, cotrimoxarole-68%
Nepal	Williams	114-22		Blood, CSF cultures, pneumoniae Ag test	2m-5year	*S pneumonie* isolated in 17(2%) cases. Common serotype 1 and12A.
Bangladesh	Arifeen	103-13	2004-07	Blood, CSF cultures	1-59m	6925 blood and 41 CSF cultures sent. 26 *S pneumonie* identified Serotypes- 1,5, 14, 18C, 19A, 38. Resistance to Cotrimoxarole 76%, IPD incidence estimated at 86/ lac/child years
Nigeria	Falade	190-6	2 yr	Blood, CSF cultures	2-59mo	23 cases of *strepto pneumonie* isolated, 11 serotypes – 5, 19F, 4. All penicillin sensitive
East Africa	Mudhune	147-52		Blood, CSF cultures		442 confirmed *S pneumonie* isolates, 302 from blood and 140 from CSF. Resistance = pen senitive, cotrimox resistance++. 7 valent vaccine coverage = 67%
Burkina Faso Togo	Troare	181-9	2002-06	CSF culture, PCR, Ag test	All ages	2689 CSF samples, 463 *Strept pneumonie*+, Vaccine cover=7 valent -7%, 10valent -70%, 13valent-77%Incidence – 14/lac population, but in <1year=77.

Radiology Forun

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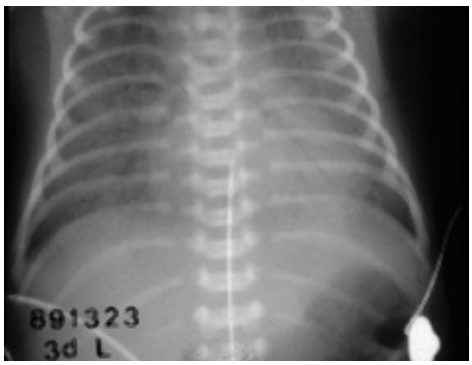
[Here 9 cases are presented with a brief history and relevant radiographic findings. The readers are requested to diagnose these cases. To check whether the readers are correct or not please see page No.29 – *Editors*]

Case 1

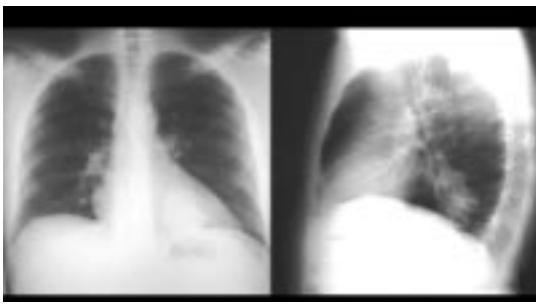
Thirty-two weeks born newborn developed respiratory distress on first day.



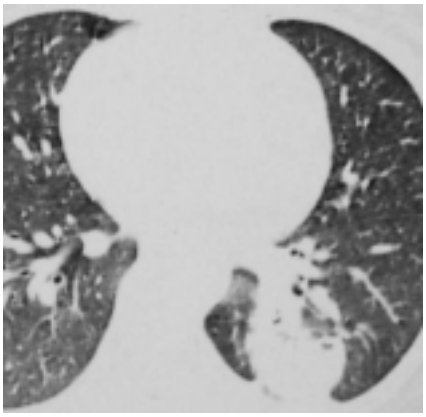
Xray Chest PA View. Diminished lung volume, bilaterally symmetrical involvement, ground glass appearance.

Case 2

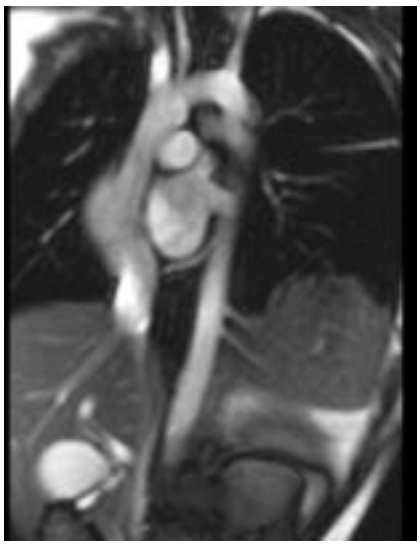
Fourteen-year old child developing recurrent pneumonia.



Xray Chest (AP & Lateral Views). Posterior left lower mass



CT Chest. Posterior left lower mass



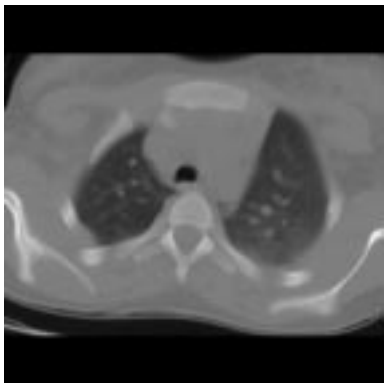
MRI. Two vessels from aorta feeding the posterior left lower mass

Case 3

Fourteen years old girl with sickle cell anemia and negative MT test, presented with chronic cough.

Xray Chest PA View. Bilateral upper lobe air space disease

Xray Chest Latral View. Does not show upper lobe air space disease

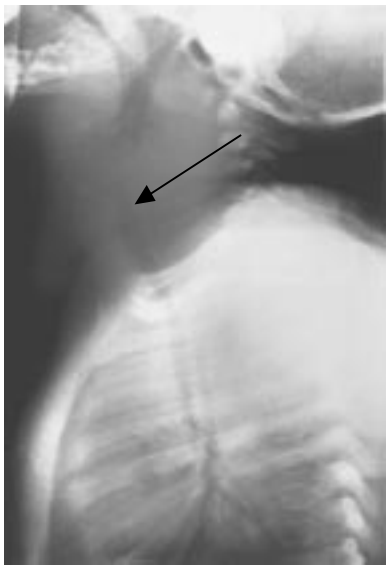


CT Chest. Clear upper lobe bilaterally

Case 4

Two year old child presented with high fever, unable to swallow anything.

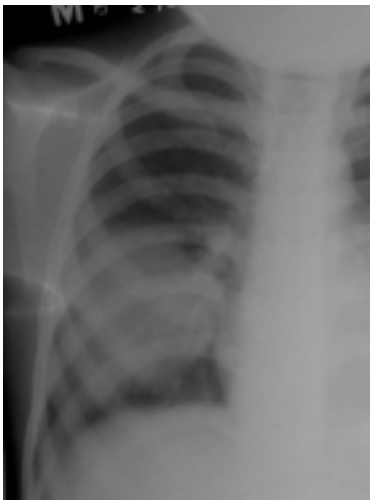
PA View of Chest Xray. Extension of the shadow in the superior mediatinum on right side



Lateral View of Chest Xray. Marked increase in retropharyngeas space and anterior deviation of trachea

Case 5

A 2 year old child presented with cough and fever. Same child 2 weeks after antibiotic therapy came with dis-apprearance of symptoms.

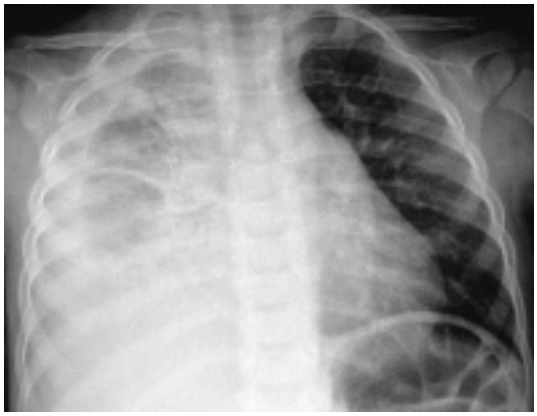


Chest Xray PA View. Round white opacity in right lower lobe

Xray Chest PA view of the same child (Case 5) 2 weeks after antibiotic therapy showing clearing of shadow

Case 6

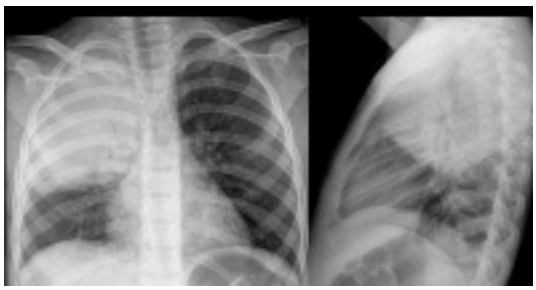
Toxic child with cough, respiratory distress and high fever.



Xray Chest PA View. Multilobar consolidation in right side. Fluid in horizantal fissure, indicating pleural effusion.

Case 7

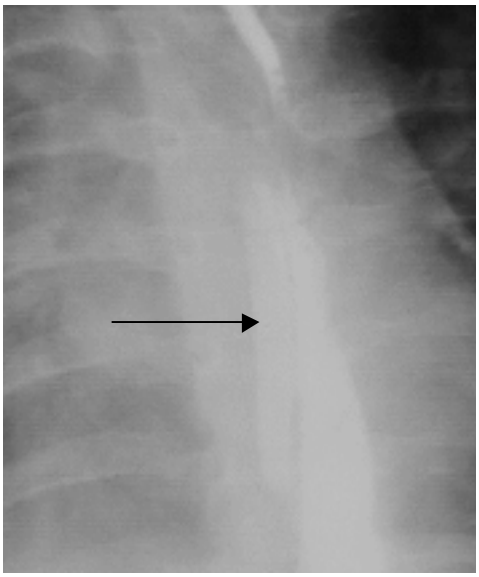
Child with high fever looked toxic presented with minimal cough,



Xray Chest PA and Lateral Views. Opacification of right upper lobe and bulging fissure. Note the sagging horizontal fissure on PA view. Oblique fissure is visible on lateral view.

Case 8

Six month old child with recurrent pneumonia.



Upper GI Contrast Study. Tubular mass filled with contrast by the side of esophagus.

Chest Xray PA View of the same girl at her age of 10 years with similar changes, ie, bronchiectatic changes and mucoid impaction

Case 9

A girl child with history of recurrent LRTI and weight loss, copius expectoration of sputum.

Chest Xray PA View of the girl at her 9 years of age with bronchiectatic changes and mucoid impaction

Chest Xray PA View of the same girl at her age of 12 years with similar changes