

PAEDIATRICS
AND
CHILD HEALTH
IN A TROPICAL REGION

3rd Edition

AZUBUIKE
&
NKANGINIEME

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PART TWENTY- ONE: CHAPTER 129

PLEURAL DISORDERS: PLEURAL EFFUSION, EMPYEMA THORACIS, CHYLOTHORAX, HAEMOTHORAX, PNEUMOTHORAX AND RELATED AIR-LEAK SYNDROMES

Abdul-Wahab B. R. JOHNSON, Rasheedat M. IBRAHEEM, and Peter O. ADEOYE

Inflammatory lesions of the lung parenchyma frequently involve the pleura. Pleural lesion of inflammatory origin is referred to as pleurisy. When there is no significant exudative pleural fluid accumulation associated with such a lesion, it is called dry or plastic pleurisy. On the other hand, pleural inflammation (frequently parapneumonic), associated with an appreciable accumulation of sero-fibrinous fluid is simply referred to as pleural effusion. A pleural effusion of purulent nature is otherwise known as *empyema thoracis*, or purulent pleurisy. In this section, the common paediatric inflammatory disorders of the pleura and their complications are discussed.

PLEURAL EFFUSION

This is the diagnostic term given to the pleural space accumulation of exudative fluid of inflammatory origin. According to Light's criteria for classifying pleural fluid (PF) accumulation, exudative fluid of pleural effusion is characterized by SG >1.016 , protein level 30gm/L ($>3\text{gm/dL}$), PF protein:Serum (S) protein ratio >0.5 . Other characteristics of exudative PF include PF-lactate dehydrogenase (LDH):S-LDH ratio ≥ 0.67 and PF-LDH $>2/3$ of normal S-LDH. The biochemical differences between an exudative and transudative pleural fluid accumulation are highlighted in **Table 129-1** below:

Table 129 -I: Biochemical differences between exudates and transudates in the pleural space

Pleural fluid content	Exudate	Transudate
Pleural fluid protein level	$>30\text{g/L}$ ($>3\text{gm/dL}$)	$<30\text{g/L}$ ($<3\text{gm/dL}$)
Pleural fluid glucose level	$<2.2\text{mmol/L}$ ($<40\text{mg/dL}$)	$>2.2\text{mmol/L}$ ($>40\text{mg/dL}$)
Pleural fluid lactate dehydrogenase (LDH) level	$>200\text{IU/L}$	$<200\text{IU/L}$
Pleural fluid protein to serum protein ratio	>0.5	<0.5
Pleural fluid-to-serum glucose ratio	<0.5	>0.5
Pleural fluid LDH and serum LDH ratio	≥ 0.67 or $>2/3$	<0.67 or $<2/3$
Pleural fluid cholesterol level	$>1.2\text{mmol/L}$ ($>45\text{mg/dL}$)	$<1.2\text{mmol/L}$ ($<45\text{mg/dL}$)
Pleural fluid pH	<7.2	>7.3
Pleural fluid specific gravity (SG)	>1.015	<1.015

In general, pleural inflammation associated with effusion, can be subdivided into:

- Dry or plastic pleurisy in which the inflammation is limited to visceral pleura, associated with fibrin deposits and adhesions between the pleural surfaces, and a small, but definite amount of serous (clear yellow), or slightly cloudy fluid with few leucocytes and occasional red cells.
- Sero-fibrinous or sero-sanguineous pleurisy is frequently associated with lung parenchymal inflammation like lobar pneumonia, and less commonly mediastinal and abdominal inflammatory lesions, and rarely pleural or pulmonary malignancies. The quality of the exudative fluid is the same with that of (i) above, but the volume is more.
- Empyema (purulent pleurisy), frequently associated with the common bacterial pneumonitides, (especially Staphylococcal and pneumococcal pneumonias), a distinct propensity for developing loculations of pus within the pleural cavities, and associated with a visibly purulent thoracocentesis fluid which has a low pH (<7.2), and a lactate dehydrogenase $>250\text{IU/L}$.

Despite the striking similarities in the chest findings, disorders associated with pleural fluid accumulation of non-inflammatory origin are generally not regarded as pleural effusion. Non-inflammatory/non-exudative pleural fluid accumulation include hydrothorax in which the nature of the pleural fluid is transudative (with a protein content $<30\text{gm/L}$ and SG <1.015), and is frequently of non-inflammatory origin. In hydrothorax, a transudate accumulates in the pleural space from passive transudation and the pleural membranes are hardly involved by the disease. Other causes of pleural fluid accumulation include haemothorax, a terminology used for the presence of blood in the pleural space, and chylothorax, defined as the presence of chyle (from the thoracic duct) in the pleural space. **Table 129-II** summarizes the diagnostic categories/ types of pleural effusions, the common associated or antecedent disease conditions, along with the corresponding pathophysiological mechanisms.

The discourse that follows highlights the relevant aspects of exudative fluid accumulation (pleural

effusion) in children, while the additional features of empyema thoracis (the commonest cause of pleural effusion in tropical infants and pre-schoolers) are highlighted separately. Although the discriminatory parameters from other causes of pleural fluid accumulation are indicated as appropriate, further details of 'non-inflammatory' pleural fluid accumulation or surgical pleural morbidities like haemothorax may be found in other standard texts. In the tropics, empyema is typically associated with a preceding *Staphylococcal pneumonia* (less so with other bacterial agents like *Streptococcus pneumoniae*, *H. influenzae*, and *Klebsiellapneumoniae*), measles, malnutrition and anaemia. On the other hand, plastic or sero-fibrinous pleuritis occur more commonly as a complication of pneumonia due to *Streptococcus pneumoniae*, *H. influenzae*, or pulmonary tuberculosis.)

Physiology of Pleural Space fluid, Aetiology & Pathogenesis of Excess Pleural Fluid Accumulation

The visceral and the parietal layers of pleura are lined with a single layer of flat ciliated mesothelial cells (~6.5µ thick) and a layer of collagen and elastic fibres (~30-40µ thick). Blood and lymphatic vessels abound between the connective tissue layer and the limiting membrane of the lung. In the normal state, the pleural membranes are permeable to liquid, but accumulation of pleural fluid is prevented by an efficient reabsorption at the venous end of the

capillaries, of close to 90% of the total volume of fluid filtered out from the arterial end; the net 10% get absorbed via the lymphatic channels. Hence, in health, the visceral and the parietal membranes are separated by only 1mL of pleural fluid. Pleural fluid accumulation occurs when there is an imbalance between filtration and absorption which are essentially governed by the well-known Starling forces associated with convective bulk movement of fluid between the vascular and extravascular compartments.

The relationship between Starling forces governing pleural fluid equilibrium between filtration and absorption is illustrated in the Table 129-III.

A net liquid re-absorption occurs because the absorptive pressure exceeds (albeit slightly) the filtration pressure. From the respective values of the filtration and absorptive forces at the parietal and the visceral pleurae, it is evident that fluid is filtered from the former, while absorption occurs mainly at the visceral end.

As indicated in the Table 129-I above, pleural effusion or fluid accumulation in the pleural space occurs when there is disequilibrium between forces that govern filtration and absorption, or there is impaired lymphatic drainage. Such situations occur when there is either: (i) Increased filtration in the presence of impaired absorption or, (ii) Normal filtration in the presence of impaired absorption, or removal via the lymphatic channels

Table 129-II: Types of Pleural Fluid and Associated Clinical Disorders

Type of Fluid (Synonymous Diagnostic terms)	Associated Clinical Disorders	Pathophysiological Mechanisms
Sero-fibrinous (Pleural Effusion, Parapneumonic effusion)	Contiguous bacterial pneumonia, Pulmonary tuberculosis, lung infarction, or malignancies [primary or metastatic], viral hepatitis, sarcoidosis, and the dyscollagenoses (e.g. rheumatoid arthritis and systemic lupus erythematosus)	Increased pleural capillary permeability, or increased oncotic pressure of the interstitial space
Purulent (empyema thoracis, Pyothorax, or Purulent Pleurisy)	Contiguous pyogenic bacterial pneumonia (especially <i>Staphylococcus aureus</i> & <i>Streptococcus pneumoniae</i>) Pulmonary tuberculosis, Lung abscess	Increased pleural capillary permeability
Hydrothorax	Hypoproteinaemic states (e.g. nephrotic syndrome, hepatic cirrhosis) peritoneal dialysis, congestive cardiac failure, pericarditis, overhydration and venous obstruction with [venous] hypertension.	Increased capillary hydrostatic pressure, decreased plasma oncotic pressure, or inappropriate lymphatic flow (with either inadequate outflow, or excessive inflow)
Chylothorax	Congenital chylothorax, hereditary lymphoedema, thoracic duct obstruction, mediastinal lymphadenopathy & fibrosis	Impaired lymphatic flow
Haemothorax	Chest trauma, pulmonary tuberculosis, blood dyscrasias, and rarely pulmonary malignancies	Vascular leakage or (neoplastic) erosion

Table 129-III: Starling relationship in the parietal and visceral pleura (After Montgomery & Sigalet, 2006)

	Parietal Pleura (cm H ₂ O)	Visceral Pleura (cm H ₂ O)
Forces moving liquid into the Pleural space (cm H₂O)		
Capillary hydrostatic pressure	30	11
Interstitial hydrostatic pressure (pleural fluid)	- 5	- 5
Oncotic pressure of pleural fluid	-6	- 6
Total pressure (cm H ₂ O)	41	22
Forces moving liquid into the Pleural Capillaries (cm H₂O)	32	32
Plasma oncotic pressure		
Net Force (cm H₂O)	- 9[out]	10[in]

Capillary filtration pressure can be increased by pleural infection, pulmonary infarction, or dyscollagenoses (e.g. rheumatoid arthritis), all of which increase the filtration coefficient because of inflammatory damage to the capillary basement membrane, or toxic damage to the endothelium. In addition, the protein content of pleural fluid contributes to the development of a higher pleural oncotic pressure with the consequent fluid accumulation exceeding lymphatic absorption. Hydrothorax may result from an increase hydrostatic pressure from systemic venous hypertension (e.g. fluid overload, right sided heart failure, pericarditis), or pulmonary venous hypertension (as is the case in congestive cardiac failure). In addition, significant reduction in plasma oncotic pressure associated with hypoproteinaemic states like nephrotic syndrome is associated with increase parietal filtration pressure and a reduction in the visceral absorption, especially when there is no commensurate increase in lymphatic drainage. Finally pleural fluid may accumulate as a result of several pathologies which are capable of impeding lymphatic drainage. These include systemic venous hypertension, parietal pleural thickening of tuberculous origin, or mediastinal adenopathy from lymphomas, chylothorax resulting from thoracic duct obstruction, overloaded lymphatic system (from peritoneal fluid absorption) as is the case with ascites associated with liver cirrhosis, and rarely, congenital hypoplasia of the lymphatic vessels (as is the case with hereditary lymphoedema). More often than not, the individual aetiological entities associated with pleural fluid accumulation, involve more than one mechanism.

The degree and type of functional pathology associated with pleural effusion depends on the volume of the fluid, the rapidity of the accumulation, and of course, the nature of the fluid. Increase in the elastic resistance to distension, reduction in lung volume (and hence the vital capacity), with little effect on the air flow, are associated with moderate or massive pleural effusion. With massive pleural effusion, mediastinal displacement may occur with the resultant impairment of contralateral lung expansion, as well as ipsilateral diaphragmatic displacement. Significant mediastinal displacement may cause kinking of the superior vena

cava with the consequent reduction in venous return, decreased right ventricular end-diastolic volume and thus, decreased stroke volume. Such an effusion with a significant haemodynamic compromise is referred to as *tension pleural effusion*. The overall functional pathology includes impaired gaseous exchange and cardiac function.

Clinical Features

The general/systemic symptoms and signs of pleural effusion depend not only on the underlying disease, but also on the size of the fluid in the pleural space, as well as the thoracic size. Indeed, these three factors apparently determine to a large extent, whether or not there are suggestive chest symptoms and findings on clinical examination. For example, while infective disorders like pneumonia and pulmonary tuberculosis may present with fever and/or weight loss, anaemia is usually associated with post-Staphylococcal pneumonia empyema. An underlying hypoproteinaemic state like nephrosis, may be associated with generalized oedema, a child with liver cirrhosis may present with jaundice and ascites, while prominent joint symptoms at presentation may suggest juvenile rheumatoid arthritis. Small effusions are generally asymptomatic, but with large effusions, mediastinal shift and significant cardio-respiratory difficulties may be inevitable.

Whereas, the triad of cough, breathlessness, and grunting respiration constitute the commonest symptoms of moderate to severe pleural effusion in children, the presence of grunting in a child with respiratory distress in a tropical climate, should narrow the diagnostic options to pleural effusion and/or lobar consolidation. Also, while breathlessness generally constitutes a prominent symptom of effusion in children, the severity of this symptom depends on the size of the fluid, duration of accumulation, and indeed, the pre-existing or primary respiratory morbidity. Small parapneumonic effusions may not be associated with additional symptoms besides those of the underlying disease, while large tuberculous effusions (frequently evolving over weeks), may not necessarily provoke a commensurate severity of dyspnoea. The older child may complain of (pleuritic) chest pain on the affected side, or if the central diaphragm is affected, may have referred shoulder or back pain. Pain may be worsened by straining, coughing, and deep breathing. Occasionally, the child may present with abdominal pain and other clinical features of acute abdomen. Indeed, such children (in whom the diagnosis can easily be missed, may be inadvertently subjected to unnecessary laparotomies for a mistaken diagnosis of possible appendicitis!

The physical findings of pleural effusion also depend on the amount of fluid, and of course, the primary morbidity. At the stage of dry pleurisy, a rough friction rub may be heard at inspiration. As a reflex mechanism of minimizing the associated pleuritic pain, the breathing may be shallow, and the child may lie on the affected side at the early stage. Grunting respiration, a *modified Valsalva manoeuvre*, apparently constitutes yet another reflex respiratory guarding to

minimize the pleuritic pain of pleurisy, and probably to enhance gaseous exchange by increasing end-expiratory alveolar pressure. As fluid accumulation progresses, thoracic excursions may be visibly impaired on the affected side, and occasionally there may be fullness of the intercostal spaces. Also, in moderate to large effusions, findings include a tracheal/cardiac apex shift to the contralateral side, dullness to percussion (which may not necessarily be stony in children in view of the smaller thoracic dimensions), decreased tactile fremitus and vocal resonance, as well as diminished or absent breath sounds. In neonates and infants however, the breath sounds may be deceptively loud and clear, or in some cases, of bronchial quality because of the small thoracic volume. Except in loculated effusions, the above signs may shift with changes in posture. Other features of respiratory import include cyanosis in severe disease, tachypnoea, chest wall retractions and orthopnoea. Additional findings suggestive of an underlying pneumonia like crepitations may also be evident.

Investigations

The aims of investigations in pleural effusion comprise confirming the presence of fluid in the pleural space, identifying the size and nature of the fluid, the co-morbid intra-thoracic lesions, as well as the microbiological characteristics of the fluid.

Confirming the diagnosis: A simple chest radiograph (postero-anterior and lateral) constitutes an invaluable investigative tool for confirming the presence of pleural collection, and identifying co-morbid intrathoracic lesions like pneumonia, pneumothorax and other air leak morbidities. The radiographs should be done with the child in the supine and upright positions to identify a shift of the fluid collection, as is the case in a free-flowing effusion (**Figures 129.1A and 1B**). This feature is usually absent in loculated effusions. With a small effusion of early disease, there is obliteration of the costo-phrenic and/or cardio-phrenic angle(s), with the fluid layer sometimes extending to the lateral chest wall.

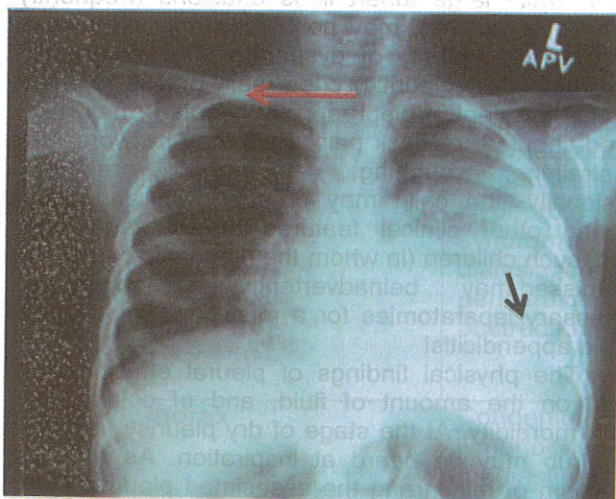


Figure 129.1A. Anteroposterior (AP) chest radiographs showing a massive left sided effusion and contralateral tracheal (mediastinal) shift.

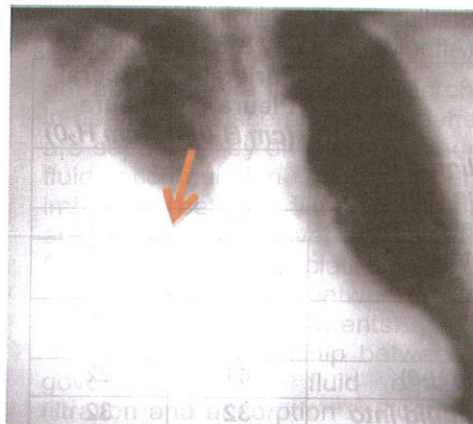


Figure 129.1B. Anteroposterior (AP) chest radiographs showing a classical massive right sided effusion and a (positive) ipsilateral meniscus sign.



Figures 129.1C. Alateral decubitus view of a chest radiograph, showing 'layering' of the fluid on the lateral chest wall.

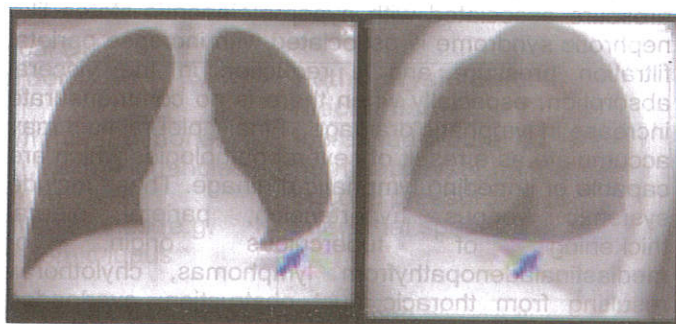


Figure 129.1D: Upright PA and lateral chest X-rays showing a dependent opacity with lateral upward sloping of a meniscus-shaped contour (meniscus sign), indicating a left-sided pleural effusion. Approximately 175mL of pleural fluid is required to produce a detectable blunting of the lateral costo-phrenic angle on a frontal view, while only 50mL is needed to blunt the posterior costo-phrenic recess on a lateral chest radiograph.

A lateral decubitus exposure is invaluable in demonstrating the presence of such small effusions in particular and the size of the fluid collection in general (**Figure 129. 1C**). A greater amount of fluid will appear as a dense homogenous opacity in the affected hemithorax, which is continuous with the diaphragmatic and mediastinal densities (**Figures 129.1A and 1B**). The

upper border of this opacity is concave as shown in **Figures 129.1B & 1D**, and may extend as far as the axilla; this is called the "meniscus sign". Varying degrees of lung collapse (with the characteristic jib-shaped shadow) and a contralateral shift of the mediastinum constitute the additional radiographic findings in massive effusion as shown in **Figure 129.1A**. Mediastinal shift may however not be evident in a sizeable loculated pleural fluid collection. On the lateral decubitus radiograph, a loculated fluid appears as semi-circular opacity on the lateral chest wall, and/or the para-vertebral gutter (**Figure 129.1C**). Other varieties of pleural effusion with fairly distinctive radiographic features are termed atypical effusions, and these include interlobar effusion (which appears as an elliptical opacity along the affected interlobar fissures, and an apparent widening of the septae along the mediastinal pleura (**Figure 129.2A**), and sub-pulmonic effusion, which simulates an apparent elevation of the affected hemi-diaphragm (**Figure 129.2B**). Ultrasonography is another useful, but hardly more informative imaging modality (than a chest radiograph) in suspected pleural effusion (See **Figures 129.3A and 3B**). Also, computerized tomography (CT) scan constitutes a useful investigative modality for differentiating (persistent) effusion from pleural thickening, or pneumonic densities (**Figures 129.4A and 4B**). In addition, the placement of a thoracentesis tube is better under ultrasonographic guidance and continuous screening device, if available.



Figures 129.2A. AP chest radiograph, showing loculated effusion, as a well-defined elliptical opacity with pointed margins.



Figures 129.2B. Sub-pulmonic effusion, presenting as an elevated diaphragm (pseudo-diaphragmatic contour) is illustrated in the dependent part (dark-blue arrow).



Figure 129.3A. Chest ultrasound showing anechoic pleural effusion. The blue arrow points to the visceral pleura.



Figure 129.3B. Chest ultrasound showing echoic pleural effusion. The blue arrows point to the visceral pleura.

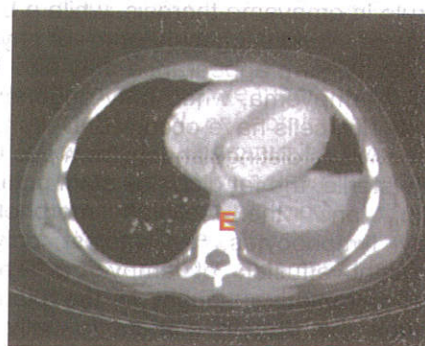


Figure 129.4A. Computerized tomography (CT) scan of the chest, showing a left-sided pleural effusion (E), appearing as a hypodense crescent shaped opacity

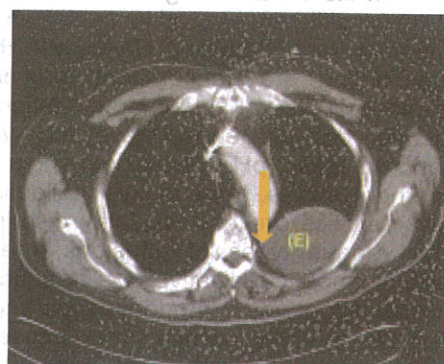


Figure 129.4B. Computerized tomography (CT) scan of the chest, showing a loculated left-sided, pleural effusion (E).

Identifying the presence and nature of the fluid (thoracocentesis and pleural fluid analysis): Thoracocentesis comprises inserting an appropriately sized needle (attached to a 20mL syringe) into the interspace, below the point of maximal dullness, using the infero-medial angle of the scapula on the affected side (confirmed by an earlier radiograph) as the landmark. The needle is preferably inserted immediately above a rib to avoid the inferiorly located large intercostal vessels, and ensuring a continuous pull of the plunger as the needle goes through the parietal pleura. Although, thoracocentesis is essentially a diagnostic procedure, it is advisable to evacuate as much fluid as possible especially when a prompt surgical tube insertion and closed thoracotomy tube drainage cannot be ensured, as is the case in the less privileged tropical communities. In children, the volume of evacuated fluid should however not be more than 800-1000mL, since rapid evacuation may cause a *re-expansion pulmonary oedema* on the affected side.

Besides confirming the presence of fluid in the pleural space, thoracocentesis and pleural fluid analysis constitute a prerequisite for differentiating the various causes of pleural inflammation (associated with true pleural effusion), from other causes of pleural fluid accumulation like hydrothorax, haemothorax, and chylothorax. A clear coloured fluid suggests a transudative collection of hydrothorax, while a straw, amber coloured, sero-fibrinous fluid (sometimes slightly cloudy) may indicate a parapneumonic exudative effusion, frequently associated with lobar consolidation. However, a purulent appearance (which may be thin or thick) is the rule in empyema thoracis, while a bloody or milky appearance would suggest haemo- or chylothorax respectively. A chyliform appearance may also be seen in long-standing empyema, when fatty degeneration of pus and endothelial cells have occurred. Despite similar appearances, chyle is distinguishable from pus because it is frequently sterile, the fat globules stain positive with Sudan stain, and the cellular morphology is predominantly lymphocytes, as against polymorphs seen in purulent effusion of empyema. The other discriminatory parameters sought in the pleural fluid analysis include the protein content (usually >3g/L in exudative collections, as against <3g/L in a transudate). Also, the specific gravity is >1.015 in exudative fluids and chyle, as against <1.015 in a transudate, while the glucose level is <2.2mmol/L (<40mg/dL) in exudative fluids, as against what obtains in a transudate and chyle, with the latter value approximating the concomitant plasma level. Other discriminatory pleural fluid characteristics include a high lactate dehydrogenase level (a value of >200IU/L) and an acidic (pH <7.20), both suggesting an exudative rather than a transudative collection. The cellular characteristics are no less fairly discriminatory between the various types of effusions; mesothelial cellular predominance is indicative of a transudate fluid (of hydrothorax), lymphocytic predominance suggests a tuberculous co-morbidity, while the presence of abundant polymorphs (with or without some erythrocytes), constitutes the cytological characteristic of exudative fluid collection. Malignant cells may also be

seen, but this is very rare in paediatric pleural fluid, although it is a conceivable feature in pleural effusion complicating metastatic Wilm's tumour.

Microbiological characteristics of the fluid: The initial Gram staining of the fluid may be positive for the causative bacterial agent in the exudative fluid of parapneumonic effusion/empyema. A lower microbiological yield of pleural aspirate compared with the blood culture, especially with staphylococcal empyema was a common observation in the South-west Nigerian series of community acquired pneumonia, some of which were para-pneumonic effusions. This may be attributable to the reportedly low pathogen count associated with pleural or ascitic fluid in general. Inoculating such fluids directly into blood culture bottle, as against sending same in sterile containers has been suggested as a useful strategy to significantly improve the microbiological yield of para-pneumonic effusions. Counter-current immunoelectrophoresis (CIE) of pleural fluid is an invaluable (microbiological) investigative tool for detecting bacterial antigens like those of *Staphylococcus aureus* and *H. influenzae*, and hence providing a presumptive aetiological basis for the initial choice of antimicrobials. An acid fast stain such as Ziehl-Neelsen stain may be required to identify an acid and alcohol-fast bacillus, and therefore confirm the tuberculous origin of the fluid. A subsequent culture and sensitivity may yield useful microbiologic data which may in turn provide invaluable guide for subsequent choice of alternative antimicrobials, should the initial empirical choice fail.

Others: These include a haematocrit and blood culture which in purulent effusion, may confirm the frequently co-existing anaemia and septicaemia respectively. The full blood count shows leucocytosis with polymorphonuclear predominance, and the erythrocyte sedimentation rate is usually high. Finally, a biopsy of the parietal pleura may be indicated in some children with suspected tuberculous aetiology (~60% of which can be detected histopathologically), or rarely in suspected pulmonary malignancies (e.g. primary pleural mesothelioma). This is preferably undertaken at the time the thoracocentesis is carried out using a cutting needle like Abrams, Cope, Harefield or Vim-Silverman needle. Pleural biopsy is however best avoided when there is minimal pleural fluid to avoid lung damage.

Course and Complications

Except for purulent effusion or empyema (vide infra), tuberculosis and the dyscollagenoses, in all of which a protracted course is expected, majority of exudative effusions, especially para-pneumonic serous/sero-fibrinous varieties are expected to resolve with treatment/resolution of the primary lesion. Although pleural thickening may be detectable radiologically long after the absorption of the fluid, long term respiratory morbidity from the inevitable but limited pleural adhesions is rare. Atelectasis, hypoxaemia and heart failure are usually transient in non-purulent effusion, but iatrogenic complications from diagnostic thoracocentesis and or chest tube insertion may include haemothorax, pneumothorax and other air-leak syndromes.

Treatment

The principles of management of pleural effusion comprise specific treatment of the underlying disease, evacuation of the fluid in moderate to large effusions, and supportive care including the frequently overlooked alleviation of pain.

Specific treatment of the underlying disease:

The administration of effective antimicrobials to treat the underlying pneumonia (initially chosen empirically while the results of microbiological studies are awaited for a possible subsequent modification or change), constitutes the best known specific therapy for parapneumonic pleural effusions. Tuberculous effusion will benefit from antituberculous agents chosen according to the national guidelines. The use of steroids in protracted tuberculous effusion is considered adjunctive by some authors. Anti-inflammatory agents, especially the non-steroidal agents are useful in pleural effusion associated with collagen vascular diseases like rheumatoid arthritis.

Evacuation of the fluid and other surgical measures:

In moderate to large effusions, this is essentially achieved via a closed thoracostomy tube drainage (CTTD), although it is also expedient to drain as much as possible (but not >1 litre) at the time of the initial diagnostic thoracocentesis. Small effusions (i.e. ≤ 10 mm on the lateral decubitus) do not need evacuation as reabsorption is expected with treatment of the underlying disease. The aim of evacuation in large effusions is to ameliorate the associated respiratory embarrassment and make the child comfortable. Indeed, the outcome of an effective drainage on the child's breathing is instantly rewarding. Repeat thoracocentesis or protracted under-water sealed tube drainage is rarely required except in empyema. Indications for CTTD include the moderate to large collections, rapidly progressive disease course, severe respiratory distress, purulent appearance of the pleural fluid at thoracocentesis, and identification of exudative characteristics in the pleural fluid except probably the presence of leucocytes (see above). Other features include an acidic fluid (pH < 7.2) with a low glucose of < 2.2 mmol/L (i.e. < 40 mg/dL), lactate dehydrogenase > 1000 IU/L and positive bacterial identification on Gram's stain or culture.

Rarely, in some empyema with a protracted clinical course, and a complicating recalcitrant broncho-pleural fistula, pleural decortication, thoracoplasty, or open flap drainage may be required to achieve a significant resolution. Associated surgical pericardiocentesis may be required in children with a contiguous pericardial collection.

Supportive care including alleviation of pleuritic pain:

These include giving analgesics (paracetamol) to alleviate the pain associated with diagnostic thoracocentesis and / or intra-pleural tube placement. One or more of the provision of supplemental oxygen to ameliorate hypoxaemia, treatment of the associated congestive heart failure, judicious fluid administration (being mindful of the possibility of the syndrome of inappropriate ADH secretion [SIADH]), as well as the provision of calorie dense feeds may also be required

as part of the supportive care in childhood pleural effusion.

EMPYEMA THORACIS (PURULENT PLEURISY, PYOTHORAX)

This is defined as the presence of (frank) pus or purulent fluid in the pleural cavity. Empyema remains the commonest variety of exudative pleural fluid accumulation in tropical paediatric practice, and indeed, one associated with the greatest frequency and number of complications.

Aetiology and risk factors

In tropical children, the most frequent underlying respiratory morbidity in empyema is bacterial pneumonia associated with necrotizing pathogens especially *Staphylococcus aureus*. Other bacterial pneumonias associated with paediatric empyema in the tropics include those caused by Gram negative agents like *Klebsiella pneumoniae*, *H. influenzae*, *Pseudomonas* and *Proteus* spp., as well as coliforms. Additionally, several anaerobes like the *Microaerophilic streptococcus*, *Fusobacteria nucleatum*, *Bacteroides* spp. and *peptostreptococcus* have been associated with non-tuberculous empyema in children with altered consciousness, dysphagia and conditions associated with potential aspiration of oropharyngeal secretions and the mouth flora contained therein. Furthermore, empyematous complication of a tuberculous effusion (an early feature of primary pulmonary tuberculosis) remains a continuing reality in tropical communities requiring a double pronged specific therapy. Indeed, in the tropics, a slower than expected resolution of empyema following adequate specific antimicrobial therapy, should always prompt a proper work-up for possible tuberculosis.

In contrast to what obtains in the less privileged tropical communities, community acquired pneumonia associated with *Streptococcus pneumoniae* appears to be the commonest co-morbidity in the developed countries. This followed the drastic reduction of *H. influenzae* type B (Hib) – associated invasive diseases, occasioned by the widespread use of Hib immunisation in developed countries. Apart from pneumonia, some cases of empyema may follow other intra-thoracic lesions like an intra-pleural rupture of pulmonary abscess, and rarely mediastinitis. The condition may also follow a thoracic extension of an intra-abdominal abscess.

Several epidemiological risk factors, some of which abound more in the less privileged tropical communities have continued to sustain the morbidity burden of 'empyema thoracis' in most tropical paediatric practice. These risk factors include a young age (especially infancy) with the well-known peak incidence in the second half of infancy; underlying malnutrition, identifiable in up to 75% of affected children; and a recent, or co-existing measles infection which was evident in up to 30% in a fairly large series from South-western Nigeria.

Pathogenesis & Pathology

Regardless of the inciting process, pleural inflammation is initially associated with the accumulation of a minimal quantity of fibrin-rich exudate and a few inflammatory cells (dry pleurisy). Even in a para-pneumonic effusion of bacterial pneumonia, this initial material is sterile. The subsequent increase in the permeability of pleural capillaries causes a commensurate worsening of the pleurisy, and a corresponding increase in the quantity of fibrin-rich (exudative) effusion. Together with the high protein content of the fluid, the resultant increase in pleural fluid oncotic pressure enhances further liquid accumulation in the pleural space (pleurisy with effusion). In empyema, or purulent pleurisy (for which the inciting process is almost always infective), the stage of pleurisy with effusion is followed by a fibrino-purulent phase, when there is a considerable efflux of polymorphonuclear cells and bacteria into the pleural cavity. At this stage, there is a tendency for the effusion to loculate extensively in one or both pleural cavities, and there is a considerable thickening of the parietal pleura. In empyema, a characteristic thick and purulent fluid is likely to be obtained at thoracocentesis. In the absence of a prompt therapeutic evacuation of the fibrino-purulent fluid, an inelastic membrane (pleural peel) is formed by fibroblast proliferation on both pleural surfaces. Apart from an inevitable atelectasis that follows moderate to severe amount of fluid accumulation, the fluid may burrow through the pleura into the lung parenchyma to form a broncho-pleural fistula and pyopneumothorax, or through the adjoining pericardium to form pyopericardium. Rarely, dissection of the pus into the chest wall (bone or soft tissue) may result in osteomyelitis of the rib, or "empyema necessitans", while a diaphragmatic dissection into the abdominal cavity may result in peritonitis. Of all these possibilities, a complicating pneumothorax (as pyopneumothorax) is the commonest, and may be followed by various air-leak morbidities including interstitial or subcutaneous emphysema (especially in post measles cases), pneumo-mediastinum, pneumo-pericardium and pneumo-peritoneum. These air-leak syndromes are further discussed under pneumothorax.

Clinical Features

Symptoms include the persistence of the fever, cough and breathlessness, associated with bacterial pneumonia in an infant or an older child, or one with a recent measles infection. At the time of presentation, symptoms would have lasted for days or weeks, but the parental concern is usually that of the worsening breathlessness. Paradoxically, the degree of prostration and severity of respiratory distress associated with empyema is worse in the older child, in whom, unlike in infants, chest pain may be a prominent symptom. Infants and toddlers may present with non-respiratory symptoms like vomiting and refusal of feeds/anorexia. Abdominal pain is also an occasional but noteworthy non-respiratory symptom of empyema in young children.

In addition to the physical findings highlighted under "clinical features" of pleural effusion above, pallor may

be evident at presentation, while features of moderate or overt malnutrition, and or the typical post-measles desquamation may also be evident. The older child with empyema thoracis usually more toxic looking. Some affected children may have features of superficial infections associated with *Staph. aureus* like impetigo or furunculosis. The import of grunting respiration as a prominent clinical parameter of pleural effusion or empyema thoracis, particularly in the resource-poor health facilities of tropical Africa, had been pointed out in an earlier overview of clinical parameters of ALRI in under-five children in Ibadan, South-western Nigeria. Fast breathing, chest in-drawing, and other features of the underlying severe pneumonia are equally common presenting features. Also, tachycardia and the other features of a complicating congestive cardiac failure are common in infants and toddlers. Tracheal shift and the other respiratory findings associated with moderate to massive pleural fluid collection may be identified as highlighted above. It must however be noted that loculated collections are common in empyema, and hence the physical and radiographic findings are unlikely to change significantly between the erect and supine positions. Also, it is equally noteworthy that the propensity for a mediastinal shift occurs at a smaller volume of fluid collection in empyema than in the serous, sero-fibrinous or transudative variety.

Investigations

The radiological and microbiological investigations undertaken in a child with suspected empyema are as highlighted above for pleural effusion in general. In some tropical facilities with limited microbiological support, soft radiological clues of possible aetiology include the presence of pneumatoceles, suggesting a possible staphylococcal aetiology, or perhaps that of other necrotizing bacterial agents. Indeed, an earlier report by Aderole *et al* from South-western Nigeria had suggested that a combination of pneumatoceles with a bulging interlobar fissure is commonly associated with *Klebsiella pneumoniae* aetiology. However, a clinical diagnosis of empyema can only be confirmed with a bedside diagnostic thoracocentesis, which is expected to yield a purulent (pleural) aspirate. Despite the usual low yield of a positive bacterial culture associated with pleural culture, a full bacteriological study (particularly microscopy culture and sensitivity) of the pleural pus must be pursued to identify the offending pathogen, and to guide a subsequent antimicrobial choice, should the initial empirical choice fail. Against the background of the high frequency of a complicating septicaemia, blood culture, in our experience, remains a rewarding (with a positive yield in over 60%) but frequently underutilized microbiologic tool for identifying the bacterial aetiology of pleural effusion in children. With regard to the biochemical characteristics of the pleural aspirate in empyema, the fluid has all the characteristics of an exudate {including a glucose of $<2.2\text{mmol/L}$ ($<40\text{mg/dL}$), protein of $>30\text{gm/L}$ ($>3\text{mg/dL}$), $\text{pH} < 7.2$ and $\text{LDH} > 1000\text{IU/L}$ } as highlighted above. Gram staining of pleural fluid may prove positive for bacterial agent(s), while pleural fluid neutrophils may exceed

100,000 cells/ μ L. Normochromic normocytic anaemia, leucocytosis with polymorphonuclear predominance and a high erythrocyte sedimentation rate constitute the common haematological findings in children with empyema thoracis. Mantoux test, gastric aspirate, sputum or bronchoscopic washings for Acid and Alcohol Fast Bacilli (AAFB) may be required for identifying or excluding an underlying primary pulmonary tuberculosis.

Complications

The common complications/co-morbid conditions in empyema thoracis can be categorized into general/systemic and intra-thoracic complications. General/systemic complications include severe anaemia, septicaemia, (extra-thoracic) osteomyelitis, septic arthritis, and pyogenic meningitis. Intra-thoracic complications and extensions include broncho-pleural fistulae, pneumothorax, interstitial/subcutaneous emphysema, and *empyema necessitans*, a condition characterized by the dissection of the purulent pleural collection through the soft tissues and the skin of the thoracic wall, simulating a discharging sinus or ulcer on the chest wall. *Empyema necessitans* frequently follows a bronchopleural extension of empyema following a peripheral necrotizing lung infection, or a lung abscess. Additional complications include lung abscess, purulent pericarditis, osteomyelitis of the rib(s) from a contiguous extension, and rarely, rupture through the diaphragm which may culminate in peritonitis.

Prognosis/Natural History

With adequate treatment, the disease outcome for most cases of empyema is good. Although, pleural thickening may persist for long, even after the disappearance of the fluid, and may indeed be mistaken for persistent collection or re-accumulation. Radiological resolution (with complete lung re-expansion) is to be anticipated in all but a few, usually within 3-4 months post-discharge from the hospital. However, a protracted in-patient care, usually for not less than 3-4 weeks is almost always required in all. This is partly because of the need for adequate chest tube drainage (especially in the well-known propensity for obstruction, or mal-positioning of the drainage tube), the tendency for the fluid to loculate, need for prolonged parenteral systemic antimicrobials (in view of the associated septicaemia), and indeed, the need to treat some of the several complications of the disease highlighted above. Some of these (e.g. tension pneumothorax), may be potentially life-threatening. Although, the presentation in non-staphylococcal empyema (e.g. that complicating pneumococcal pneumonia), may be less severe when compared with the stormy course of staphylococcal disease, the duration of inpatient therapy is unlikely to be significantly shorter than 2-3 weeks. Long term respiratory morbidity like restrictive lung disease is exceedingly uncommon with the current management strategies as detailed below. Associated mortality is low, but when it occurs, it can be ascribed to severe hypoxaemia/respiratory failure, anaemia, and indeed an underlying severe malnutrition.

Treatment

As is the case with exudative pleural fluid accumulation of a serous or sero-fibrinous nature, the elements of treatment comprise:

- Treatment of the underlying inciting process/disease.
- Prompt surgical evacuation of the purulent fluid from the pleural cavity.
- Supportive measures including alleviation of pain and measures aimed at enhancing a more effective drainage of loculated pleural pus.
- Invasive, but only occasionally-required cardiothoracic surgical interventions.

Treatment of the inciting infective disorder: For most cases of empyema, the treatment of the primary staphylococcal or other forms of necrotizing pneumonia is achieved by the administration of effective systemic antimicrobials, preferably guided by a subsequent in-vitro sensitivity profile of the offending pathogens. Initial antimicrobial choice is based on the known epidemiological pattern of (complicated) bacterial pneumonia in the environment, clinical data including the presence or otherwise of risk factors/predisposing clinical scenarios like the propensity for aspiration pneumonia), as well as the pharmacological properties of the empirical antimicrobial agents. Adequate coverage for Gram-positive and Gram-negative agents like *S. aureus* and *Klebsiella* spp. are required for most tropical cases of empyema, and hence the initial choice of cloxacillin, a semisynthetic penicillin (or ampicillin-cloxacillin fixed combination) combined with an aminoglycoside like gentamicin. The choice of an anti-pseudomonas, anti-proteus or anti-anaerobic agent(s), is determined by the presence of clinical risk factors of the relevant pathogens [e.g. a green pus which yields pseudomonas species on culture, culture of the pathogen from pleural fluid or wound swab/or superficial sinus in those with a concomitant *empyema necessitans*, an unconscious child or one that had been confined to bed for a while and hence the risk of aspiration of mouth flora anaerobes], and the antibiotic sensitivity pattern. Such cases may benefit from or carbenicillin, or ceftazidime (for *Pseudomonasaetiology*), quinolones (as reported recently for cases of *Proteus* species-associated empyema thoracis with *empyema necessitans*), or penicillin G, chloramphenicol, or metronidazole (for cases with suspected anaerobic aetiology).

Effecting a prompt drainage of the pleural fluid: The initial thoracentesis should attempt to drain as much as possible (but not >1L per session), especially if the fluid is massive and a prompt chest tube drainage cannot be ensured. For most non-loculated effusions, the seventh postero-lateral intercostal space is dependent enough for effective needle aspiration, which may be repeated on re-accumulation, provided there is no associated pneumothorax (pyopneumothorax) associated with broncho-pleural fistula. Chest tube drainage is mandatory in pyopneumothorax, empyema thoracis (with broncho-pleural fistula), and other exudative effusions in order to:

- prevent significant restriction of pulmonary function;

(ii) prevent an undue prolongation of the septic process, and;

(iii) avoid a fibrotic incarceration of the lung in a state of partial collapse.

As much as practicable, the largest soft rubber catheter available should be inserted into the intercostal space (using local anaesthesia), after cutting several additional holes near the tip, in order to sustain drainage, even after blockage of the terminal orifice. After securing the catheter to the chest wall, it is connected to an underwater drainage-suction system, applying a negative pressure of 15-18 cm H₂O at the catheter end. In loculated empyema, repositioning of the tube, or indeed, additional tubes may be required. If the antimicrobial treatment is adequate, removal of the tube should be possible within 2-3 weeks of in-patient care, but the presence of broncho-pleural fistula and pneumothorax may dictate withholding the removal of the tube till there is cessation of bubbling of the underwater segment of the drainage system. A pre-extubation clamping for 24 hours, and a post-extubation radiograph would be required to ascertain true closure of the broncho-pleural fistula.

Supportive measures: Of these, alleviation of pain is probably one of the most easily overlooked, but nevertheless essential supportive measures. Simple measures when the pain is mild/moderate include local heat and oral acetaminophen. The severe sharp pain associated with breathing (which appears to be characteristic of the stage of dry pleurisy) may benefit from an immediate splinting of the involved hemi-thorax (by making the child lie on the side), administering more powerful analgesics like codeine, or a non-steroidal anti-inflammatory agent. Topical ethyl chloride and 10% intravenous calcium gluconate may provide alternative analgesia and ameliorate the muscle spasm. A permanent relief of the pain usually comes with the advent of significant effusion (sero-fibrinous pleurisy), usually after the stage of dry pleurisy.

Other supportive measures include bed rest, and for the hypoxaemic child with restlessness, chloral hydrate in sedative dose (25-30mg/Kg), supplemental oxygen, and depending on the presence of features of heart failure, an anti-failure regimen and diuretics. Also, intrapleural instillation of streptokinase or similar agents for 5 days may be beneficial in loculated fibropurulent or organizing effusions. Finally, oral fluids and feeds may need to be augmented to offset the increased insensible loss, and to provide the extra caloric demands of a high fever.

Invasive, but only-occasionally-required cardiothoracic interventions: These include open-flap drainage, pleural decortication (using video-assisted thoracoscopic surgery), thoracoplasty or open thoracotomy. However, these interventions have been proved to be largely unnecessary in modern times, especially if the initial pleural fluid evacuation is prompt, and the choice of antimicrobial treatment is guided by microbiological data.

MANAGEMENT OF OTHER TYPES OF PLEURAL FLUID ACCUMULATION

Tuberculous Pleural Effusion

The features and other aspects of this aetiological category of pleural effusion are highlighted in greater details elsewhere in this text (See Chapter on Tuberculosis). Pleurisy with effusion is a common early complication of primary pulmonary tuberculosis, usually occurring in the school aged child, but it is equally not uncommon in infants and toddlers. Tuberculous pleurisy usually evolves from pleural rupture of a sub-pleural Ghon's focus, or sometimes as a pleural hypersensitivity to tuberculin. Often, the causative agents are scanty in the pleural fluid. Although the features of primary pulmonary disease may predate the onset of pleural inflammation of tuberculous origin, the presentation is usually acute with a dull chest pain (worse on coughing), breathlessness and limitation of respiratory excursions on the affected side. Massive effusions may be associated with bulging intercostal spaces, and may evolve in a clinical background of a relatively well child. The diagnosis of tuberculous pleurisy should be explored in any child or adolescent with pleural effusion who has a positive tuberculin reaction (even if the child looks relatively well), or one who is not responding to conventional antimicrobial agents and the usual supportive care/surgical drainage. An immediate diagnostic thoracentesis is required, and unless there is massive effusion with respiratory embarrassment, or tuberculous empyema, the volume of pleural fluid aspirated should not be more than 30mL and an "aliquot" of this should be sent for microbiologic and biochemical analyses. The fluid is usually serous or straw coloured, but may be purulent in tuberculous empyema, this usually occurring when there is a bronchopleural fistula. Whether serous or purulent, the characteristics of an exudate (with elevated protein and cellular contents, and a low glucose) are evident as detailed above. The cells, which may be as high as 200-1000/mm³ are however predominantly lymphocytes, except perhaps at the earliest stage of the disease. Microbiological identification (of *Mycobacterium tuberculosis*) from the pleural fluid is usually infrequently accomplished, but this should nevertheless be explored (with AAFB staining technique, and culture using Lowenstein-Jensen medium). A pleural biopsy may also be helpful in establishing the tuberculous nature of the pleural inflammation.

Apart from empyema, other complications include spontaneous pneumothorax. Also, extensive involvement of the pleura may occur in some children with fibro-caseous tissue obliteration of the pleura and a consequent protracted disease course. Pleural thickening and calcification may ensue and rarely, scoliosis may be associated with pleural adhesions and contraction of the hemi-thorax.

Treatment is aimed at achieving an early resolution of the effusion, reducing the risk of long term (restrictive) respiratory morbidity, and the need for invasive cardiothoracic surgical intervention. A prompt initiation of specific therapy with anti-tuberculous agents (in accordance with the national TB treatment guidelines), along with an oral corticosteroid (prednisolone, 1mg/Kg in three divided daily doses should be initiated. The latter should be continued for as long as it takes the

effusion to resolve, after which the dose is tapered). It is generally agreed that steroids hasten the disappearance of the effusion and control of fever, but should not be initiated before starting the specific anti-tuberculous agents. As is the case with other types of pleural fluid accumulation, a massive tuberculous effusion will obviously require drainage at the acute phase.

Hydrothorax

This is the designated terminology for the accumulation of pleural fluid of non-inflammatory origin, in which the fluid has the characteristics of a transudate (i.e. specific gravity of <1.015 , protein, 30gm/L ($<3\text{gm/dL}$), $\text{LDH}<200\text{IU/L}$). The common causes and basic pathophysiological mechanisms of a transudative pleural fluid accumulation are highlighted in the table above. Basically, transudation across the pleural surfaces occurs when the alteration of the Starling's forces of mechanical and hydrostatic pressures favour liquid filtration in excess of absorption. It is interesting to note that despite the low plasma oncotic pressure associated with the hypoproteinaemic states of kwashiorkor and marasmic-kwashiorkor, hydrothorax and ascites are rare clinical occurrences in these conditions. The clinical manifestations are essentially those of the underlying disorder (e.g. nephrotic syndrome, hepatic cirrhosis, congestive cardiac failure), while the chest findings of pleural fluid accumulation are as detailed earlier in this chapter. However, hydrothorax is more likely to be bilateral, and with a change in posture, the upper level of dullness to percussion is more likely to change more rapidly, compared with what obtains in exudative effusions like sero-fibrinous effusions and empyema. Also noteworthy in hydrothorax, is the tendency for "third space" or subcutaneous fluid collection elsewhere in the body such as ascites and facial puffiness to occur as is the case in nephrotic syndrome.

Investigations are directed towards confirming the presence and the nature of the pleural fluid, as well as identifying the primary disease. A chest radiograph and a diagnostic thoracentesis will provide an anatomic diagnosis and the pleural fluid analysis (of the thoracentesis fluid) will confirm the transudative nature as detailed above. Treatment comprises the specific therapy of the primary disease. In moderate to massive collection with significant cardiorespiratory disturbance, there is a need for a prompt evacuation of the fluid at the time of the diagnostic thoracentesis. The quantity of the fluid removed at a time, should however not exceed 1000mL to prevent post-drainage re-expansion pulmonary oedema. If there is a need for repeated thoracentesis in a child, a closed thoracostomy tube insertion is required to circumvent the otherwise inevitable tendency to upset the child with repeated needle aspirations. Judicious use of diuretics will slow down fluid re-accumulation, and may eliminate or reduce the need for thoracostomy tube drainage, or frequent thoracentesis. Small to moderate hydrothoraces associated with rapidly removable conditions like fluid overload, or peritoneal dialysis may resolve spontaneously after addressing the primary condition.

Chylothorax

This condition is usually the consequence of leakage of chyle from the thoracic duct into the pleural cavity. Majority ($>50\%$) occur as a complication of corrective cardiothoracic surgical interventions (including repair of diaphragmatic hernias), but others may result from enlargement of lymph nodes with compression/erosion of the duct, penetrating or crush injuries of the chest, metastatic intrathoracic malignancies, lymphangiomata and rarely, as a consequence of a congenital lymphatic fistula. Chylothorax may occur spontaneously in neonates, in whom the condition constitutes a common cause of pleural fluid accumulation.

The clinical features of chylothorax are essentially those seen in other forms of pleural fluid accumulation. Bilateral chylous effusion is rare, but the majority of cases are located on the left side. Diagnosis is confirmed with the identification of the frequently sterile chylous fluid which usually has a fairly characteristic milky-white appearance. A positive Sudan stain would confirm the presence of fat globules in the chylous fluid. Other features of a chylous pleural fluid include a total fat exceeding that of plasma, protein 30gm/L ($>3\text{gm/dL}$) and an elevation of the cellular contents, particularly lymphocytes. Chyle should be differentiated from chyloform pseudo-chylous fluid. The latter has a similar milky appearance, but unlike chyle, it does not clear when shaken with alkali or ether, and has an elevated cholesterol level, as against the high triglyceride level of chyle. Chyloform pseudo-chylous fluid usually results from fatty degeneration of pus and endothelial cells, usually from a long-standing serous effusion, or empyema.

Although spontaneous resolution may occur in more than half of infants with chylothorax, treatment is frequently required to address life-threatening cardiopulmonary embarrassment (via a prompt but single thoracentesis and complete drainage), prevent the consequent nutrient drain (via the provision of medium chain triglyceride (MCT) as the major dietary fat, cutting down on fatty meals, and providing high protein, and calorie dense diets), and effecting an immediate surgical repair (via a thoracotomy), in those cases resulting from thoracic surgical misadventure. The rationale for replacing fat with MCT is that it is absorbed directly into the portal venous system. Thus, it allows spontaneous closure to occur, usually within two weeks of the dietary replacement/restriction. Additional dietary provisions include the provision of multivitamins, especially the fat-soluble ones like vitamin A and D. Those with rapid re-accumulation (of the chyle) will benefit from fasting and concomitant parenteral hyperalimentation, using a vein different from the left subclavian. In difficult cases, the leaking thoracic duct may be located and ligated. For obvious reasons, parenteral alimentation is presently hardly favoured in most tropical health facilities. Similarly, novel approaches like controlled ventilation (to achieve a positive end-expiratory pressure), inhalation of nitric oxide, and the insertion of pleuroperitoneal shunts for recalcitrant cases are currently hardly feasible options in most tropical facilities.

Haemothorax

The presence of blood in the pleural cavity is referred to as haemothorax. Although uncommon in children, when it occurs, it usually follows blunt chest trauma, vascular erosion by intrathoracic space occupying lesions like tuberculous lymph nodes, empyema and neoplasms. Other possible antecedent lesions include sub-pleural rupture of a bleb, traumatic rupture or erosion into one or more of the great vessels (as is the case with patent ductus arteriosus, and aortic coarctation), arterio-venous malformations, strangulated diaphragmatic hernia, pulmonary sequestration, and rarely, from bleeding diatheses, anticoagulant therapy and blood dyscrasias. Spontaneous haemothorax may also occur in neonates. Especially following chest trauma, haemothorax may coexist with pneumothorax, in which case it is referred to as haemopneumothorax.

The respiratory clinical parameters are not different from those of other forms of pleural fluid accumulation, but the antecedent events like a recent chest trauma, as well as features of the pre-haemothorax disease states (e.g. patent ductus arteriosus or aortic coarctation) may be evident. The diagnosis is usually predicated on the findings at diagnostic thoracentesis (i.e. a bloody fluid at thoracentesis, provided the procedure itself is not traumatic). Besides confirming the nature of the pleural fluid, investigations are also expected to identify the cause, and the choice of these will be determined by the suspected underlying condition.

Expectedly, treatment is directed at addressing the cause, treatment of the associated shock with an immediate intravenous infusion of normal saline or similar plasma expanders, as well as the provision of analgesia. Adequate evacuation of the collection is required to forestall a subsequent restrictive lung disease due to fibrin organization. Direct surgical ligation of the actively bleeding vessels may be required at the acute phase. Intra-pleural instillation of fibrinolytic enzymes may be needed to dissolve clots, and rarely, decortication may eventually be required in an extensive but poorly evacuated haemothorax, particularly those that are associated with markedly impaired lung function parameters.

PNEUMOTHORAX

A condition is defined as the presence of extra-pulmonary air in the pleural space. Pneumothorax may be classified according to the presence or otherwise an underlying cause. Using this as the basis, the terminology "spontaneous pneumothorax", refers to an unexpected leakage of air into the pleural space without a preceding trauma. When the unexpected pleural leakage of air occurs in the absence of a preceding/associated lung or pleural disease, it is referred to as "primary spontaneous pneumothorax", while "secondary spontaneous pneumothorax" is one in which there is pleural air in the absence of trauma, but associated with a preceding/ lung or pleural disease. Other types of pneumothorax include post-traumatic pneumothorax (which follows a variety of chest trauma including penetrating wounds, crush injuries and rib fractures), "iatrogenic pneumothorax" (which often

follows diagnostic or therapeutic procedures like tracheostomy, thoracentesis, trans-bronchial biopsy and intubation/positive pressure ventilation), and "catamenial pneumothorax" (which occurs in association with menstruation).

Regardless of the cause(s), the severity of pneumothorax depends on the anatomic variety, of which three types are recognized. These include an open pneumothorax (in which the communication between the parenchymal rupture and the pleural cavity remain open, usually via a broncho-pleural fistula), a closed pneumothorax (in which the pleural breach closes after a short time with consequent absorption of the intra-pleural gas). In view of the need for prompt intervention, the most important anatomic variety is a tension (or valvular) pneumothorax, with or without a concomitant pleural fluid. This condition is a life threatening paediatric emergency in which the breach in the pleural surface acts as a valve allowing a unidirectional air movement into the pleural space during inspiration. The inability of the pleural air to escape during expiration causes a progressive rise in intra-pleural pressure above the atmospheric level with a resulting progressive atelectasis in the affected hemithorax, and a contralateral shift of mediastinal structures, and a possible compression of the great vessels.

By far the commonest type of pneumothorax seen in children is the secondary spontaneous pneumothorax, and indeed, in paediatric medical practice, the term pneumothorax used unqualified refers to secondary spontaneous pneumothorax. In the tropics, spontaneous pneumothorax rarely exists in isolation. It is commonly associated with purulent effusion or empyema (frequently complicating staphylococcal pneumonia or similar necrotizing pneumonias like Klebsiellapneumonia), and is then referred to as pyopneumothorax. Beyond the neonatal period, malnutrition and/or measles constitute the common comorbidities/risk factors of pneumothorax in the tropics. The association of pneumothorax with transudative pleural fluid collection (hydrothorax) is referred to as hydropneumothorax.

Aetiology & Pathogenesis

The permeability of the pleural membranes to gas, and the significant pressure gradient of ~68cm H₂O (which represents the difference between the total gas pressure of 73cm H₂O sub-atmospheric in venous blood and the intra-pleural pressure of 5cm H₂O sub-atmospheric) appear to favour a continuing absorption of gas from the pleural space into the circulation. Thus, the continuing diffusion of gas out of the pleural space, along the direction of this pressure gradient usually ensures that the pleural space is completely air free. Indeed, in a closed or loculated pneumothorax (which is devoid of a continuous air leak), breathing 100% oxygen hastens the absorption (~sevenfold) by increasing the nitrogen pressure gradient between pleural air and blood. The pathogenesis or pathological process of pneumothorax and other related air-leak syndromes like pneumo-mediastinum, pulmonary interstitial and subcutaneous emphysemas had long

been clarified by the 1944 classic report of Macklin and Marklin. Alveolar rupture and escape of air into the perivascular interstitium, occurs when a pressure gradient emerges between the alveolus and the interstitial tissue. This scenario is frequently seen in a clinical setting which generates a high inspiratory pressure (as is the case with intubation and aggressive resuscitation in newborns). Subsequently, the accumulated air in the perivascular interstitium may track along the 'vascular rays' to present clinically as (pulmonary) interstitial emphysema. On the other hand the air may track to the surface of the lung, to the mediastinum or indeed the skin with the resultant clinical entities of pneumothorax and pneumo-mediastinum and subcutaneous emphysema respectively. Thus, pneumothorax, pulmonary interstitial emphysema pneumo-mediastinum and subcutaneous emphysema are probably the clinical consequences of a single pathological process emanating from alveolar rupture, or as is the case with the necrotizing pneumonias like staphylococcal pneumonia, from a broncho-pleural fistula. Occasionally in children, pneumothorax may also follow the advent of a chest wall defect through the parietal pleura connecting the atmosphere to the pleural cavity. The latter route of pleural air accumulation results from blunt chest trauma, penetrating injuries, or rarely, an aggressive external cardiac massage.

In the newborn, spontaneous pneumothorax is a common occurrence. The relatively higher prevalence of pneumothorax among newborns has been attributed in part to the paucity of the pores of Khon, a 'defect' that hinders effective inter-alveolar pressure redistribution (following a surge in pressure in collateral alveoli). Similarly, the related propensity of neonates for developing high trans-pulmonary pressure across normally aerated portions of the lung (following perinatal occlusion of some airways from mucus, blood and squamous epithelial cells), may also account for the increased disease vulnerability of newborns. Furthermore, the development of spontaneous alveolar pressure (with the resultant multiple areas of atelectasis), which occurs frequently with neonatal hyaline membrane disease, may also be contributory in preterm infants. Whereas, pneumothorax generally tends to complicate difficult deliveries, or too vigorous positive pressure ventilation in the newborn, the pathogenesis of pneumothorax in older subjects appears to be disease specific. In necrotizing pneumonias, pneumothorax results from broncho-pleural fistula following liquefactive alveolar necrosis. Similarly, in cavitary or progressive primary tuberculosis, pleural air accumulation results from liquefaction of sub-pleural caseous infiltrates, with consequent (visceral) pleural membrane necrosis and rupture. Extrinsic tuberculous nodal compression of primary tuberculosis, metastatic or primary tumour compression may also cause a ball-valve obstruction with a resulting alveolar rupture. Rarely, a primary or metastatic pulmonary malignancy (as exemplified by a sarcoma) creates broncho-pleural fistulae by outgrowing their blood supply, becoming necrotic and rupturing the bronchus into the pleural space. In

asthmatic children, mucoid impaction and bronchial obstruction, with the resultant middle lobe syndrome and perhaps, the reportedly thinner visceral pleura in chronic asthma (with the consequent greater propensity for rupturing at near-normal transthoracic pressures) constitute the pathogenesis of alveolar rupture and development of pneumothorax in these children.

Table 129-IV highlights the common conditions associated with alveolar rupture and pneumothoraces in children. Regardless of the age or the cause(s), the elevation of intra-pleural pressure to a level above atmospheric (tension pneumothorax) causes ipsilateral atelectasis because the elastic recoil can no longer be counteracted by the outward pull of a negative sub-atmospheric intra-pleural pressure.

There is hyperinflation of the contralateral lung, and the greater force of retraction at expiration causes a mediastinal shift to the contralateral side with consequent venous compression and impedance of venous return to the heart. Hypoxaemia and hypercapnia may ensue. Ventilatory impairment results in a tendency to make a greater inspiratory effort (to generate sufficient negative pleural pressure to ventilate the normal lung), and this tends to worsen the tension pneumothorax and mediastinal shift. Compensatory tachycardia compromises the cardiopulmonary status further by decreasing venous return and ventricular output.

Table 129-IV: Conditions associated with alveolar rupture, pneumothorax and air leak (Modified from Montgomery & Sigalet, 2006)

• First breath
• Diagnostic and therapeutic manoeuvres
• Thoracocentesis
• Aspiration lung biopsy
• Percutaneous pleural biopsy
• Cardiothoracic surgery
• Resuscitation
• Ventilator therapy {especially with positive end-expiratory pressure [PEEP]}
• Lower Respiratory tract diseases
• Tuberculosis (especially miliary or cavitary)
• Hyaline membrane disease
• Aspiration syndrome
• Asthma (especially severe acute asthma)
• Bronchiolitis
• Pneumonia
• Pulmonary malignancies (primary & metastatic)
• Blunt thoracic trauma

Clinical Features

Perhaps the most important clinical tool for an early recognition of pneumothorax is a high index of diagnostic suspicion, whenever any of the following clinical scenarios (which constitute the risk factors of spontaneous pneumothorax) is identified. These include severe malnutrition, measles, staphylococcal or Klebsiella pneumonia, pertussis, bronchiolitis, bronchial asthma, progressive primary tuberculosis, hydatid

cysts, blunt chest trauma (with or without rib fracture), and in the newborn, a stormy delivery associated with birth asphyxia, requiring endotracheal intubation and aggressive resuscitation. Rarer antecedent scenarios in the tropical practice include metastatic intrathoracic tumours (e.g. a sarcoma). Cystic fibrosis is a common underlying morbidity in the Caucasians.

The presence or otherwise of the relevant clinical manifestations in a child with pneumothorax depends on the extent/size of the intrapleural air collection, whether or not it is expanding (as is the case with tension pneumothorax), the corresponding extent of lung collapse and displacement of mediastinal structures. Small to moderate pneumothoraces, with no shift of mediastinal structures, may present with little or no additional symptoms. On the other hand, the onset of symptoms in significant or severe pneumothorax is usually sudden, with the advent of, or exacerbation of breathlessness (associated with the underlying pulmonary morbidity), persistent irritating cough, and in older children and adolescents, as well as a sharp pleuritic chest pain. The latter is usually severe, and unilateral, but the severity of the pain does not reflect the extent of ipsilateral atelectasis. The pain may be referred to the shoulder tip on the same side due to the associated irritation of the diaphragmatic pleura. These features are usually superimposed on those of the underlying lung disease like necrotizing pneumonia and/or empyema, or the onset can be traced to a recent chest injury.

Physical findings are again absent in small to moderate pneumothoraces, but in severe pneumothorax with mediastinal displacement, the signs are usually predictable, and are generally more severe in those with tension pneumothorax. Large pneumothoraces may be associated with central cyanosis, chest wall retractions and other features of respiratory distress. If tachypnoea is a feature of the underlying disease, it is likely to get worse with the advent of pneumothorax, and the respiration more shallow with the accumulation of significant intrapleural air. Tachycardia is also a near universal feature of significant pneumothorax. Reduced chest movement/expansion of the affected hemithorax, tracheal and apical displacement to the contralateral side, hyperresonant notes to percussion and distant or absent breath sounds on the same side constitute the spectrum of expected chest findings. Other signs include the features of a coexisting pleural effusion with dull/flat percussion notes at the dependent part of the affected side. The dull percussion notes of effusion elicited, has a sharp transition to an area of hyperresonance representing that of pleural air collection. Amphoric breath sounds, and the so called "scratch sign" may also be elicited. The latter is a loud and harsh sound heard over the mid-sternum when the affected side is stroked with a finger or a dull instrument. Rarely, gurgling sounds (suggesting an open bronchopleural fistula) may be heard synchronously with the respiratory excursions.

Investigations

In high risk clinical scenarios, an early clinical recognition of pneumothorax requires an equally prompt investigative corroboration, as a prerequisite for initiating effective management. Diagnostic confirmation of pneumothorax is usually predicated on the radiographic findings of absence of lung markings between the well-defined edge of the collapsed lung and the chest wall. Radiographic confirmation is best sought early, when cardiorespiratory decompensation is minimal. An anteroposterior (expiratory) exposure, and one with the patient in the supine position (**Figures 129.5A and 5B**), as well as a horizontal beam, cross-table lateral view are considered particularly most informative in detecting relatively small pneumothorax. A large pneumothorax may show concomitant shift of mediastinal structures (**Figure 129.5B**). Besides confirming the presence of pneumothorax, chest radiographs are also invaluable in confirming the underlying pulmonary lesion(s) like pneumonia, with or without pneumatoceles, pleural effusion or a rib fracture. Furthermore, a chest radiograph may exclude other intrathoracic space-occupying lesions like parenchymal cavitary lesions, compensatory hyperinflation, lobar emphysema, lung cysts, and 'intrathoracic translocation' of an abdominal viscus (associated with diaphragmatic hernia). The latter can be confirmed with a radiographic evidence of thoracic location of an intragastric tube (with a radio-opaque tip), inserted before taking the radiograph. Serial chest radiographs are also invaluable, not only for monitoring the response to therapeutic interventions, but also for identifying a continuous air leakage associated with a tension pneumothorax. The latter is identified with the radiographic evidence of a worsening lung collapse and shift of mediastinal structures, occurring over a short period of time. However, some disadvantages of the chest radiograph include its inability to differentiate between a pneumothorax and either a large bulla, or an emphysematous lung. Also, a skin fold or the scapula may mimic a pleural line on the chest radiograph. A chest CT-scan is also currently considered a useful (radio-imaging) investigative tool in the management of pneumothorax. Indeed, a chest CT-scan is reportedly one of the most reliable radio-imaging modalities for confirming a diagnosis of pneumothorax (**Figure 129.5C**). Not only can a chest-CT identify multiple blebs and bullae, it can also differentiate bullae (especially large bulla) and an emphysematous lung from a pneumothorax. Regrettably, and presumably because of the poor availability and cost considerations, this investigative tool is hardly recommended for routine use in contemporary tropical practice.

Non-radiographic/imaging tools like intermittent or continuous ECG monitoring (with oscilloscopic display) are useful for detecting pneumothorax in high-risk situations, especially in neonates with meconium aspiration syndrome, hyaline membrane disease, and those on mechanical ventilation.

A rapid escape of air under pressure (frequently with the hissing sound) on diagnostic thoracocentesis, or insertion of a thoracostomy tube (into the second intercostal space), may be used to confirm a tension pneumothorax.



Figure 129.5A. Chest X-ray showing a hyperlucent left hemi-thorax suggesting a left-sided pneumothorax and ipsilateral lung collapse; Note the absence of lung markings in the affected hemithorax.

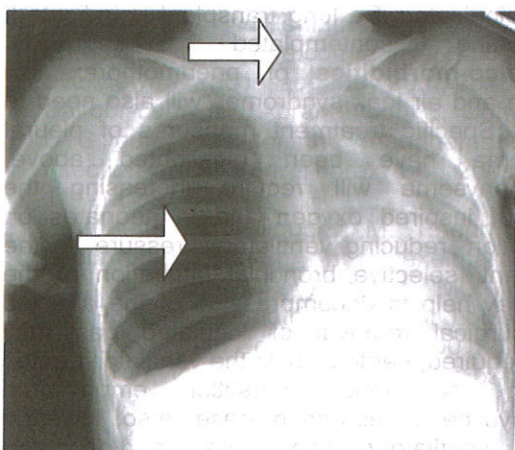


Figure 129.5B. Chest X-ray showing a classical chest radiograph in a child with a right-sided tension pneumothorax and tracheal (mediastinal) shift to left; the absence of lung markings on the affected right is also striking.

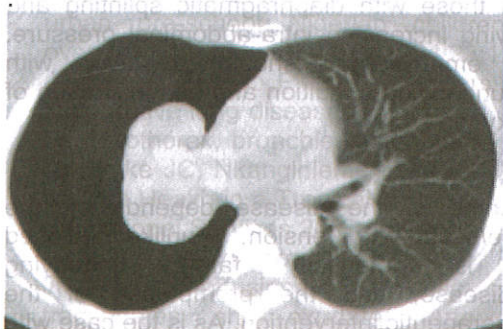


Figure 129.5C: Chest CT-scan, showing a classic right-sided pneumothorax; note the ipsilateral absence of lung markings

However, by creating a fistula between a misdiagnosed intra-parenchymal cavity and the pleural cavity, these two investigative procedures are associated with a small but definite risk of a complicating (iatrogenic) tension pneumothorax. Detection of pneumothorax can also be achieved with chest transillumination, using a powerful

fibreoptic light probe, but such equipment is hard to come by in contemporary tropical practice. Besides their value in confirming the diagnosis of pneumothorax and/or the presence of coexisting high-risk scenarios of the disease, these non-radiographic investigative modalities are also useful in monitoring disease resolution after initiating treatment.

Complications and associated clinical syndromes

The most important and indeed, potentially fatal complications of pneumothorax include tension pneumothorax (Figure 129.5C), worsening lung compression/collapse, severe mediastinal shift and a distinct possibility of kinking of the great vessels. An important co-morbidity/underlying condition of this potentially fatal condition is the underlying bacterial pneumonia, especially if this is due to necrotizing pathogens. In such cases, there is usually an exudative effusion, or empyema, alveolar rupture and broncho-pleural fistula. Other possible co-morbidities/progression of pneumothorax include several air leak syndromes like interstitial and subcutaneous emphysema, pneumo-mediastinum, pneumo-pericardium, pneumo-peritoneum, and pulmonary pseudocyst. An early identification of one or more of these clinical entities is essential because of the need for additional therapeutic measures, as detailed subsequently in this chapter.

Differential Diagnoses

These include compensatory hyperinflation (with contralateral lung collapse) diaphragmatic hernia, congenital lobar emphysema, congenital cystic lesions of the lung. However, the chest radiograph findings are sufficiently discriminatory between these diagnostic considerations. As highlighted above, a chest X-ray, especially one taken after passing intragastric tube with a radio-opaque tip, constitutes an additional discriminatory tool in identifying suspected diaphragmatic hernia from a pneumothorax.

Treatment

Whereas a small closed pneumothorax will almost invariably be reabsorbed within days, a large volume of intrapleural air, or one under a higher pressure than the atmospheric value as is the case in tension pneumothorax, may not only take much longer to reabsorb, but may also precipitate cardiorespiratory complications like respiratory failure (especially in subjects with underlying poor respiratory function), and a potentially fatal kinking of mediastinal structures. The major goal of treatment therefore, is to facilitate a rapid reabsorption of the pneumothorax. Hence, the treatment modality is determined by the size of the pneumothorax, whether or not it is recurrent, the presence of features of tension pneumothorax, as well as the related severity of the underlying disease and respiratory distress. In this regard, the size of the air of a pneumothorax is usually determined by the percentage of the hemithorax occupied. A conservative approach is favoured in the presence of a small closed pneumothorax (<5–10% in children, or 20–30% in adolescents and adults) in an otherwise stable patient. This treatment approach is favoured, especially if it is the first episode of

pneumothorax, the underlying disease unlikely to cause a recurrence (e.g. cystic fibrosis, or malignancy), and there are no features of tension pneumothorax. Conservative or medical treatment includes the treatment of the underlying condition (e.g. antibiotic therapy for Staphylococcal pneumonia), analgesia/sedation with a mild narcotic like codeine, frequent small volume feeds (especially in neonates), and a careful in-patient monitoring for signs of progressive cardiorespiratory deterioration (including blood gases or oxygen saturation). Depending on the "size" of the pneumothorax, as well as the cardiorespiratory status, administration of supplemental 100% oxygen may also be required to facilitate early reabsorption. As explained earlier, the latter hastens the reabsorption (of the pleural air collection) about seven-fold. Oxygen therapy is generally required in neonates, and those with potentially life-threatening underlying diseases, or one associated with significant hypoxaemia like hyaline membrane disease, meconium aspiration syndrome and severe acute asthma. It should however be used with caution in patients in chronic hypoxaemic states (in whom the hypoxic drive is crucial for sustaining respiration), and in preterm infants, in whom disease states of oxygen toxicity like retrolental hyperplasia may ensue. Tension pneumothorax, worsening severity of an open or closed disease variant, recurrence, an increasing radiologic size and failure to achieve a prompt resolution with time, may each indicate the need for a more specific or invasive therapeutic options like mechanical evacuation of the trapped air (via needle aspiration, using a one-way valve system, or an underwater sealed thoracostomy tube drainage system). Other indications for immediate closed thoracostomy tube insertion include a reduction in the volume by $\geq 50\%$ in a dyspnoeic child with a poor respiratory reserve. A one-way valve system is frequently sufficient to achieve evacuation in the older child with mild to moderate symptomatic pneumothorax of the spontaneous type. Those with underlying progressive pulmonary tuberculosis, acute asthma, metastatic malignancies and cystic fibrosis will require an underwater sealed drainage system. In recurrent pneumothoraces (e.g. in malignancies, cystic fibrosis or congenital lesions), alternative treatment options which may be explored (provided the relevant expertise and facilities are available), include an open but limited thoracotomy, or more recently thoracoscopy to effect a surgical closure of the underlying fistula, pleural stripping to ensure subsequent adhesions, and plication of emphysematous blebs.

A prompt mechanical evacuation especially to relieve tension pneumothorax can be achieved by direct needle aspiration at the second or third intercostal space (mid-clavicular or anterior axillary line), using an 18G needle and 20mL or 50mL syringe initially, and subsequently attaching a catheter. The latter is then connected to one of either a one-way flutter valve (Heimlich valve), or an underwater sealed drainage system with the distal end of the intra-pleural tube inserted not deeper than 2cm under water, while the proximal suction end is kept well above the water level. A suction pressure of -5 to -15cm H₂O should be ensured. Alternatively, a trocar-mounted semi-rigid Argyle catheter (preferably a French gauge 12) with several side holes for

better air evacuation may be introduced into the pleural space (using the same landmarks). A special care needs to be taken to avoid deep chest penetration and damage to the lung parenchyma and vessels. This latter option is preferable in neonates with moderate sized pneumothoraces and paucity of underlying lung (parenchymal) disease(s).

Non-surgical specific treatment options of a poorly resolving pneumothorax (with consequent failure of lung re-expansion) after a duration of 2-3 months, or recurrent pneumothorax include the intra-pleural instillation of agents like quinacrine hydrochloride in saline (100mg in 15 mL of saline). Less favoured agents in children include the local instillation of iodized talc, camphor in oil or tetracycline to induce adhesion between the lung and thoracic wall (chemical pleurodesis). The instillation of intrapleural agents, as well as stripping and abrasion of the pleural membranes are aimed at creating inflamed raw surfaces which heal by adhesions thus sealing the breach in the pleural membrane(s). Pleurodesis is however contraindicated if a lung transplant is indicated, or is being contemplated subsequently.

Complications/co-morbidities of pneumothorax like pleural effusion and air leak syndromes will also need to be addressed. Specific treatment measures of pleural effusion/empyema have been highlighted above. Interstitial emphysema will require increasing the concentration of inspired oxygen and in neonates on artificial ventilation, reducing ventilatory pressure. In the occasional infant, selective bronchial intubation of the normal side may help in decompression of the affected lung, while surgical removal of loculated gas may eventually be required, particularly in the ventilator-treated preterm with persistent interstitial emphysema complicating hyaline membrane disease. Also, lowering the inspired ventilatory flow rate, and/or pericardiocentesis to relieve pericardial tamponade may be necessary in those with a complicating pneumopericardium. Pneumo-peritoneum in a stable child is best treated conservatively by raising the concentration and flow of inspired oxygen, but abdominal paracentesis may be inevitable in those with diaphragmatic splinting and dyspnoea following increasing intra-abdominal pressure. Subcutaneous emphysema should resolve with addressing the underlying condition and administration of supplemental oxygen.

Prognosis

The outcome of the disease depends on the anatomic type/severity (tension, unlike closed pneumothorax is potentially fatal), underlying parenchymal disease, and the promptness of the diagnosis and therapeutic intervention. As is the case with other potentially fatal conditions, diagnostic anticipation of pneumothorax in high-risk clinical scenarios (detailed above), as well as the institutional preparedness for a prompt intervention (e.g. availability of a tray containing a 50mL syringe, a bottle of sterile saline, a three-way stop-cock, and a kidney dish in the emergency room and the neonatal intensive care unit) are crucial for achieving a successful disease outcome.

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