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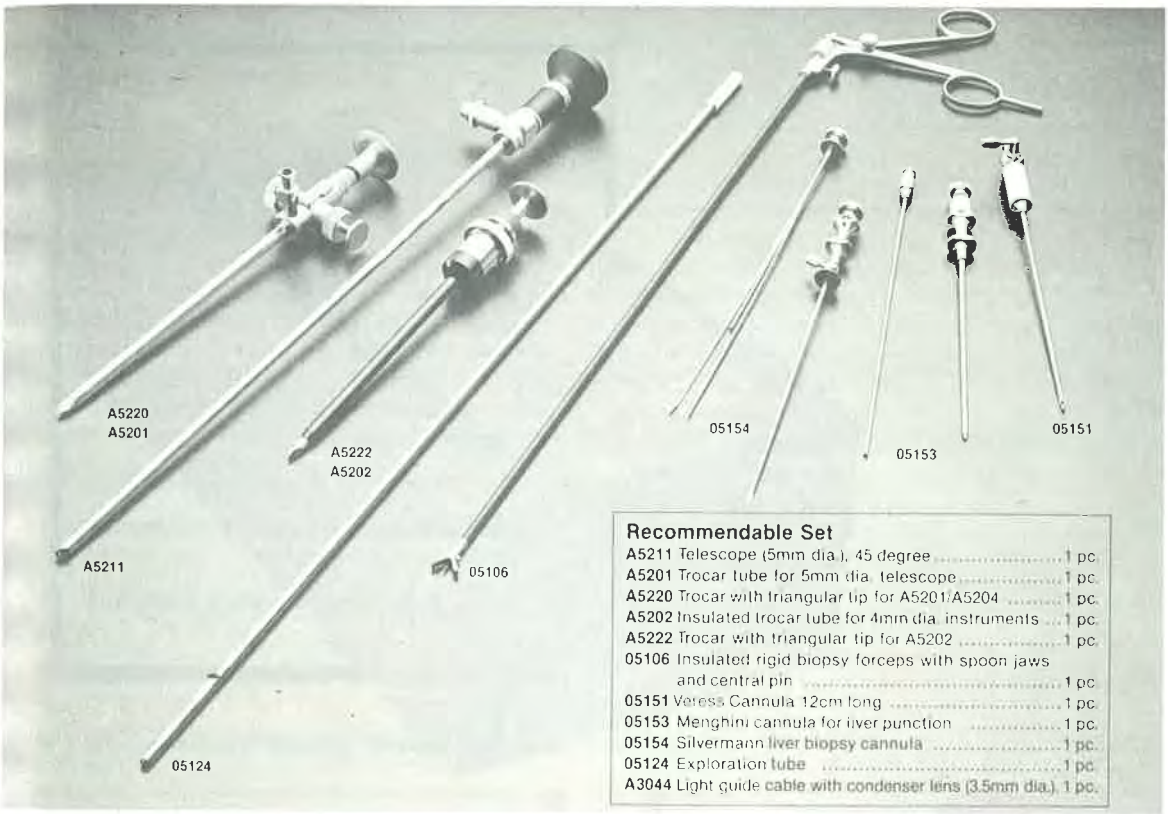
Laparoscope for Double Puncture



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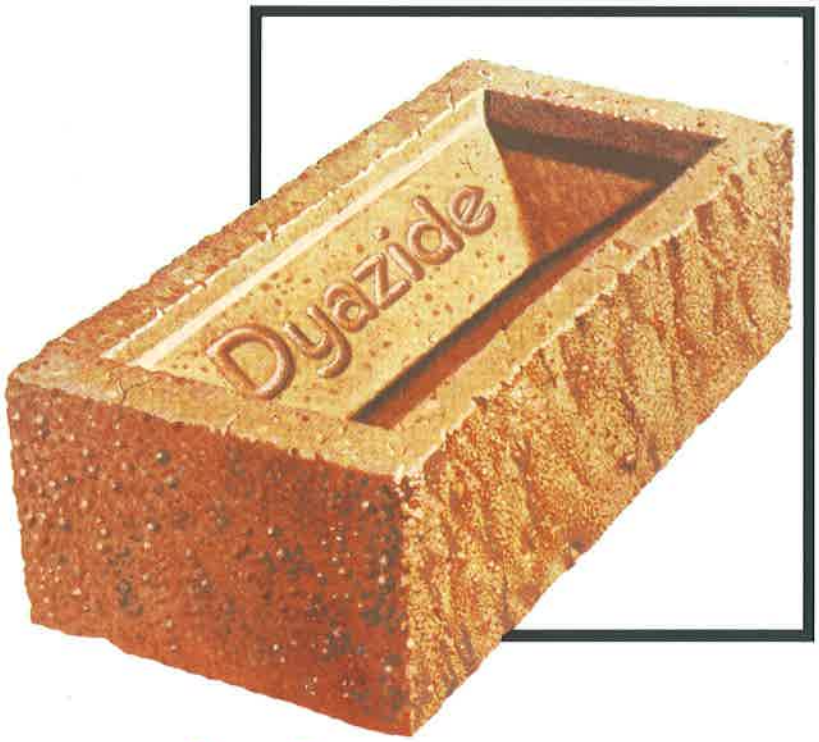

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* WHO - International Code of Marketing of Breast Milk Substitutes, WHA 34.22, May 1981.

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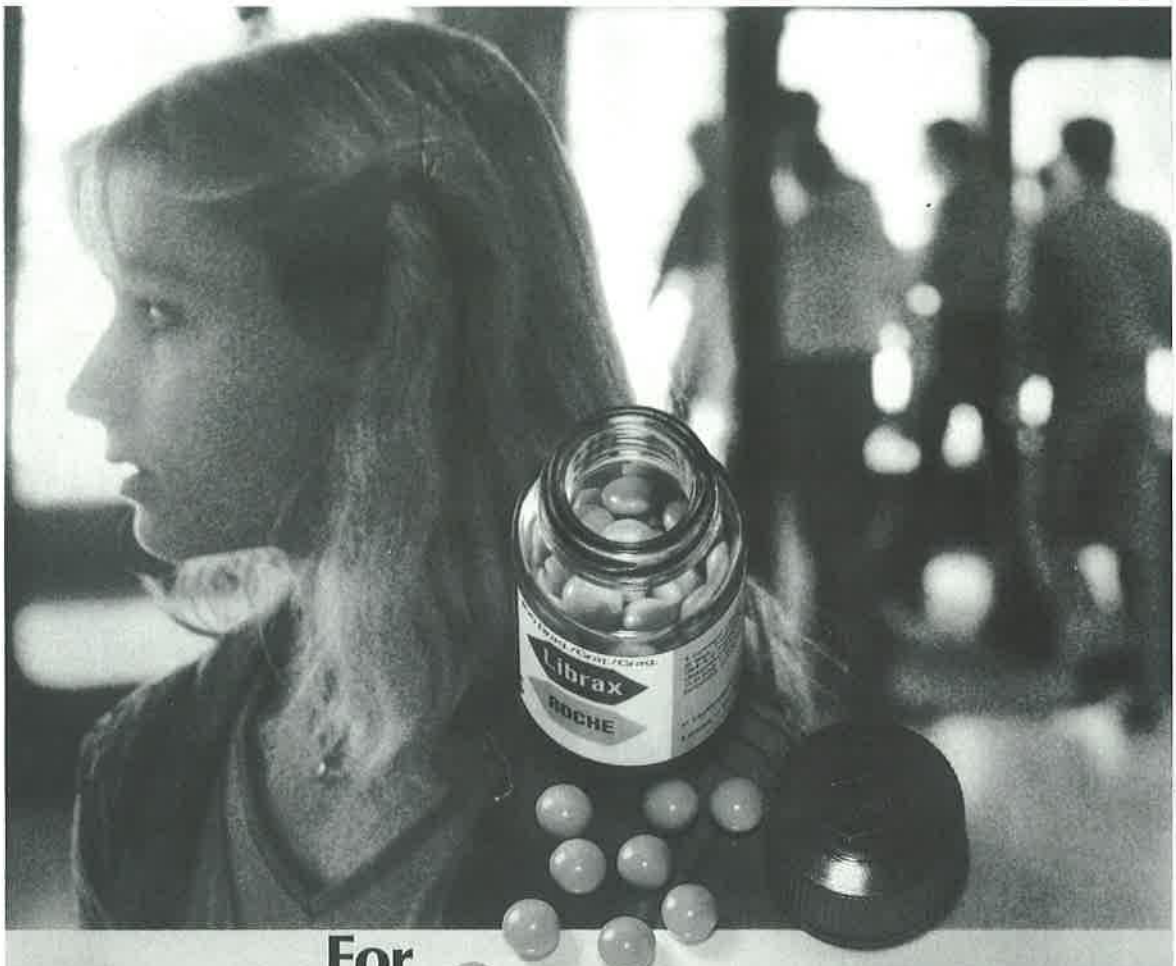

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EDITORIAL

"On Reporting and Notification"

In 1976 attention was focussed on Gonorrhoea when Penicillinase Producing Neisseria gonorrhoeae were described in the United States of America and the United Kingdom. Even before this excitement could abate, STD has again caught the public eye. In 1981 a few isolated cases of a serious mortal malady were discovered in homosexual males. Close epidemiological surveillance and case detection unfolded the new Syndrome of AIDS (Acquired Immune Deficiency Syndrome). The unfolding of this story is a triumph to epidemiological detection.

How was this possible? The first two cases were reported by Dermatologists in **private** practice in widely separated States of America. Warning signals were heeded to, more cases were notified and the whole picture emerged. Sceptics, who opinionate reporting plays a little part in understanding diseases and their treatment and control, should seriously pause to rethink.

In 1977 some of the sexually transmitted diseases were made notifiable in Singapore under the Infectious Diseases Act 1976 Ordinance. To heed to the often repeated plea of practitioners to protect the confidentiality of their patients, the decision was made to make notification non nominal, that is, providing numbers of cases seen with no personal particulars of the patient. It was felt this would provide a more accurate picture on trends of the disease, though tracing of the contacts would not be possible. Available notification data from 1976 to 1982 suggests that **almost equal numbers** of STDs were seen in the public and private sectors. Though no data is available, all will agree this cannot be true. Trends, from available disease data, showed a rise in gonorrhoea and the PPNG strains between 1976 to 1978. A decision was made to recommend amino-glycosides in favour of penicillin as the first choice antibiotic in treating gonorrhoea. Gonorrhoea and PPNG stabilized. In 1980-1981, early infectious syphilis, the incidence of which had dropped, began to rise again. Non PPNG strains began to show increasing treatment failure to Kanamycin therapy. Combination therapy was recommended to reverse this. Here was a case where treatment recommendations were changed periodically utilising available disease data. In 1976 practitioners were encouraged to provide information on contacts, **if their patients had no objections. It is a matter of regret not a single form has been filed in to date.**

Epidemiological data does not collect dust in offices. It is used to formulate control and treatment policies. Complete data will help to arrive at more accurate decisions. If administrative procedures hinder notification of disease, this can be ironed out by dialogue. Greater understanding and co-operation from the profession will be appreciated.

VSR

Views expressed in the Editorial are not necessarily the official views of the College.

The child with abdominal pain

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INTRODUCTION

Abdominal pain is a common complaint in Paediatrics. Usually the pain is transient and has no serious consequence. When the pain is more severe or prolonged, the child will be brought to see a doctor. There may be other associated symptoms such as fever, vomiting and diarrhoea.

The problems facing the doctor looking after such a child are multiple. The multiplicity of organic causes makes the clinical diagnostic approach difficult. It is not always justifiable to make a clear distinction between organic and emotional (psychogenic) illnesses. The other problems peculiar to Paediatrics are related to the age of the child. Young children are seldom able to describe and localise their pain. When asked to locate the site of pain, they invariably point to the umbilicus. Furthermore, abdominal examination is often difficult in children, especially when they are in pain.

MECHANISMS OF ABDOMINAL PAIN

The abdominal viscera are pain-sensitive in their capsules (liver, spleen, kidneys) and peritoneal lining. This includes the gut and the abdominal wall. The mucosa can be cut without painful sensation. The only exception to this is the pain of peptic ulcer where gastric acidity probably plays a role.

The nerves to the capsular and peritoneal structures are both somatic and autonomic. Thus, it is no surprise that psychosomatic influences can be operative in the genesis of abdominal pain.

The commonest organ responsible for abdominal pain is the gut and in most instances, it is the stretch of the gut or direct involvement of the peritoneal lining which causes pain. The movement of the gut, peristalsis, is a complicated process which involves on-going waves as well as localised segmental waves. Any upset in this fine-tuned mechanism can result in stretch and abdominal colic.

CLASSIFICATION

Generally speaking, abdominal pain can be divided into two big groups:—

(I) Acute, isolated episode
(II) Recurrent or chronic abdominal pain

(I) Acute Abdominal Pain

Included under this heading are all the painful abdominal syndromes, sometimes benign, frequently potentially lethal. The common denominator is sudden or rapidly appearing pain with accompanying local or generalised signs and symptoms. This group is also called "acute abdomen" or "surgical abdomen".

Causes of Acute Abdominal Pain

Table 1 shows some of the causes for acute abdominal pain. This list is not meant to be exhaustive. When a child complains of abdominal pain, many other organs outside the abdominal cavity can be responsible. Retroperitoneal organs such as the kidneys and the ureters are known to be responsible for acute colicky pain. Basal pneumonia can present as pain in the abdomen. Other causes include radiculitis and metabolic illness such as drug intoxication, angioneurotic oedema, diabetic ketoacidosis and uraemia. In other words non-gastrointestinal causes should always be kept in mind.

Important associated signs and symptoms of acute abdominal pain

(1) Gastrointestinal

Vomiting is by far the commonest associated symptom. However, it may not be present in some cases of acute abdomen. Severe vomiting may cause laceration of the lower end of the oesophagus, the so-called Mallory-Weiss Syndrome. In children, vomiting always carries the risk of aspiration pneumonia. Chemical and bacterial

peritonitis is generally associated with vomiting. In intestinal obstruction, the vomitus is usually bilious.

Acute abdominal pain may be associated with other bowel symptoms, such as bloody stools or watery diarrhoea. Bloody stools usually indicates bleeding in the gastrointestinal mucosa. It may be infective such as dysentry or due to ischaemia such as intussusception. Gastroenteritis can also present as abdominal colic and diarrhoea.

Generally speaking, the finding of an abdominal mass associated with cramping pain is an indication of intestinal obstruction. In children, it is usually due to intussusception or volvulus.

Other abdominal signs include distention, loss of bowel sounds and localised tenderness. Abdominal distension and absence of bowel sounds are seen in paralytic ileus. Depending on the site, localised tenderness is a useful pointer to the organ that is giving trouble.

(2) Systemic signs and symptoms

In a child with abdominal pain, vital signs such as pulse rate, respiratory rate and blood pressure are useful in monitoring the progress of the child. Suppression of abdominal breathing and the presence of tachypnoea are indicative of peritoneal irritation.

Vasomotor changes during painful episodes such as pallor and sweating are common in severe abdominal colic due to organic causes. Psychogenic or emotional abdominal pain is seldom accompanied by positive signs and vasomotor changes.

Clinical approach to a child with acute abdominal pain (Table 2)

Step I

This is the most important step. A full history and careful physical examination are mandatory. One should pay special attention to the mode of onset, character, site, radiation, aggravating and relieving factors. Acute and violent onset usually indicates perforation or torsion. Pain taking a few hours to attain its greatest intensity is frequently due to inflammatory process. Constant and continuous pain is also indicative of an inflammatory process or stretched capsule. Pain that is colicky in nature usually originates from hollow tubes such as the intestine, ureter and biliary tree. The initial localisation of pain is helpful. Classically, acute appendicitis presents with paraumbilical pain before it is localised at the right iliac fossa. The radiation of pain may help in the diagnosis. A good example is ureteric colic which radiates down the loin to the iliac fossa.

Physical examination should include all other systems besides the abdomen. There are many useful clues which have been mentioned earlier.

Step II

If possible, a few simple laboratory investigations are necessary. These include a blood count, urinalysis and stool examinations. These tests are simple and can be carried out in the office.

Step III

This step involves the continued observation of the child. If the child is still having pain and the cause still not ascertained, more invasive investigations are necessary. By and large, such children should be managed in a hospital where more facilities are available.

(II) Chronic, recurrent abdominal pain

The term chronic abdominal pain is used here to include all abdominal pain that has lasted for a prolonged period. It may last for weeks, months or longer. It may be continuous or interrupted by pain free periods.

Table 3 shows the prevalence of recurrent abdominal pain in children. It is about 10%, with females commonly affected than males. Miller (1974) reported an overall prevalence of as high as 18%.

Table 4 shows some of the organic causes of recurrent abdominal pain. It is not possible to describe all the causes here. A few common causes seen in Singapore will be discussed.

(1) **Lactose intolerance**

This is a common cause of recurrent abdominal pain in Singapore children. Depending on the ethnic groups, 50%-90% of the children older than 5 years of age have evidence of hypolactasia. The clinical presentations include recurrent abdominal pain, flatulence, vomiting and diarrhoea. Diagnosis can be confirmed by lactose tolerence test, hydrogen breath test and diminished lactase activity. Omission of lactose from the diet would result in clinical improvement.

(2) **Cow's milk allergy**

In some reported series, the incidence of cow's milk protein allergy is as high as 10% in children.

The gastrointestinal manifestations of cow's milk allergy include recurrent abdominal pain, vomiting, diarrhoea, malabsorption and bleeding. In older children, it is usually associated with respiratory and dermatological manifestations such as rhinitis and atopic dermatitis. In younger children, soya bean preparation and meat base formulas can be used as milk substitutes. In older children, all cow's milk and milk product should be avoided.

(3) **Choledochal cyst**

Choledochal cyst is the cystic dilatation of the common bile duct. It is due to congenital weakness of the duct.

In Singapore, it is more commonly seen in Chinese girls. The classical presentation is a triad of recurrent abdominal pain, jaundice and a mass in the right hypochondrium. Diagnosis can be confirmed by ultrasonography or barium meal. Treatment is surgical.

(4) **Constipation**

Faecal masses can upset the normal peristalsis. Attending to this problem often removes the pain. Frequently, dietary manipulations, oral purgative and rectal suppositories are helpful in the initial management. When the distended bowel has regained its tone, bowel movements will be regular.

(5) **Worm infestations**

Parasitic infestations can be the cause of the recurrent abdominal pain. However, worm infestation is less common nowadays in Singapore, except in the rural areas. In the management of a child with worm infestation, care should be taken to prevent reinfection and in the improvement of personal hygiene.

(6) **Recurrent abdominal pain of emotional origin (psychological)**

This was well described by John Apley. Such children tend to show characteristic personality traits. They are described as highly strung, fussy, excitable, anxious, timid and apprehensive. Other expressions of emotional disturbance are also common such as nocturnal enuresis. There is also a high incidence of abdominal pain in their relatives. Such children and their families need emotional support and care.

CONCLUSION

Abdominal pain is a common problem. Acute abdomen needs urgent diagnosis and treatment. Many of the causes are surgical and prompt treatment will result in improvement.

Recurrent abdominal pain is usually a diagnostic problem. Often, an organic cause cannot be found. It is this group of children that need regular medical care. Every possible means must be made used of if the pain is severe enough to disturb their sleep and impair their physical growth.

TABLE 1

CAUSES OF ACUTE ABDOMINAL PAIN

(A) Abdomen
1. Accidental trauma
2. Peritoneal irritation
   Chemical (blood, urine, bile, pancreatic juice) bacterial
3. Perforation of hollow organs
4. Rupture, laceration of a filled organ
5. Torsion (gastric, gall bladder, bowel, ovarian cyst)
6. Occlusion of hollow organs
   Small and large intestines, bile duct, pancreatic duct, urinary tract
7. Vessels
   Rupture, obstruction

(B) Retroperitoneum
   Urinary tract, spontaneous haematoma or traumatic abscess

(C) Thorax
   Trauma, Basal penumonia and pleuritis, pneumonia

(D) Vertebral column
   Radiculitis (Herpes Zoster)
   Compression of nerve roots

(E) General causes (metabolic) (medical acute abdomen)
1. Exogenous
   Intoxication – drugs; lead intoxication
2. Endogenous
   Uraemia; incipient diabetic coma; certain familial hyperlipidaemia, Hereditary angioneurotic oedema, Haemolytic crisis; Adrenal insufficiency.

TABLE 2

APPROACH TO A CHILD WITH ACUTE ABDOMINAL PAIN

STEP 1
Detailed history and physical examination:
- mainly to exclude
- If surgery indicated, proceed to confirm the diagnosis and request for surgical opinion

STEP II
Some simple laboratory investigations:
- F.B.C., urinalysis, amylase, electrolytes – blood sugar, C.X.R., A.X.R. stool examinations

STEP III
Monitor clinical progress:
- Repeat step II when indicated
  Ultrasonography, endoscopy and C.T. scanning when necessary
  Always have a second opinion if in doubt

Abbreviations:

F.B.C. = Full blood count
C.X.R. = Chest X-ray
A.X.R. = Abdominal X-ray
C.T. = Computerized tomography

TABLE 3

PREVALENCE OF RECURRENT ABDOMINAL PAIN

Reference	Overall Prevalence	Male	Female
Apley and Naish (1958)	10.8%	10%	12%
Pringle et al. (1966)	–	14%	15.7%
Miller et al. (1974)	18%	–	–
Oster (1972)	14.4%	12.1%	16.7%

TABLE 4

ORGANIC CAUSES OF RECURRENT ABDOMINAL PAIN

I) INTRA-ABDOMINAL
- a) Gastro-intestinal e.g.
  Henoch-Schonlein Disease
  Peptic Ulceration
  Meckel's diverticulum
  Food allergy
  Lactose intolerance
  Aerophagy
  Duplication cyst
- b) Liver and gall bladder – e.g.
  Choledochal cysts
  Hepatitis
- c) Pancreas – e.g.
  Pancreatitis
  Cystic fibrosis
- d) Spleen – e.g. Splenic infarction
- e) Renal – e.g.
  Urinary tract infection
  Hydronephrosis
  Renal calculi
- f) Lymoh glands e.g. Mesenteric adenitis
- g) Peritoneum e.g. Peritonitis

II) REFERRED PAIN

Pleura, Spine, Testes, Ovaries, Spinal Nerves

III) METABOLIC

Diabetes Mellitus
Hypoglycaemia
Ketosis

IV) SYSTEMIC INFECTION

V) NEUROLOGICAL

Epilepsy

Uses of peritoneoscopy in gastroenterology

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INTRODUCTION

Diseases of the abdominal cavity have conventionally been diagnosed by good history taking, thorough clinical examination and standard laboratory & radiological studies including contrast X-Ray's. However, a proportion of cases go undiagnosed unless an exploratory laparotomy is made. Peritoneoscopy is a form of 'mini-laparotomy' which does not require a general anaesthetic for its performance. Through a fiber-optic lens system, the examiner is able to visualise much of the peritoneal cavity. In particular, the pelvic organs and the area of the liver is eminently accessible. Exploratory laparotomy carries a mortality of 30% in the presence of hepatic pathology (Wang & Li, 1963; Chan, 1967). In comparison peritoneoscopy carries a negligible mortality & morbidity rate (Bruhl, 1966; Berci, 1976).

The purpose of this communication is to demonstrate the many diagnostic uses of peritoneoscopy and includes an account of the endoscopic procedure and its potential complications.

APPLICATIONS:

I. **INVESTIGATION OF LIVER DISEASE:** Peritoneoscopy offers a means of observing the diagnosis, prognostication, results of treatment and the progress of chronic hepatitic-liver cirrhosis and hepatocellular carcinoma. Biopsy of hepatic lesions under direct vision is definitely safer than blind liver biopsy. Examples of diseases that can be diagnosed immediately under direct vision are: Dubin-Johnson Syndrome, haemochromatosis, cystic liver etc.

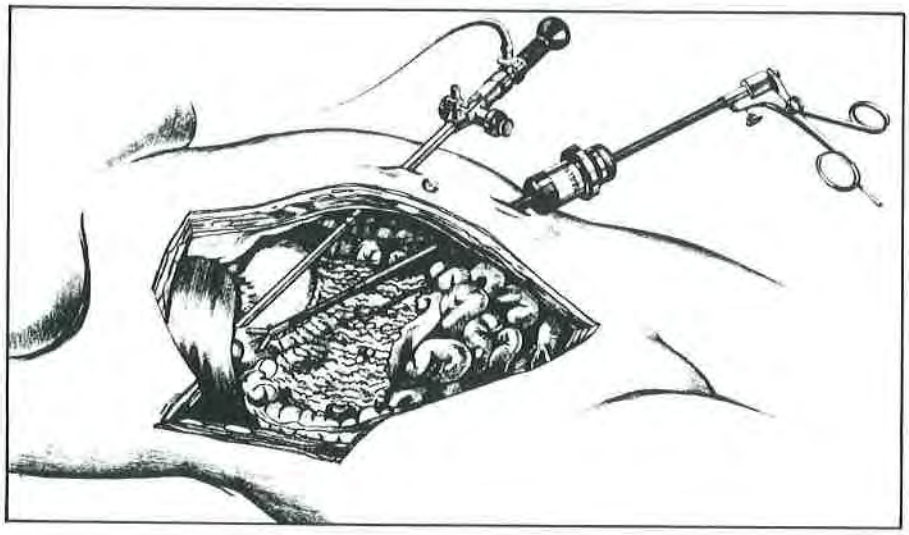
II. INVESTIGATION OF GALLBLADDER & BILIARY TREE: The importance of peritoneoscopy lies in the possibility of determining primary gallbladder cancer at a glance & also in deciding whether further surgery is warranted in obstructive jaundice. Direct vision needling of the gallbladder & injection of contrast medium enables radiographic demonstration of the biliary passages.

II. **INVESTIGATION OF GALLBLADDER & BILIARY TREE:** The importance of pericysts, inflammation, cancer growth, vessel engorgement etc., a macroscopic diagnosis is often possible. Supra- or infra-gastric pancreascopy enables more accurate visual diagnosis and biopsy sampling.

IV. **INVESTIGATION OF GASTROINTESTINAL DISEASE:** Gastric cancer can be easily visualised in cases where cancer infiltration has spread to the serosa of the anterior wall, especially of the antrum. Peritoneoscopy thus helps determine the need for radical surgery based on observation of the changes in the surrounding tissues and organs. Large and small intestinal tumours can similarly be assessed. All enteric processes which cannot be seen clearly from the luminal aspects of the intestine and in which mucosa is often involved eg. subserosal leiomyoma, neurofibroma, carcinoid, as well as inflammatory changes such as intestinal tuberculosis & Crohn's Disease are all admirably suitable for peritoneoscopic diagnosis.

V. **INVESTIGATION OF PERITONEAL DISEASE:** At times peritoneoscopy may be the only means of determining changes in the peritoneum caused by tuberculosis and peritonitis due to cancer and pseudomyxoma. Peritoneal adhesions present difficulties in the introduction of the instrument but with experience less vascular adhesions may be divided with the operating peritoneoscope.

VI. **INVESTIGATION OF SPLENIC DISEASE:** Differential diagnosis of splenic enlargement can be made from those of kidney and enlarged left liver lobe and difficult left hypochondrial tumours. Splenic biopsy under direct vision is also feasible. Splenoportography un-



Biopsy Under Peritoneoscopic Control

der direct peritoneoscopic control is gaining popularity. Other diagnoses that can be made include leukemia and Banti's disease.

THE TECHNIQUE:

Conventional peritoneoscopy requires hospitalisation and use of the operating room. After the administration of a local anaesthetic to the subumbilical area, a needle is inserted into the peritoneal cavity so that a pneumoperitoneum can be created using carbon dioxide as gas. The pneumoneedle is then withdrawn and an examining trocar and cannula is inserted. Withdrawal of the trocar allows insertion of an examining telescope to view the contents of the peritoneal cavity. Telescopes with 3 directions of view are generally available, viz. 0 degree (Straightforward), 30 degrees (Forwardoblique) & 90 degrees (Lateral).

To enable biopsies to be taken, an operating peritoneoscope can be utilised in place of the simple instrument designed for inspection only. If organs are to be moved, use of a second, smaller accessory trocar can be inserted at another site (usually one of the abdominal quadrants). A palpation probe can then be inserted after withdrawal of the stylet. This probe is used as an 'extended finger'. The sensation gained by palpating a cystic or solid organ is transmitted to the fingertips and gives information about the consistency of the tissues in question.

When the examination is completed, the instrument is withdrawn and the gas escapes simultaneously. The skin need only be sutured if the puncture site is unusually large or there is associated ascites, in which case the peritoneum should be closed by a single stitch.

CONTRAINDICATIONS:

The absolute contraindication for peritoneoscopy is intestinal obstruction with gross abdominal distention due to dilated bowel (Chan, 1962). In such cases the risk of puncture of bowel is too great to justify the procedure. Peritoneoscopy is also ill-advised in the presence of peritonitis & uncorrected haemorrhagic diathesis. We do not advise the procedure in the presence of an abdominal aneurysm or diaphragmatic hernia.

Multiple previous operations constitute a relative contraindication. A single previous operation may not contraindicate peritoneoscopy as the operation may have been in a region away from that to be viewed. Severe coexistent medical conditions eg. cardiac disease and chronic obstructive lung disease fall within this category.

COMPLICATIONS:

The incidence of complications is low. Ruddok (1957) had 10 cases of perforated viscus out of 1500 examinations; no mortality was recorded because the complication was usually recognised during the endoscopy or soon afterwards and appropriate treatment instituted. Bruhl (1966) reported 19 deaths out of 63,845 cases while Wildhirt (1969), Horwitz (1972) gave mortality rates of 0.02% and 0.18% respectively.

The morbidity (patients requiring exploration

because of perforation or bleeding post-peritoneoscopy) varies from 0.1 to 0.8% (Berci, 1976). The commonest minor problem is that of subcutaneous emphysema which usually does not require active treatment. Great omentum emphysema can occur but is rare. Rarer still are transient arrhythmias, biliary peritonitis, and other infective complications. The competence of the examiner is one of the most important factors in keeping the complication rate at an acceptable level.

ACCURACY:

Overall accuracy rate of peritoneoscopic diagnosis was reported by A. Yosioka et al as 82% (1968). The diagnostic rates for the various intra-abdominal organs in this Japanese series appear in Table I.

TABLE 1:

ACCURACY OF PERITONEOSCOPY:

Liver Diseases:	83%	(503/599)
Bile Duct Diseases:	77%	(136/175)
GIT Tumours:	74%	(75/101)
Pancreatic Disease:	72%	(26/36)
Peritoneal Disease:	95%	(61/64)

Overall Accuracy (A. Yosioka et al: 1968): 82%

In the West, Rivera & Boyce (1972) reported an overall accuracy rate of 94.6% while Berci & coworkers (1973) recorded a 92% accuracy.

CONCLUSION:

In the field of Gastroenterological Endoscopy, peritoneoscopy is becoming increasingly attractive as a safe and useful diagnostic tool due to the definite & reasonably accurate diagnosis which this method offers to the gastroenterologist and hepatologist.

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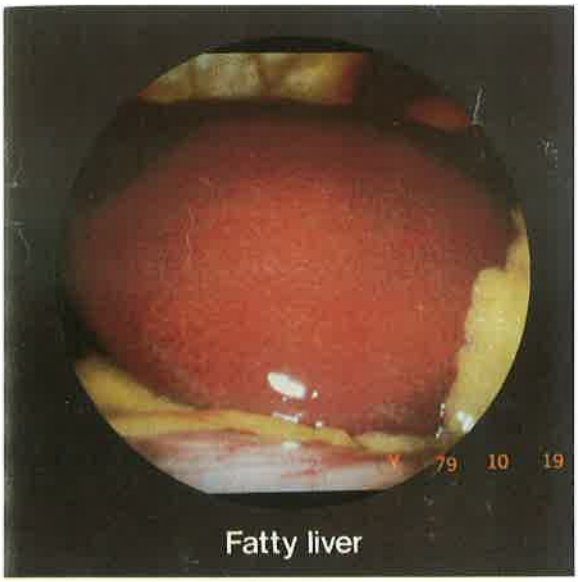
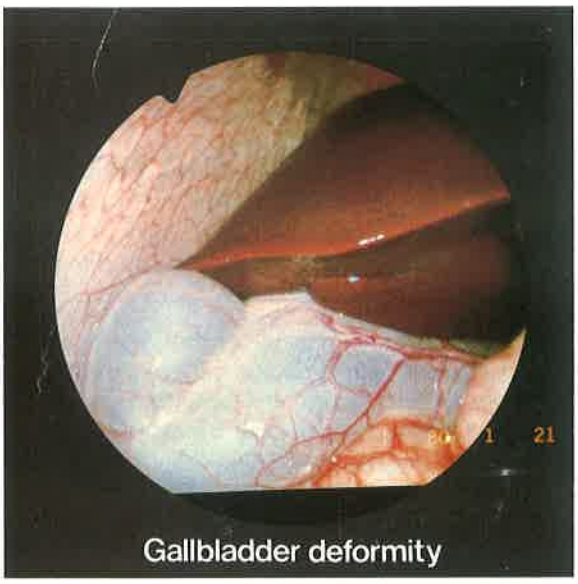
Typical Endoscopic Pictures* Taken With The Olympus Peritoneoscope And Camera



Fatty liver



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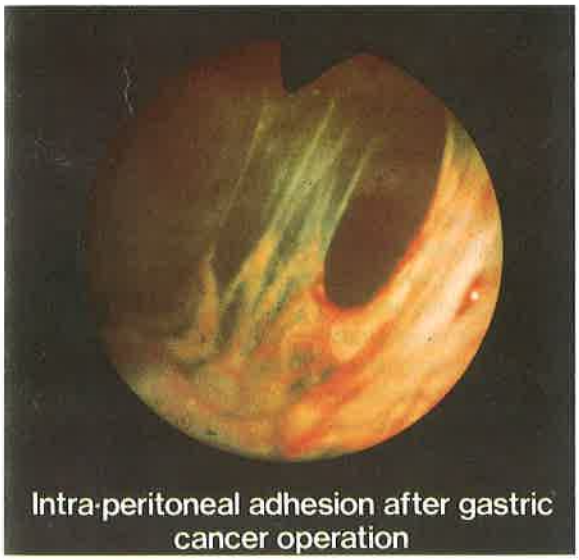
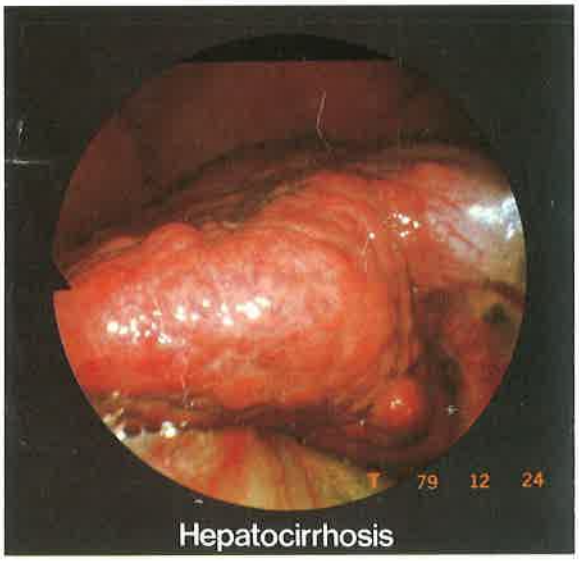
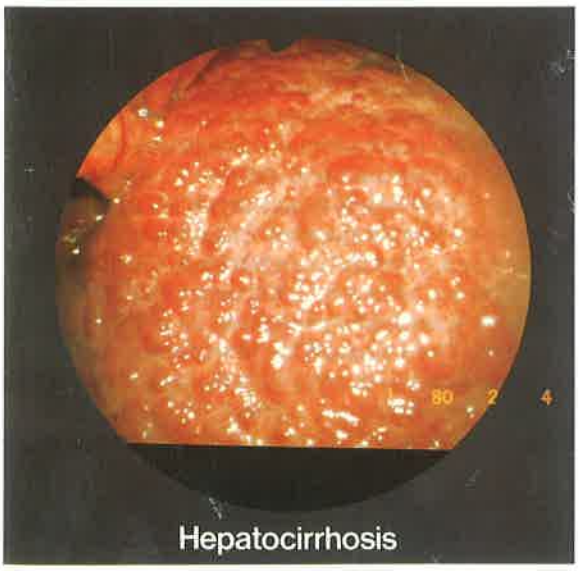


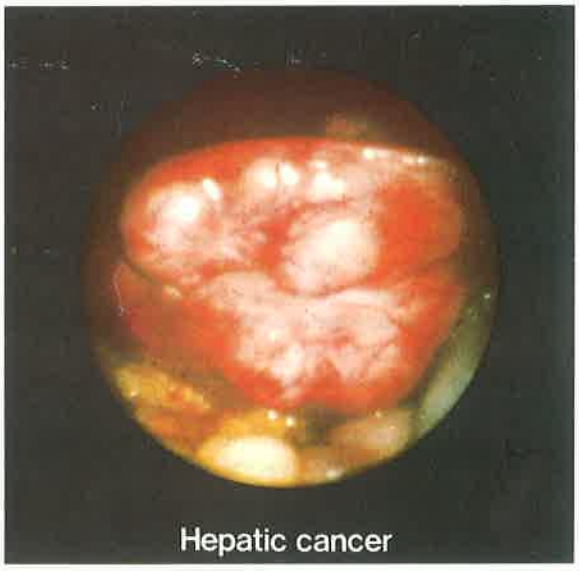
Intra-peritoneal adhesion after gastric cancer operation



Hepatocirrhosis



Hepatocirrhosis



Hepatic cancer

** Endoscopic photographs coutesy of Olympus Opitcal (Japan).*

Chronic Cervicitis — A review of an old problem

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Although this term is one of the commonest in gynaecological textbooks and clinical practice, it is a very difficult one to define. Chronic cervicitis was a term used to describe the sequelae to acute cervicitis when that condition was much commoner and was itself a consequence of trauma, childbirth and septic abortion. The symptom of chronic cervicitis was a purulent mucoid discharge and it is primarily to this symptom that the diagnosis of chronic cervicitis has become adapted in recent years.

To quote C.J. Dewhurst "Cervicitis is less easy to define. If there is considerable mucus production in either ectropion or erosion, it is probable that cervicitis is present to some extent. This does not imply predominantly an effective condition with virulent organisms present. It implies rather that there is a general increase in the glandular mucus-producing elements in the cervix, and that superficial minor infection is probably present in those areas. Certainly, the mucus seen on such a cervix is often yellowish and unpleasant, and seems likely to be due, in part at least, to a minor infective element".

Cervicitis implies first and foremost infection. Patients who have an erosion or ectropion, display a reddened cervix because there is only a thin layer of superficial glandular epithelium covering the very vascular cervical stroma. This together with high vaginal swabs showing organisms or cervical biopsies showing a leucocytic infiltration, are labelled chronic cervicitis. The use of the term 'cervicitis', implying infection of the cervix, would appear to be reasonable where organisms are present and there is a white cell reaction, but these two points do not bear closer bacteriological and histological examination.

The purulent appearance of cervical mucus is deceptive and in chronic cervicitis, bacterial cultures rarely ever confirm the clinical diagnosis of the "infection". More often than not, normal commensals or no organisms at all are reported. [Pinkerton, Calmand and Claireaux, 1962]. With a few exceptions such as gonorrheal infections, trichomoniasis and acute pelvic sepsis, the high vaginal swab must be one of the most used and least productive of laboratory investigations.

The other parameter for labelling infection is leucocyte infiltration and this observation leads to chronic cervicitis being diagnosed almost invariably on hysterectomy specimens. However, there is a possible explanation for the inflammatory reaction other than infection. Cervical metaplasia is naturally and physiologically accompanied by death of cells [Glucksman, 1951; Forsberg, 1967). The leucocyte infiltration is more likely to be a "mopping-up operation" of physiologically dead cells.

An alternative explanation for this leucocytic infiltration has been put forward by Coppleson. He has demonstrated that foreign nucleic acids can be transmitted through coitus and can enter the metaplastic cervical cells. There is then an immunological response. This response, together with a thick cervical mucus and shed necrotic cells, forms a mucopurulent-looking discharge. Chronic cervicitis is an understandable, if inaccurate, term for what is an immune response at a cellular level. This response is commonest in the first few years after coitus has begun and in those with several sexual partners [Singer, 1975].

Thus, bacteriological culture is rarely relevant or important in chronic cervicitis and the inflammatory reaction may merely be a way of disposing of foreign antigens such as dead cells and spermatozoa.

Diagnosis

If bacteriological cultures and leucocytic infiltration are unimportant in the diagnosis of chronic cervicitis, how are we to define it?

It is certainly a condition of the reproductive age, being rare before the menarche and after the menopause, and is a common cause of leucorrhoea. The thick, tenacious discharge may be white, yellow or occasionally green in colour and abnormal bleeding such as postcoital or intermenstrual bleeding is rare unless there is an associated additional pathology.

The cervix commonly will be lacerated and partially everted, and the gland-bearing endocervical epithelium becomes thickened and reddened. As

the squamocolumnar junction moves, so some of the glandular epithelium which has been infected is recovered, first by metaplastic and subsequently by squamous epithelium. This occludes glands causing mucus retention cysts which appear as small white or grey vesicles [nabothian follicles]. If these are deep in the cervical tissue, they may not be apparent on inspection and are palpable as irregularities. On cautery or puncturing these vesicles they discharge a clear mucus.

Influence on Infertility

A clean, healthy, ovulatory cervical mucus is an essential part of sperm capacitation and of fertilization. The thick, unhealthy mucus associated with chronic cervicitis must inhibit sperm motility and function to some extent. Sperm-mucus hostility and incompatibility are common findings in infertile couples who are otherwise apparently normal. The part which immunological factors play in the leucocytic infiltration found so commonly in the cervix has been discussed already. The role which these immunological factors may play in the mucus and in infertility is still much debated and far from clear.

Treatment

(1) No Treatment: One of the most helpful things a doctor can do for his patient is to explain and to get the patient to accept the condition when no treatment is necessary. If a patient with leucorrhoea, mucus retention cysts and an everted cervical canal or associated erosion has no abnormal bleeding and can accept her discharge, then she should be encouraged to do so and not to pursue treatment for treatment's sake. If the symptoms are unacceptable, the following treatments are available.

(2) Cauterization: Hot
Cold

Radial cauterization using the hot cautery was first reported by Hunner in 1906 and has been a standby of treatment ever since. The technique is to make radial incisions from external os outwards to normal epithelium spacing the incisions 1 to 2 mm apart. The effect is that unhealthy epithelium is destroyed and replaced by healthy squamous epithelium.

This method of treatment of chronic cervicitis and erosion is now being increasingly replaced by cryocautery [Ostergard and Townsend, 1969]. Various probes are available and the usual refrigerants in gynaecological practice are nitrous oxide or carbon dioxide which are safe and easy to store. Before using cryosurgery in chronic cervicitis, it is important to have a normal smear or colposcopic examination. The cervix is best subjected to cryocautery just after a period has finished. This allows the most active phase of regeneration to occur before the next menstrual flow. It also avoids the risk of cauterizing the pregnant cervix [Townsend, 1976]. In the view of the author, this method is now the treatment of choice for chronic cervicitis.

(3) Surgical treatments: Various surgical approaches have been described which include electrical conisation [Hyams, 1928] and Sturmdorf's tracheloplasty [Sturmdorf, 1916]. These two operations are now largely of historic interest and have been replaced by cryocautery. There is a reasonable place for hysterectomy if chronic cervicitis is accompanied by other uterine pathology and the cervix may be amputated as part of the treatment of prolapse.

(4) Antibiotics: There is no place for systemic antibiotics in chronic cervicitis. Topical antibiotic and sulphonamide preparations are also ineffective.

Conclusion

Chronic cervicitis is a common diagnostic label but hard to define because the pathological appearances bear little relation to the clinical reality of leucorrhoea.

More often than not, no treatment is required unless the symptoms are unacceptable to the patient.

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Endotracheal intubation — A practical approach

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COMMON PROBLEMS

A 42-year old Malay lady was brought to the Accidents and Emergency Department of Changi Hospital, "going unconscious and blue". It was found that a dislodged denture was the cause (removed at laryngoscopy).

A 17-year old Chinese school girl was brought for "drowning" at Changi beach in a state of asphyxia.

Both the above cases were intubated expeditiously and subsequently sent to the ICU for further management. Suffice it to say, that both the patients left the hospital alive and well.

The above two incidents may serve as possible examples of patients who may unexpectedly present at a busy general practitioner's clinic and the intitial procedure of endotracheal intubation may therefore be a life saving manouvre. It is therefore very useful if not mandatory that all doctors should familiarize themselves to achieve a certain amount of proficiency in the art of endotracheal intubation. A brief but practical approach for the procedure is presented below using minimal equipment. The aim is life saving.

INDICATION

The usual indications for endotracheal intubation which the General Practitioner or the Family Physician may face are:—

1) Upper Respiratory Tract obstruction of any form and
2) Resuscitation.

EQUIPMENT

1) Laryngoscope: The Macintosh curved adult blade is perhaps the most versatile which can also be used in children up to say age 6.
2) Endotracheal tubes: The most commonly used are sizes 9 for males and 8 for female adults. In any case sizes 6 to 9 will serve for most purposes in our local population. A malleable introducer may be occasionally needed for the firmer Oxford or Portex tubes and in difficult situations.
3) Connectors — Noseworthy connectors; a set of 2 pieces, one for the endotracheal tube and one for the tube leading to the Air Viva are required.
4) A 10 ml syringe for inflating the cuff.
5) Magill's forceps is not an absolute necessity but may become handy to guide the tube towards the trachea.
6) Suction apparatus such as the foot operated type.

THE PROCEDURE

Having checked the equipment the doctor may proceed as follows:—

1) Stand at the head end of the patient.
2) Support the head on a pillow so that the neck is flexed.
3) Extend the head by tilting backwards as far as possible. To assist this sometimes one has to place one hand behind the neck and tilt the head backwards by the other hand placed on the forehead. (This manouvre will flex the neck, extend the head and raise the tongue away from the back of the throat. The reason for all these is to bring the acute axes of the oral cavity, the pharynx and the trachea towards an obtuse axis to facilitate intubation on an easier "straight line" directional advantage).
4) Hold the laryngoscope in the left hand (if you are right handed) and introduce your tube with the right hand. Here, if the upper third of the handle is held between the index finger and thumb, the procedure becomes easy as this serving as a fulcrum will provide definite mechanical advantage (law of force). Also the other 3 fingers could help to hold the lower jaw.
5) Gently introduce the blade into the mouth slightly to the right and push in sweeping the tongue towards the left.

6) The epiglottis will be then seen and the blade passed upwards between it and the base of the tongue. As the epiglottis is thus lifted the larynx should come into view.
7) The tube can then be passed preferably at abduction of the cords.
The actual procedure should be smooth and easy.
Not uncommonly, the tube enters the oesophagus. To ascertain its proper position in the trachea, ensure air flow through the endotracheal tube or observe (including auscultation) rise of the chest during ventilation.
8) Once intubation is performed, the tube must be secured firmly either by a plaster or a bandage.
9) Inflate the cuff till no air leak is heard.

SOME PROBLEMS IN ENDOTRACHEAL INTUBATION

1) One should not use the teeth as a fulcrum for the laryngoscope.
2) Any loose teeth dislodged inadvertently should be removed.
3) Ensure that the tube is not too long as then it may go into the right bronchus.
4) Cass et al have listed the common anatomical variations that may make the procedure difficult viz:—

a) Short muscular neck with full set of teeth.
b) Receding lower jaw with obtuse mandibular angles.
c) Protruding upper incisor teeth associated with relative overgrowth of pre-maxilla.
d) Poor mobility of the mandible.
e) A long high-arched palate associated with a long narrow mouth.
f) Increased alveolar-mental distance which necessitates wide opening of the mandible for laryngoscopy.

However the above presentations are fortunately infrequently encountered. When encountered, intubation could be accomplished with the help of an introducer and with the acquisition of some proficiency in the art.

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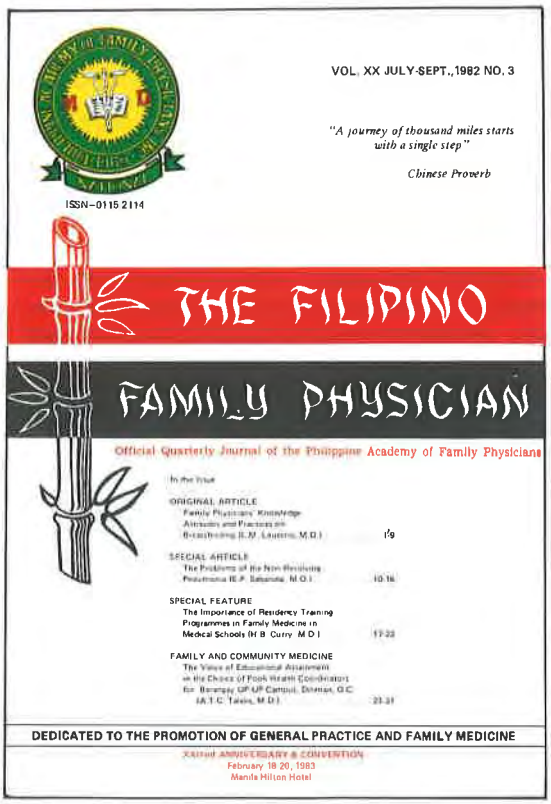
The Filipino Family Physician Journal



"The Filipino Family Physician" is the official Journal of the Philippine Academy of Family Physicians (PAFP), published Quarterly. The Inaugural Number came out in 1962-63, with Dr. Ramon R. Angeles, the Academy's Founding President, as Editor-in-Chief, a position he still holds to the present. There is a panel of a Editorial Staff, Contributing Editors and an Advisory Board, as the policy-making bodies.

The regular Contents are an Editorial Page, Original and Special Articles, a Special Feature. "The President Speaks ..", Office of the National Executive-Secretary, PAFP News, a Pictorial Review of recent activities and news of World-Wide interest, for Family Physicians, in particular and for the members of the Medical Profession, in general.

The format of the **Front Cover** is featured with the official emblem of the PAFP, colored green, yellow and red; with an open book with Malayan or Sanskrit writing, between the letters "M" and "D" depiciting knowledge. This official emblem was designed by Dr. Gregorio G. Lim, a Charter Member of the Academy and art painter.

The piece of a cut bamboo from the bamboo tree, which grows abundantly in the Philippines, is "pliant and strong, that bends before the wind, but stands proudly after the storm".

The cover-design of the Journal is not changed since its first issue to the present, so that it can maintain its true identify and can easily be recognized or picked-up from other medical journals.

Continued publication of the Journal is funded mainly by the Pharmaceutical Industry and with special assessment for Academy members, and regular subscriptions, local and foreign.

We have more than 30 local and foreign regular exchanges with other Journals.

The attention of our readers is directed to the Chinese Proverb on the right upper quadrant of the cover which reads:–

"A journey of thousand miles starts with a single step."

Information and writeup of The Filipino Family Physician Journal are contributed by DR. RAMON R. ANGELES, MD, EDITOR-IN-CHIEF, The Family Physician Journal, c/o R-508 Mercedes Building, Plaza Miranda, Quiapo, Manila, The Philippines.

Ed.

Epidemiology of sexually transmitted disease in Singapore

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Introduction

STD in Singapore has been well contained by the implementation of epidemiological control over the last 6 years. The incidence of the major STD which include Gonorrhoea and Syphilis have declined steadily over the years. Special mention of the changes in the trend of early Syphilis over the last 2 years however requires special elaboration.

Since 1976 following a rise of Gonorrhoea in the Republic, the incidence has declined steadily. Further evidence of the good control of Gonorrhoea is shown in the reduced incidence of Ophthalmia Neonatorum. This is evidence of early and prompt treatment of Gonorrhoea before dissemination is possible.

Similarly, the incidence of early congenital Syphilis has declined markedly over the last few years again denoting early diagnosis and treatment of the infectious stages of the disease and also good ante-natal screening and treatment.

The incidence of early Syphilis however has risen significantly over the last 3 years and this may cause concern. This is probably related indirectly to the increased incidence of PPNG infection when from 1979 Kanamycin a non treponemacidal antibiotic has been frequently used as treatment for Gonorrhoea. By 1980 and 1981, Kanamycin was recommended as a first line drug for the treatment for Gonorrhoea. In the earlier years when high dose penicillins were used as the first line drug it is believed that the drug could have eradicated many cases of incubating Syphilis whilst being administered for treatment of Gonorrhoea. This could have accounted for the sharp rise of Syphilis.

As regards other STD, the incidence of Chancroid appears to be on the decline after peaking in 1976. This decline may be the effect of treatment of asymptomatic prostitutes. Like many countries in the world the incidence of genital herpes has increased slowly and until effective treatment is available there may be a problem in controlling this rising trend.

Penicillinase Producing N Gonorrhoeae (PPNG) situation in Singapore

The incidence of PPNG infection continues to rise over the last few years since it was first reported in 1976. This figure has risen from 0.07% in 1976 to 34.32% last year (1982). There appeared to be a slightly higher incidence of PPNG infection in the public than in the prostitutes. The percentages in the public and the prostitutes are 32.5% and 38.8% respectively.

The rise in the PPNG infection is also reflected in the cases of Ophthalmia Neonatorum. Prior to 1977 no cases of PPNG Ophthalmia was seen but the percentage has risen from 9.1% in 1978 to 33.3% last year. This is indeed a high figure.

We do not have figures of PPNG pelvic inflammatory disease as most cases are seen by the gynaecologists. But the figures would probably be reflecting a similar trend.

Contact Tracing

The number of forms issued to primary contact has increased yearly. The percentage of successful contact has dropped from 82% in 1978 to 71% last year. This is partly contributed by inadequate information received from patients for proper contact to be traced. Last year this figure has fallen to 71% successful contact and the number of prostitutes contact has dropped to 883 compared to over one thousand earlier. This is probably due to foreign prostitutes especially the Thais where 'free-lance' prostitution and communication problems between client and prostitutes hindered with collection of proper contact tracing informations.

The incidence of disease in the primary contact has increased slightly. This may reflect earlier case detection, before patients seeked treatment and the detection of the disease during the asymptomatic incubating phase. On the other hand it could also mean increased virulence of the infecting organisms with high infectivity potentials.

The Medical Scheme for Prostitutes

The Medical Scheme has been in existence since 1976. Over these years the number of prostitutes registered in the Scheme has increased. 1,845 new prostitutes were registered into the Scheme last year making the total registered to 10,051. This included 852 Thai prostitutes registered from October to December last year. Malaysian prostitutes still form the majority of the prostitutes in the Scheme.

The average GC infection rate is 7.2% for 1982 compared to 6.7% for 1981.

The incidence of Gonorrhoea in the prostitutes has increased slightly last year to 6,812 compared to 5,362 in 1981. The number of cases throughout 1982 varied around 300 to 400 cases a month with the exception of the last 3 months where the figure reached 550 infections in November. This figure is probably due to the influx of new Thai prostitutes registered during this period.

The Gonorrhoea infection rate for Singaporean prostitutes was 7.2% compared to 8.4% for Malaysian prostitutes last year. There is thus a slight increase compared to 1981 figures. The PPNG infection rate for Malaysian prostitutes was 26.4% compared to 21.4% for Singaporean prostitutes. The Thai prostitutes who were in the scene during the last 3 months had a gonococcal infection rate of 8.9% and PPNG infection rate of 25.2%.

The incidence of infectious Syphilis has risen since 1980 and last year a total of 108 cases were seen. The explanation was given earlier.

The gonoccal isolation rate from Middle Road Hospital was 7.73% that from OPS being 5.81% and from GP being 5.20%. Compared to the previous year (1981) the figures amongst the three bodies are slightly closer. This will require further looking into.

Treatment changes in gonorrhea

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In 1937, Dees and Colston showed that sulphonamides were effective for the treatment of gonorrhoea. This was a notable advance but in later years it became evident that less than 25% of patients responded to the standard courses of therapy. With the introduction of penicillin in 1945, sulphonamides fell into disuse. The efficacy of penicillin was so great that some venereologists predicted that the major venereal diseases would be eradicated with its use. This has proved to be a fallacy for not only has there been an increase in incidence of gonorrhoea but also a gradual reduction of sensitivity of the organism to penicillin. This change in sensitivity of the gonococci is particularly encountered in the Far East and is further complicated by the emergence of strains of gonococci which produce B-lactamase. Fortunately, our therapeutic armamentarium has grown and other antimicrobial agents are available. This should, however, not encourage complacency. Indeed the ability of the gonococci to evolve into more resistant strains further emphasizes the need for vigilance. Indiscriminate use of antibiotics should be discouraged. This may halt the trend towards increasing resistance or even reverse it. There is some evidence that some countries eg the United States of America and the United Kingdom have been able to achieve this.

Principles of Treatment

The aim of treatment is to eradicate the gonococci as soon as possible.

The ideal antibiotic should be:

1. 100% effective.
2. Given as a single standardized dose
3. Available at reasonable cost.
4. Free of toxic, allergic and microbiologenic side effects eg. development of resistance and fungal overgrowth.
5. Capable of completely aborting simultaneously acquired or co-existing treponemal infection.

From an epidemiological stand-point effective treatment should be one which gives at least 95% cure rate.

Regimens of therapy need to be constantly reviewed to detect changes in antimicrobial sensitivity.

Treatment of uncomplicated infections

The following antimicrobial agents have been used in the treatment of gonorrhoea. Choice of a particular antimicrobial should be based on the prevailing antimicrobial susceptibility observed amongst the strains of organism in the population served.

1) **Pencillins** — Penicillin remains the drug of choice for the treatment of non-PPNG infections. It has been well tried and its efficacy is well established. Indeed the cure rates obtained with new antimicrobial agents must compare favourably with those obtained with pencillin or risk being labelled as "ineffective". 3 pencillin regimens have been well studied and shown to be highly effective. They are:—

Regimen	Reference
1. Benzyl penicillin 5 mega units plus 1 g probenecid	Rajan, 1972[4] Thin 1974[2], Evans 1980[3]
2. a) Procaine penicillin 2-4 mega units plus 1-2 g probenecid	Rajan, 1972[4] Taylor, 1975[5]
b) Procaine penicillin 4,8 mega units plus 1-2g probenecid	Smithurst, 1974[6] CDC recommendations 1979[7]
3. Ampicillin 2 — 3.5g plus 1 g probenecid	Rajan 1972[4] Karney 1974[8]

Other penicillins which have been used successfully include:—

Regimen	Reference
1. Amoxicillin 3 g ± 1 g probenecid	Karney 1974[8], Priice 1975[9], Nat. Gonorrhoea therapy monitoring study USA 1972-1978[10]
2. Pivampicillin 1.5 g plus 1 g probenecid	Rajan 1979[11]
3. Talampicillin 1.48 g plus 1 g probenecid	Prince 1977[12]
4. Indamy1 carbenicillin 1.5 g plus 1 g probencid	Rajan 1974[13]

2) **Aminoglycosides**

Regimen	Reference
Gentamicin 240 mg im	8-15% failure rate (Bowie 1974[14] and Morrison 1975[15])
Bekanamycin 1200 mg	6% failure rate (Rajan 1979[11])
Kanamycin 2 g	3% failure rate (Wilkinson 1967[16])

3) **Spectinomycin**

Regimen	Reference
Spectinomycin 2 g	2-8% failure rate (Rajan 1972[17] and Willcox 1974[18])

4) **Cephalosporin**

Regimen	Reference
Cephaloridine 2 g plus 1 g probenecid	8% failure rate (Brownlow 1974[19])
Cephalexin 5 g plus 1 g probenecid	4-50% failure rate (Landes 1972[20], Brownlow 1974[19])
Cephazolin 2 g plus 1 g probenecid	24% failure rate (Duncan 1974[22])
Cefuroxime 1-1.5 g plus 1 g probenecid	1.2% failure rate (Prince 1978[22])
Cefoxitin 2 g plus 1 g probenecid	0% failure rate (Jones 1979[23])
Cefotaxime 500 mg plus 1 g probenecid	3% failure rate (Rajan 1980[24])

5) **Tetracyclines**

Regimen	Reference
Tetracycline 0.5 g units total dose 9.5 g	5% failure rate (Judson 1976[25])
Doxycycline 300 mg	7-25% failure rate (Schofield 1974[26] Rajan 1979[12])
Minocycline 300 mg	4% failure rate (Shahichillah) 1975[27])
Minocycline 250 mg	60% failure rate (Rajan 1979[11])
Minocycline 500 mg	30% failure rate (Rajan 1979[11])

6) **Co-trimazoxole**

Regimen	Reference
Co-trimoxazole 8 tabs stat	4% failure rate (Rahim 1975[28])
Co-trimoxazole 8 tabs followed by 8 the next day	30% failure rate (Rajan 1979[12])

7) **Erythromycin**

Regimen	Reference
Erythromycin 1.5 g followed by 0.5g qds for 4 days	24% failure rate (Brown 1977[29])
Erythromycin 0.5g qds for 5-7 days	33% failure rate (Arya 1978[30])

Treatment of PPNG Infections

In late 1975, strains of gonococci which were completely resistant to penicillin began to emerge. Most of the strains had originated from the Far East and West Africa. The Far East strains are unique in their ability to transfer the B-lactamase R factor to other gonococci due to an additional conjugative plasmid. This is thought to account for the relatively high prevalence of PPNG in the Far East. The Far East strains are also more likely to be resistant to tetracyclines. Around 30% of the gonococcal strains isolated locally produce B-lactamase.

The followings antibiotics are thought to be effective against B-lactamase producing gonocci:—

1. Spectinomycin
2. Cefuroxime
3. Cefoxitin
4. Cefotaxime
5. Ceftriaxone
6. Kanamycin

7. Gentamicin
8. Rosoxacin) trial in
9. Amoxicillin/Clavulanic acid) progress

Spectinomycin

This is the drug of choice. Its present high cost, however, limits its use in developing countries. In vitro resistance is very rare although clinical failure with 2 g spectinomycin occurs at a rate of less than 4%. These treatment failures respond to a second injection of 4 g spectnomycin.

Cefuroxime

So far very cases have been treated cefuroxime. Of 8 patients given cefuroxime without probenecid, all 8 patients were cured (Arya 1978[30]). Blood levels are enhanced by probenecid.

Kanamycin

Cure rate of 98% has been reported in a retrospective study carried out in Singapore (Rajan 1979[31]).

Gentamicin

Cure rate of 91% was obtained with gentamicin given in a single dose of 280 mg im (Rajan 1980[32]).

Cefotaxime

Cure rate of 98% was obtained with 500 mg Cefotaxime plus 1 g probenecid (Rajan 1980[24])

Ceftrioxone — 100%. Cure rates obtained with 32.5 mg — 125 mg im plus 1g probenecid (Rajan 1982[34])

Rosoxacin — In vitro studies showed it to be effective against PPNG and most other isolates. It was less active against non — B-lactamase producing moderately resistant gonococci but this reduction was mostly small and unlikely to diminish clinical efficacy (Warren 1981[33]). A trial is in progress in Middle Road Hospital.

Amoxil/Clavulanic acid (Augmentin) — There is evidence that clavulanic acid markedly inhibits the effect of penicillin or ampicillin on PPNG strains[35] Previous studies done in Middle Road Hospital using single doses of 3 g Amoxil/125 mg clavulanic acid and 3 g Amoxil/250 mg clavulanic acid resulted in a high failure rate. A study is currently in progress using 2 doses of Amoxil 3g/250 mg clavulanic separated by a 4-hour interval

Middle Road Hospital treatment of non-PPNG infections — previous experience

Antibiotic	Failure rate %
Penicillins	
Procaine penicillin 2.4 mega units plus probenecid 1 g	4
Crystalline penicillin 5 mega units plus probenecid 1 g	Nil
Ampicillin 2 g, probenecid 1 g	Nil
Carbenecillin sodium 1.5 g plus probenecid 1 g	4
Pivampicillin 1.5 g plus 1 g probenecid	6
Aminoglycosides	
Bekanamycin 600 mg	35
Bekanamycin 1200 mg	6
Kanamycin 2 g	10
Cephalosporins	
Cefotaxime 500 mg plus probenecid 1 g	3
Tetracyclines	
Tetracycline 3 g	18.2
Doxycycline 300 mg	25
Oxytetracycline 2 g plus 250 mg	28
Minocycline 250 mg	60
Minocycline 500 mg	30
Other Antimicrobials	**Failure rate %**
Spectmomycin 2 g	2
Rifampicin 900 mg	8
Spiramycin 2.5 g	49
Thiamphenicol 2.5 g	16
Cotrimoxazole 8 tabs followed by 8 the next day	30

In the past procaine penicillin in varying doses of 0.6 — 1.2 mega units had resulted in a high failure rate of 30-40%. From 1978 till 1980, the dose

was increased to 4.5 mega units but the failure rate was still 25%. Clearly a new antibiotic was required. Kanamycin appeared to be a good alternative and between 1979 and 1981 it was frequently used. Between 1981 and 1982 Kanamycin became the recommended treatment. In 1981, however, there was a notable increase in the incidence of secondary syphilis from 68 cases in 1980 to 144 cases in 1981, a jump of 111%. Early latent syphilis also recorded a jump of 72.2% from 54 cases to 93 cases in 1981 (Middle Road Hospital annual report 1981). It was felt that the increase was due to the use of Kanamycin as first line treatment of gonorrhoea. As Kanamycin had no treponemocidal action it would appear that many patients had coexistence of simultaneously acquired syphilis. Further evidence for this comes from the observation of a decrease in the number of cases of early syphillis since the use of combination therapy. Our present therapy introduced in June 1982 comprises Kanamycin 2 g, Ampicillin 3.5 g and Probenecid 1 g. Latest data shows the combined failure rate for PPNG and Non PPNG infections to be 3% for male patients compared with 32% with Ampicillin 4g/Probenecid 1g and 10% with Kanamycin 2g. The present combination therapy has the advantage of being effective against most strains of gonococci, is relatively cheap and will probably abort concomitant syphilitic infections.

Present recommendations (Middle Road Hospital)
Uncomplicated Infections

A. Smear positive cases, culture results not ready

Ampicillin 3.5 g po)	
Probenecid 1 g po)	$2.10
Kanamycin 2 g im)	

B. Culture and sensitivities known

Non-PPNG

1. Procaine penicillin 4.5 mega units im $1.00
   Probenecid 1g po

or

2. Ampicillin 3.5 g – 4 g po $1.10
   Probenecid 1 g po

PPNG

1. Ampicillin 3.5 g po
   Probenecid 1 g po $2.10
   Kanamycin 2 g im

or

2. Spectinomycin 2 g im $13.00

or

3. Cefotaximo 500 mg im $10.75
   Probenecid 1 g po

or

4. Gentamicin 280 mg im $4.20

Complicated infections
Gonococcal PID, epididymo-orchitis

A. 1) Treatment on smear results, culture results not ready
2) For PPNG

Spectinomycin 2 g im daily for 5 days.

B. Non-PPNG

Procaine penicillin 4.5 mega units im
Probenecid 1 g orally followed by
Ampicillin 500 mg qds) x 5 days.
Probenecid 1 g orally)

Gonococcal ophthalmia neonatorum

A. 1) Treatment on smear results, culture results not ready
2) For PPNG

1% Kanamycin eyedrops – 2 drops to be instilled as follows:–

1st hour – every 10 minutes
2nd, 3rd, 4th hour – every 30 minutes
Thereafter for 60 hours – every 60 minutes

and

Kanamycin 0.5 g im stat
or

Cefotaxime 100 mg/kg im stat.

B. Non PPNG

Penicillin eyedrops 10,000 units/ml 2 drops to be instilled as follows:

1st hour – every 10 minutes

2nd, 3rd, 4th hour – every 30 minutes
– every 60 minutes

and

Crystalline penicillin 100,000 units 6 hourly x 4 doses

Both mother and father to have full investigations.

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Laboratory Diagnosis of Gonorrhoea

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The laboratory diagnosis of gonococcal urethritis in males and conjunctivitis have traditionally been based on the gram smear microscopy. For the diagnosis of cervicitis in females the cultural method has always been preferred because of its sensitivity. However, with the emergence of the penicillinase-producing Neisseria gonorrhoeae (PPNG), the gram smear microscopy is inadequate because of its inability to differentiate PPNG from non-PPNG strains. Hence the cultural method is increasingly being used, especially in PPNG-endemic areas.

The diagnosis and management of a patient with gonorrhoea involves a chain of events which influence the outcome of the control programme. For the programme to be cost-effective, each link in the chain must function optimally. A break in the efficiency of one of the links will adversely affect the efficiency of the other links in the chain. Over the years, it has been observed that a number of deficiencies recur frequently, and we wish to highlight them for discussion so that the isolation rate can be improved.

Most of the deficiencies come from clinics that have only a few patients in the programme. These clinics have an inadequate stock of the Neigon JEMBEC medium. The medium used is often old, dried and contaminated. Hence we often do not get positive isolates from these clinics. When unsuitable medium is used it is necessary to recall the patients for a retest. Misunderstanding can arise with the patients. The specimen should be well streaked across the surface of the whole plate so that isolated colonies are observed. If the streaking is only confined to a small area, then the growth is so confluent that it is impossible to detect colonies of gonococci amidst the commensals. After streaking, a carbon dioxide generating tablet is placed in the well of the plate, and the plate must then be enclosed in a plastic container. The inoculated plate should be despatched to the laboratory within 24 hours. Observance of these critical points will improve the isolation rate from private practitioners.

Ever since the programme started, there has been a steady decline in the prevalence of gonorrhoea in the prostitutes. This indicates that the programme has been beneficial. At the same time there have been changes in the susceptibility of the gonococcus to certain antibiotics. The percentage of non-PPNG isolated in this part of the world which are partially resistant to penicillin is about the highest anywhere. It is certainly far higher than what has been found in developed countries. Soon after the programme started there was an increase in the susceptibility of such strains to penicillin and other antibiotics. Thus the non-PPNG being isolated now require a lower concentration of penicillin for inhibition than previously. This is the result of using a standardised treatment regime for gonorrhoea. It is important that the treatment success rates of the regimes be constantly reviewed to make them relevant to the prevailing strains.

It is known that the resistance of the gonococcus to penicillin and tetracycline are genetically linked. It is therefore not surprising that strains in this region tend to be also fairly resistant to tetracycline. This accounts for the fairly high treatment failure rates seen with tetracycline in Singapore.

In recent years newer methods have been developed which are faster than the cultural technique in the diagnosis of gonorrhoea. This includes the limulus amoebocyte lysate method, which depends on the ability of the gonococcus cell wall to cause gelation of contents from the cells of the Horseshoe Crab. Another method, which is commercially available, is the use of an immunoenzyme method for the detection of gonococcal antigen in the urethral discharge. Both methods, however, have the disadvantage of being costly, and are unable to distinguish PPNG from non-PPNG strains.

Interpretation of Syphilis Serology and treatment of Syphilis

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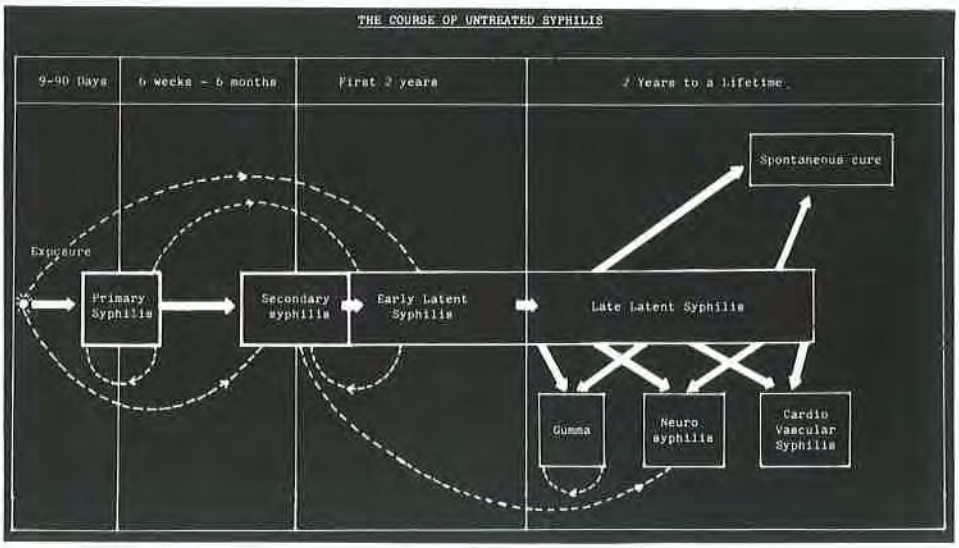
Syphilis Serology

Before attempting to interprete syphilis serology, one should understand the complex natural history of untreated syphilis.

The next stage calls for a good understanding of the various tests available, the principles of the tests, the uses and limitations.

Finally, the interpretation of positive tests in the light of the patient's historical and physical findings.

The natural history of untreated syphilis is summarized in the diagram below.



The Ideal Serological Test for Syphilis:

Sensitive
no false positives
no false negatives
consistently reproducible results
cheap
technically simple to perform

The various tests available are summarized in the following chart:

Serum Tests for Syphilis

Antibody	Antigen	Principle	Test
Non-specific (anti-lipoidal)	Cardiolipin	Complement fixation	CWR
	Cardiolipin-coated particles	flocculation	VDRL RPR ART
Group-specific (all treponemes)	Extract of non-pathogenic treponemes	Complement fixation	RPCFT
Specific (pathogenic treponemes)	Live T. pallidum	Immobilization	TPI
	Dead T. pallidum	Immunofluorescence	FTA-Abs
	Disrupted T. pallidum	Enzyme-linked immunosorbent assay	ELISA
	Disrupted T. pallidum coated on red cells	Haemagglutination	TPHA

CWR – Cardiolipin Wassermann reaction
VDRL – Venereal Disease Research Laboratory Test
RPR – Rapid Plasma Reagin test
ART – Automated Reagin test
RPCFT – Reiter Protein Complement Fixation Test
TPI – Treponema pallidum Immobilization test
FTA-Abs – Fluorescent Treponema Antibody Absorption test
Elisa – Enzyme-linked Immunosorbent assay
TPHA – Trephonema pallidum Haemagglutination Assay

(1) VDRL RPR etc

VDRL

1st described by A Harris in New York 1946.

Uses

- principal screening test for syphilis
- useful for epidemiological investigations
- quantitative VDRL; best serological test for assessing results of treatment, especially in early syphilis.
- a rising titre: (1) recent infection
  (2) in serofast patients: re-infection or sero relapse
  (3) newborns with congenital syphilis.

Advantages

Well standardized, inexpensive, widely available, easy to do, can be conveniently performed on large numbers of specimens.

Disadvantages

(1) insensitivity during certain stages of syphilis in:
(a) 25% of primary syphilis (overcome by serial testing)
(b) 25% cases of late latent and tertiary syphilis (significant drawback).

(2) occurrence of biological false-positive reactions
(a) Acute
(b) Chronic: incidence in 3 times greater than Titre can be high.

False Positives

1 Human and technical errors
2 Biological False Positives (acute/chronic)

False negatives

1. Human and technical errors
2. Prozone phenomenon with undiluted serum (in 1-2% of secondary syphilis)
3. Early primary, late latent and tertiary syphilis) .. order FTA-Abs or TPHA as a
   Congenital syphilis — early or late) **routine** if late syphilis suspected

Biological False Positives:

When VDRL positive
FTA-Abs/TPHA negative

Biological False-Positive Serum Reactions:

Type	Causes	Examples
Acute		
<6/12 duration	physiological	Pregnancy
	spirochaetal infections	leptospirosis, relapsing fever, rat bite fever
	viral infections	cytomegalovirus, herpes simplex, herpes zoster, infectious mononucleosis, measles, mumps, vaccinia, varicella, viral hepatitis, viral pneumonia, following viral vaccines (smallpox and yellow fever), TAB vaccine.
Chronic		
>6/12 duration	physiological	old age, some "healthy" individuals
	Chronic infections	Kala-azar, leprosy, malaria, lymphogranuloma venereum,
	Autoimmune disease	Systemic lupus erythematosus, polyarteritis nodosa, reheumatoid arthritis, Sjogren's syndrome, autoimmune thyroiditis, autoimmune haemolytic anaemia, etc.
	debilitated states	malnutrition, malignancy, liver cirrhosis, drug addiction.

(2) **FTA-Abs**

1st described by Deacon et al 1957

Grade	Microscopy finding	Interpretation
4+	Brilliant fluorescence	)
		)
3+	Very bright fluorescence	) Positive
		)
2+	Moderate fluorescence	)
1+	Weak, but definite, fluorescence	Borderline →retest
0	Vaguely visible or not visible	negative

Uses

- confirmatory test for syphilis
- more sensitive than VDRL in latent and late syphilis, thereby detecting cases missed by negative VDRL.
- retrospective confirmation of previously treated syphilis.

Advantages

- highly specific (false positives unusal)
- 1st test to become positive in primary syphilis

Disadvantages

- not useful for post-treatment follow-up (may remain positive for as long as 2 years after adequate treatment of early syphilis in 80% cases)
- more complex, costly, time-consuming.
- needs a fluorescent microscope and a skilled technician for interpretation.

False positive

Mainly in conditions associated with increased abnormal immunoglobulins, eg. autoim mune or connective tissue diseases.

SLE, Rhematoid Arthritis, Scleroderma, MCTD,
Drug addiction, viral infection, smallpox vaccination, pregnancy,
hepatic cirrhosis, lymphosarcoma, Autoimmune Haemolytic Anaemia,
(beaded fluorescence in SLE caused by anti-DNA antibodies)

False Negative

Some early primary, late latent and tertiary syphilis.

(3) **TPI**

Nelson and Meyer 1949.
Positive if >50% treponemas immobilized.

Advantage specific for treponematosis

Disadvantages

technical difficulties, length of time required to complete each test, high cost, dangerous to the technician.

False negative

1. Recent treatment with any penicillin or treponemicidal drug, eg. metronidazole.
2. primary syphilis, late congential and late syphilis eg. Tabes.

NB: TPI is being increasingly abandoned in most centres, including CDC.

(4) **TPHA (MHA-TP)**

Rathlev 1965

Uses: as for FTA-Abs
AMHA-TP most reliable and practical for mass screening of syphilis.

Advantages (compared to FTA-Abs)

Initial cost less, cheaper, easier to perfrom, can be automated, results can be read with naked eyes, easier to read, requires less skilled technician for interpretation, highly reliable and reproducible.

Disadvantages

less sensitive than FTA-Abs and VDRL in primary syphilis. Quantitation, though possible, not useful for post-treatment follow up.

False positive

very rare. in sera known to show biological false positives. In infectious mononucleosis (esp. in presence of high heterrophile antibody). In pregnancy, leprosy, connective tissue diseases.

False negative

early primary, late latent, tertiary syphilis.

(5) **ELISA**

enzyme-linked immunosorbent assay
relatively new test performed inside tubes or in wells in flat-bottomed micro-titre plates. These are coated with antigen prepared from a sonicate of T. pallidum to which the test sera are added, incubated, then washed. The antibody marker of anti-human gammaglobulin conjugated with the enzyme alkaline phosphatase is then added. After further incubation and washing, a measured amount of nitrophenyl phosphate indicator is added, and if the enzyme is retained, a yellow color is produced and measured spectrophotometrically.
Elisa evaluated by Veldkamp & Visser (1975) BJVD) – found test simple, reliable, relatively quick, its sensitivity in all stages of syphilis is equal to the FTA-Abs. Its specificity is probably also high.

Comparative sensitivity of VDRL, FTA-Abs and TPHA during various stages of syphilis

Stages	Comparative sensitivity
Primary	FTA-Abs > VDRL > TPHA
Secondary	FTA-Abs = VDRL = TPHA
Latent	TPHA = FTA-Abs > VDRL
Late	TPHA = FTA-Abs > VDRL

(1) **Serology of untreated syphilis**

Untreated Early Syphilis

FTA-Abs usually first test to become positive, within 3-4 weeks of infection. VDRL, TPHA following within the next 2-3 weeks. TPI becomes positive about 6-8 weeks following infection. All tests are positive in secondary syphilis (9-12 weeks of after infection). The titre in serology is highest in Secondary Syphilis except TPI, which reach its maximal titre after the seconadary stage.

Untreated late syphilis

The VDRL may become negative in 25% cases. The TPI may become negative in some cases of tabes dorsalis and late congenital syphilis, but in most cases it stays positive, as the other tests.

(2) **Serology of treated syphilis**

Treated Early Syphilis

VDRL titre drops or becomes negative within 6 months in primary Syphilis, in 12-18 months in secondary syphilis.

FTA-Abs and TPHA remain positive for a longer period, eg. 24 months or more.

Treated Late Syphilis

VDRL reversal is slow and variable
FTA-Abs, TPHA, TPI remain positive for life.

Interpretation of Serum Tests for Syphilis

VDRL	TPHA	FTA-Abs	Interpretation
0	0	0	No evidence of treponemal disease but could be in incubation period.
0	0	+	Early primary syphilis or old (probably treated) treponemal disease of long standing.
0	+	0	treated syphilis or false positive TPHA
0	+	+	treated early Syphilis or late treated or untreated treponemal disease

+	0	0	false positive VDRL
+	0	+	primary syphilis or problem serum* repeat required: if no change, TPI if available.
+	+	0	problem serum+: repeat required: if no change, TPI if available.
+	+	+	untreated or recently treated treponemal disease.

NB : 'Treponemal disease' means syphilis or yaws or other endemic treponematosis.
* could be false-positive FTA-Abs and therefore a biological false-positive reaction.
+ could be false positive TPHA, in which case a biological false-positive reaction, or a false-negative FTA-Abs.

APPROACH TO PROBLEM OF LOW-TITRE REACTIVE VDRL, REACTIVE FTA-ABS

Possibilities:

1 Late latent yaws
2 Late congenital syphilis
3 Latent syphilis: early or late, treated or untreated.

Management

1 Exclude yaws — Age group, historical background, place of birth
— Look for tissue-paper scars in legs, juxta-articular nodes, etc.
2 Exclude congenital syphilis — family history, no sexual exposure
— stigmata of late congenital syphilis eg. depressed bridge of nose, rhagades, Hutchinson's teeth, keratitis, deafness, sabre tibia, etc.
3 Ascertain stage of latency (if possible):
(i) early latent (<2 years)
(ii) late latent (>2 years)
— last sexual exposure, history of penile sore, previous VDRL record, etc.

If in doubt, manage as late latent.
Clinical examination for:
(i) CVS signs eg. Aortic Incompetence (Do CXR as indicated)
(ii) CNS signs eg. Argyl-Robertson pupils, Fundi optic atopy, Rhomberg sign, loss of vibration/position sense, are flexia (Do LP as indicated).
(iii) Skin or bone gummata.

If late complications detected, the diagnosis is tertiary syphilis; admit for management.

4 Check if there is past history of treatment for syphilis: If treatment adequate (documented), and no late signs — label as serological scar.

If treatment inadequate/suspect, manage as late latent as outlined.

5 Check family members, other contacts.

SEXUALLY TRANSMITTED DISEASES RECOMMENDED TREATMENT SCHEDULE

SYPHILIS

Early Syphilis
(Primary & Secondary)

1. Benzathine Penicillin 2.4 mega units I.M. one stat or weekly x 2 (1.2 mega into each buttock); or
2. Aqueous Procaine Penicillin 600,000 units I.M. daily 10-12 days (total of 6 to 7.2 million units).

Patients sensitive to Pencilillin

1. Tetracycline 500 mg 6 hourly x 15 days (oral) (taken 1 hour before meals); or
2. Erythromycin 500 mg 6 hourly x 15 days (oral)

Latent Syphilis

1. Benzathine Penicillin 2.4 mega units I.M. weekly x 3 injections;
   or
2. Aqueous Procaine Penicillin 600,000 units I.M. daily x 21 days.

Patients sensitive to penicillin

1. Tetracycline 500 mg 6 hourly x 21 days (oral)
   or
2. Erythromycin 500 mg 6 hourly x 21 days (oral).

Cardiovascular Syphilis (Admit patient to the Ward)

1. Benzathine Penicillin 3 mega units I.M. weekly x 3 injections
   or
2. Aqueous Procaine Penicillin 600,000 units I.M. daily x 28 days.

Neuro Syphilis (Admit patient to the ward)

1. Aqueous Procain Penicillin 2.4 mega units I.M. daily) x 28 days
   Probenecid 0.5 gm 6 hourly oral)
   or
2. Crystalline Penicilline 500,000 units 6 hourly I.M.) x 21 days
   Probenecid 0.5 gm 6 hourly oral)

Congenital Syphilis (Admit patient to ward)

First two years after birth
1. Procaine Penicillin 200,000 units I.M. daily x 10 days (or 500,000 units per kg body weight – total dose)

After 2nd year of life – treat as for in adults.

Treatment of all sex partners (and parents if necessary) after investigations

Follow-up after treatment

Clinical examination and serological tests at intervals for a total period of two years
(1 month; 3 months;
6 months; 12 months;
18 months; 24 months).

HOME STUDY SECTION
Respiratory Problems in Family Practice — Section 2 Pages 71 to 101

Pulmonary Infections

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This article is a review of 3 aspects of pulmonary infection:— pneumonia, lung abscess and bronchiectasis.

Pneumonia

Pneumonia continues to be an important cause of morbidity and mortality in the community. Pneumonia is an inflammation of the lung parenchyma caused by infection, allergy, chemicals and physical agents. However, in clinical practice, infection is the most common cause of pneumonia. The etiologic diagnosis of pneumonia in a patient can be a very difficult task as infection can be due to any one of a long list of micro-organisms, especially when the patient has been immunosuppressed or debilitated by chronic illnesses. The problem is compounded by the recent report of new infective agents such as Legionella and Atypical Legionella Like Organisms.

Classification

Pneumonia can be classified according to the pattern of lung infection (eg lobar, segmental and bronchopneumonia) or etiology. The division of pneumonia into lobar or bronchopneumonia is not very useful as many organisms can cause a similar pattern of infection in the lung. Although selection of the appropriate antibiotic is made easier by a diagnosis based on the etiology of the pneumonia, not infrequently, an etiologic diagnosis cannot be made because the sputum culture is negative. The main cause of a negative culture or a culture with a mixed bacterial flora is prior intake of antibiotics.

Causative Agents

Certain generalisations can be made regarding the etiology of pneumonia in the community and in the hospital environment. Pneumonia acquired by a previously fit person in the community is more likely to be due to pneumoncoccus, virus or mycoplasma. A patient in a hospital developing pneumonia is more likely to be infected by staphylococcus aureus, gram negative bacteria and anaerobic organisms. The same organisms may also cause infection in predisposed individuals such as the elderly and debilitated, alcoholics and diabetics, irrespective of whether their infection was acquired in the community or in hospital. The pathogens causing pneumonia in immunosuppressed patients include not only the common infective agents mentioned above, but also unusual or exotic organisms which are referred to as opportunistic organisms.

History

Pneumonia is suspected when a patient complains of fever, cough, pleuritic pain and dyspnoea. It should be borne in mind that fever may be absent in elderly patients and patients on steroids. Cough may be unproductive initially but subsequently the patient may produce yellow or rusty sputum.

Diagnostic Clues

The history may provide clues to the diagnosis of the etiologic agent. Patients with non bacterial infections usually have marked constitutional symptoms overshadowing the respiratory symptoms which are inconspicuous. Cough is usually non productive and if sputum is produced, it is mucoid. There may be a history of an outbreak of infection among close contacts or among members of the family.

Pneumococcal pneumonia is characterised by a sudden onset of chills followed by rigors and high fever. Herpes labialis is usually present. When pneumonia develops in alcoholics, diabetics, elderly and debilitated individuals, Kliebsiella pneumoniae is strongly suspected. In Singapore, pulmonary tuberculosis is not uncommon in these patients. Haemophilus influenzae is a cause of serious pneumonia in children below the age of 5 years. A history of steroid medication and immunosuppressive therapy is useful in alerting the physician to the diagnosis of rare or opportunistic infections. Anaerobic infection is suspected in patients with aspiration pneumonia which is seen in the following conditions:- loss of consciousness, diseases causing dysphagia, oesophageal disease, chronic oral sepsis and chronic sinusitis.

The occupational and social history is relevent

in diseases such as Q fever (contact with sheep and cattle), psittacosis (contact with birds) and Legionnaire's disease (association with construction sites and air conditioning systems). L pneumophila has been found in streams, ponds, soil, and in the water towers and condensers in air conditioning systems.

The geographic setting is important in diseases such as histoplasmosis and coccidioidomycosis as they may occur in patients who come from endemic areas eg certain parts of the United States and Mexico.

Physical Examination

The physical findings will depend on the site and extent of lung infection. Fever and tachypnoea are present and a pleural rub may be heard. Crepitations or bronchial breathing may be present depending on the stage of infection. Mental confusion is seen in the elderly. Cyanosis and septicaemic shock occur in patients with fulminant pneumonia.

Chest x-ray

A chest x-ray is usually ordered after the clinical examination and provides information on the following:—

(1) It confirms the suspicion of pneumonia and shows the site and extent of pneumonia
(2) It can provide diagnostic clues on the etiology of infection: small abscesses and pneumatocoeles indicate staphylococcal infection; involvement of dependent parts of the lung suggests aspiration pneumonia; apical involvement usually indicates tuberculous infection
(3) It may reveal the presence of underlying lung pathology eg bronchial carcinoma
(4) It may show whether complications have occurred eg lung abscess, pleural effusion, hydro-pneumothorax or lung collapse.

Laboratory Investigations

White cell count

Leucocytosis is seen in patients with bacterial pneumonia. The white count is normal or slightly elevated in patients with viral or mycoplasma infection.

Sputum Examination

Sputum should as far as possible be collected before starting antibiotic therapy. The sputum should be examined by smear and culture for pyogenic organisms and occasionally for fungi. A gram stain of the sputum can be very helpful in enabling diagnosis to be made rapidly eg when gram positive diplococci, clusters of gram positive cocci and gram negative coccobacilli are seen. The tubercle bacilli should not be forgotton especially in patients who have not responded after 1-2 weeks of chemotherapy. The sputum culture is usually negative when patients have received previous antibiotic therapy as the sensitive strains of bacteria are killed by the antibiotic. Not infrequently, the sputum culture may show a mixed growth of organisms, which are actually resident organisms from the upper respiratory tract and are not the cause of infection in the patient. Thus the results of sputum culture should be interpreted with caution and viewed in relation to the clinical state of the patient. Mucoid sputum should not be sent for culture.

Blood Culture

Blood culture is positive in 20-30% of patients with severe pneumonia. It is also useful when patients are unable to produce sputum for examination.

Pleural Aspiration

When pleural effusion is present, it should be aspirated and the fluid sent for culture.

Serology

Serological examination is useful in the diagnosis of viral, mycoplasma rickettsial and chlamydia infection. Legionnaires' disease can also be diagnosed by serology using the indirect immunofluorescent antibody test.

Skin Test

Skin test is useful when fungal infections eg aspergillosis, histoplasmosis ans coccidioidomycois are suspected.

Invasive Procedures

These procedures include transtracheal aspiration (TTA), fibreoptic bronchoscopy, percutaneous lung aspiration or biopsy and open lung biopsy. These procedures are performed when patients with serious infection do not respond to antibiotic therapy after 48-72 hours and sputum or blood cultures give negative results.

Different Types of Pneumonia

An outline of the etiology and main features of pneumonia is shown in Table I.

Lobar Pneumonia

Lobar pneumonia is usually due to Streptococcus pneumoniae or Pneumococcus. It is a gram positive diplococcus. Infection is spread through the pores of Kohn. Radiologically there is consolidation and air bronchogram is present.

Bronchopneumonia

Lobular or bronchopneumonia occurs when there is patchy involvement of the lungs. Infection may be due to virus, bacteria or mycoplasma. Bronchopneumonia occurring in the elderly and bedridden patient is usually due to retention of secretions and congestion of the lung bases giving rise to bilateral basal crepitations and patchy lung opacities. These patients have difficulty in coughing out sputum because of weakness and the retained secretions are invaded by organisms of low virulence from the upper respiratory tract. Infection is treated by broad spectrum antibiotics.

Staphylococcal pneumonia

Staphylococcus aureus is a gram positive coccus and causes a more serious infection than pneumococcus. Staphylococcal pneumonia is more often seen in debilitated patients, patients with underlying diseases and drug addicts. It may also complicate influenza infection.

Kliebsiella pneumoniae

This is a gram negative bacillus producing a severe pneumonia. The sputum in this infection is red and tenacious. Radiologically, the main features:— involvement of the upper lobes, bulging of the fissures due to intense exudation in the alveoli and abscess formation. Pleural effusion is not an uncommon feature.

Pseudomonas aeruginosa

Pseudomonas infection is seen more frequently in patients with tracheostomy and respirator therapy. The nebulizer attached to the respirator is a main source of Pseudomonas infection. Gram negative pneumonia occurs in patients with certain risk factors which are outlined in Table I.

Anaerobic Organisms

Anaerobic infection is usually due to aspiration of infected material from the mouth into the lungs. Infection of the lung may lead to pneumonitis, lung abscess, necrotizing pneumonia and empyema. Foul smelling sputum is usually produced. The diagnosis of anaerobic infection can be confirmed by obtaining sputum for culture which is uncontaminated by organisms in the mouth. This is done by transtracheal aspiration or bronchoscopy using a special double catheter.

Mycobacterium tuberculosis

The importance of m. tuberculosis as a cause of lung infection in Singapore cannot be overemphasised. It should be considered in any patient with pneumonia, lung abscess and bronchiectasis.

Legionella pneumophila

The organism causing Legionnaires' Disease is a gram negative coccobacillus. It is estimated that about 1% of pneumonias in the community and 4-10% in hospitalised patients are due to L pneumophila in the United States and it is considered after the big 3 causes of pneumonia in the community i.e. Pneumococcus, Mycoplasma and virus. The clue to diagnosis is a serious pneumonia with multisystem involvement. Diagnosis is confirmed by demonstrating a 4 fold rise in antibody titre by the Indirect Immunofluorescent Antibody test (IFA). A more rapid diagnosis is possible using the Direct Immunofluorescent Antibody test (DFA) to detect the organism in pleural fluid, sputum and lung tissue.

Non Bacterial Pneumonias

The infective agents include mycoplasma, viruses, rickettsia and chlamydia. The main feature of non bacterial pneumonia is the marked constitutional symptoms which overshadow the respiratory symptoms.

Mycoplasma pneumoniae

Primary atypical pneumonia is caused by Mycoplasma pneumoniae. Pneumonia develops in 3-10% of mycoplasma infection. Cough is usually unproductive and the sputum is mucoid. Pleuritic pain is uncommon. Patients may develop hemolysis (due to cold haemagglutinins) and other extrapulmonary manifestations. Radiological clearing may take up to 4-6 weeks.

Viruses

In adults, pneumonia is caused by influenza, parainfluenza and adenovirus. Viral infection is usually complicated by secondary bacterial infection. Rarely, primary influenza pneumonia may progress to a fulminant form with a high fatality. This is seen in patients with pre-existing diseases of the heart or lungs.

In children, pneumonia is caused by respiratory syncytial virus, parainfluenza virus, adenovirus, and measles. Chicken pox pneumonia is more likely to occur in adults.

Opportunistic Infections

The opportunistic organisms include the following:- Pneumocystis carinii, toxoplasma gondii, aspergillus, candida, crytococcus neoformans, cytomegalovirus. Infection by these organisms is seen in the immunosuppressed patients. Diagnosis of the etiologic agent usually requires an invasive procedure.

Management of Pneumonia

The general practitioner has to decide whether the patient can be treated in his home or be referred to a hospital. In general, the indications for referral to hospital are:-

(1) the patient is very young or old
(2) the patient has evidence of severe lung infection eg respiratory failure
(3) the patient has underlying disease of the heart and lungs
(4) the patient has not improved after 48 hours of chemotherapy

Management during the first 48 hours

Treatment consists of symptom relief and supportive measures eg controlling fever, correcting dehydration and relieving chest pain. Severe pleuritic pain can cause patients to suppress their cough and this can lead to retention of sputum, bronchial obstruction and atelectasis. A broad spectrum antibiotic such as ampicillin or tetracycline is prescribed to control infection as it is usually due to pneumococcus or mycoplasma. Erythromycin can also be used as it is effective against pneumococcus, staphylococcus and mycoplasma. In addition it is also effective against Legionella pneumophila which is important as a cause of pneumonia in a country such as the United States. When the etiology of pneumonia is known, the most appropriate antibiotic can be selected according to Table II. Dyspnoeic patients should be given oxygen beginning with 28% and increasing when the arterial blood gas shows severe hypoxia without carbon dioxide retention. Coughing should be encouraged in patients with underlying chronic bronchitis and excessive sputum. Patients with fulminant pneumonia and septicaemic shock should be given ampicillin, cloxacillin gentamicin and hydrocortisone intravenously.

After 48 hours

Effective treatment usually results in improvement within 48-72 hours. When there is no clinical improvement after 48 hours, the initial diagnosis should be reviewed. The results of sputum examination and blood culture should be checked and if an organism is detected, the antibiotic should be changed according to the sensitivity tests. Results of serological investigation may indicate a non bacterial pneumonia. When bronchial carcinoma is suspected, a bronchoscopy should be done. Patients with life threatening infections or infections in the immunosuppressed patients with negative sputum and blood cultures usually require invasive investigations to establish the cause of infection.

Complications of pneumonia

Infection may be confined to the lung (eg lung abscess) or it may spread to the pleura (pleural effusion) or to other organs eg arthritis meningitis, pericarditis etc. Jaundice is not uncommonly seen in patients with severe pneumonia and is due to toxaemia or hypoxia. Pneumonia may precipitate respiratory or cardiac failure in patients with underlying cardiorespiratory disorders.

Recurrent Pneumonia

Recurrent attacks of pneumonia should be investigated further as there may be an underlying disease of the lungs, oesophagus or a defect in immune defence mechanism.

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TABLE I: OUTLINE OF ETIOLOGY AND MAIN FEATURES OF PNEUMONIA

ORGANISM	CLINICAL FEATURES	CHEST X-RAY	INVESTIGATIONS	COMMENTS
Streptococcus pneumoniae	onset – sudden, chills, rigors, fever, rusty sputum, pleuritic pain, herpes labialis	segmental, lobar consolidation	sputum (gram stain), blood, pleural fluid	mortality rate 5%, higher in elderly, asplenia, sickle cell anaemia
Staphylococcus aureus	patients at risk: debilitated, pre-existing disease, immunosuppressed, drug addicts	multiple abscesses, pneumatocoeles	sputum, blood, pleural fluid	mortality rate 20% may complicate influenza infection, cystic fibrosis
Klebsiella pneumoniae	alcoholics, diabetics, debilitated individuals	predilection for upper lobes, bulging of fissures, cavitation, effusion	as above	mortality rate 50% TTA may be indicated
Pseudomonas aeruginosa	predisposing factors as above, association with respirator treatment	lobar or bronchopneumonia cavitation, effusion	as above	mortality rate 50 – 80% TTA may be indicated
Anaerobic bacteria	predisposing factors: coma, dysphagia, poor oral hygiene	aspiration, pneumonia, gravity dependent, areas – lung abscess, effusion	blood pleural fluid	TTA or bronchoscopy, to collect sputum free from contamination by anaerobes in mouth
Mycobacterium tuberculosis	severe infection of the lungs may be associated with few or no respiratory symptoms	upper lobes, involved especially in post primary TB – cavitation, effusion, military picture	sputum direct smear and culture	higher incidence in older patients, alcoholics, diabetics, immunosuppressed patients
Legionella pneumophila	tendency to affect middle-age or older patients – associated with construction sites and air-conditioning systems	bronchopneumonia, may have cavitation and effusion	blood (IFA – indirect fluorescent antibody test), sputum, pleural fluid, lung biopsy for DFA	DFA = direct immuno-fluorescent antibody disease – mild to fatal
Mycoplasma pneumonia	teenagers & young adults, marked constitutional symptoms, few respiratory	reticular shadows segmental or lobar consolidation	blood for antibody titre, cold agglutinins	may have extrapulmonary manifestations e.g. ear infections – hemolytic anemia
Influenza virus	symptoms similar to mycoplasma infection	segmental consolidation, bronchopneumonia, effusion rare	blood for antibody titre	primary influenza pneumonia may become fulminant especially in patients with pre-existing cardiac or pulmonary diseases
Respiratory syncytial virus	more common in infants and children, cause of bronchiolitis and bronchopneumonia	bronchopneumonia	blood for antibodies	
Chlamydia psittaci	symptoms similar to mycoplasma, cause of psittacosis	lobar or bronchopneumonia	blood – rise of antibody titre	contact with birds
Coxiella burneti	clinical features similar to above, cause of Q fever	segmental consolidation	blood – for antibody levels	may have endocarditis, splenomegaly, jaundice
Pneumocystis carinii	immunocompromised patients	bilateral perihilar reticulo-nodular shadows spreading outwards	TTA, bronchoscopy, open lung biopsy	invasive procedures if no response after 48-72 hours of treatment
Aspergillus	immunocompromised patients – fungus becomes invasive and disseminates	segmental shadow may spread extensively and cavitate		invasive procedures

TABLE II: ANTIBIOTIC THERAPY IN PNEUMONIA*

ORGANISM	DRUG OF CHOICE	ALTERNATIVE DRUG (S)
Pneumococcus	Penicillin	Erythromycin or Cephalosporin
Staphylococcus		
Penicillin sensitive	Pencillin	Vancomycin
Penicillin resistant	Cloxacillin	Methicillin
Klebsiella pneumoniae	Gentamicin plus Cephalosporin	Aminoglycoside plus Tetracycline
Haemophilus influenzae	Ampicillin	Chloramphenicol
Gram negative bacilli	Aminoglycoside	Carbenicillin or Cephalosporin
Pseudomonas auroginosa	Gentamicin plus Carbenicillin	Aminoglycoside (Amikacin) plus Ticarcillin
Proteus mirabilis	Ampicillin	
Serratia marcescens	Gentamicin	
Anaerobic bacteria	Penicillin	Metronidazole (Flagyl) Clindamycin Chloramphenicol
Legionella pneumophila	Erythromycin	Tetracycline Rifampicin
Mycoplasma pneumoniae	Tetracycline	Erythromycin
Chlamydia psittaci	Tetracycline	Chloramphenicol
Coxiella burnetti	Tetracycline	Chloramphenicol
Pneumocystis carinii	Septrin or Bactrim	Pentamidine isethionate

* Adapted from M A Mufson.
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Lung Abscess

Lung abscess is due to suppuration and necrosis of lung tissue. By convention, discussion of lung abscess is limited to infection caused by pyogenic organisms as causes such as tuberculosis, fungal infection, lung cancer are usually discussed under the respective diseases.

Mechanism of Infection

Abscess of the lung may arise from the following mechanisms:—

1 **Aspiration**
Aspiration occurs when there is inhalation of vomitus or infected material from the mouth into the lungs. Aspiration is more likely to occur in patients with impairment or loss of cough reflex due to coma, dysphagia, laryngeal palsy and oesophageal obstruction.

2 **Pyaemia**
Septic emboli may spread to the lung from any infected focus in the body. Rarely, secondary infection may develop in an area of pulmonary infarction.

3 **Complication of pneumonia**
Pneumonia due to certain bacteria may develop into lung abscess eg Klebsiella pneumoniae, Staphylococcus aureus, Anaerobic organisms.

4 **Trauma to lung**
Trauma to the lung may introduce infection through an 'open' chest wound or infection may develop in the traumatised lung where a hematoma has formed.

5 **Spread from sub-diaphragmatic focus of infection**
Infection in the liver may spread upwards to involve the lungs eg amoebiasis.

Clinical Features

Initially, the symptoms may resemble pneumonia. Subsequently when the abscess has formed, symptoms become more serious. The patient has a swinging fever and he may cough out large amounts of yellow or blood stained sputum. Putrid sputum is due to anaerobic infection.

Physical Signs

Consolidation is elicited over the site of infection. When there is obstruction to the bronchus leading to the abscess, the physical finding is that of collapse. After an abscess has communicated with a bronchus, crepitations and amphoric breathing can be heard if the abscess is situated just below the lung surface. Clubbing is seen in patients with lung abscess which has been present for some weeks.

Investigations

The white count is usually above 20,000 c mm. Sputum should be sent for culture and tuberculosis must be excluded. The sputum culture is not helpful when antibiotic therapy has been given previously.

Bronchoscopy

It should be done when there is a suspicion of bronchial obstruction. Bronchoscopy is also useful in obtaining pus for culture of anaerobes and removing as much pus out of the lung as possible.

Chest x-ray

A lung shadow can be seen which increases in size as the abscess enlarges. Aspiration lung abscess is suggested when the pulmonary opacity is situated in the apical segment of the right lower lobe or the anterior or posterior segments of the right upper lobe. These are the gravity dependent parts of the lung in the supine and right lateral positions containing a fluid level is seen when the abscess has ruptured into a bronchus. Similarly hydropneumonthorax is present when the abscess has ruptured into the pleural cavity. A malignant abscess usually has an irregular, thick walled cavity.

Differential Diagnosis

A pyogenic lung abscess should be differentiated from cavitated lung tumour, tuberculosis, amoebiasis, fungal infection eg actinomycosis and infected lung cysts.

Management

Medical treatment consists of antibiotic therapy and postural drainage. A non specific lung abscess is treated with penicillin injections in a dosage of 2-6 mega units for 4-6 weeks as the organisms are usually of low virulence and originate from the upper respiratory tract. Higher concentrations of penicillin (6-12 mega units) should be given when the sputum is putrid as this indicates that infection is due to anaerobic organisms. Flagyl 400 mg tds can be added when response to penicillin is not satisfactory. Postural drainage is important in ensuring that pus in the abscess cavity is let out.

Surgery

Most patients will respond to prolonged treatment with antibiotics. However, surgery is indicated when the abscess persists in spite of an adequate duration of chemotherapy or when an empyema has developed. Surgical treatment is necessary when the abscess is secondary to bronchial obstruction caused by a malignant lung tumour.

Prevention

Patients with chronic dental infections should be referred to a dental surgeon for treatment to reduce the risk of aspirating infected material. Any foreign body in the bronchus should be removed immediately by bronchoscopy or surgery if bronchoscopy fails.

Further Reading

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2 Baum G L ed. Textbook of Pulmonary Diseases. 2nd Edition. Boston. Little, Brown and Company, 1974.

Bronchiectasis

Bronchiectasis is a dilatation of the bronchi and is usually associated with retention of sputum and infection of the bronchial wall. Bronchiectasis of the upper lobes does not give rise to symptoms because the secretions can drain out under the influence of gravity. However bronchiectasis affecting other lobes produces symptoms of cough and sputum.

Etiology

Bronchiectasis may be congenital or acquired.

Congenital Causes

Infection of the bronchi may occur as a result of structural abnormality of the bronchial wall, dysfunction of the respiratory ciliary activity, defect in the quality and quantity of mucus produced and impairment of the immune defence mechanism. Examples of bronchial wall abnormality are bronchomalacia, sequestrated lung and Macleod's syndrome (or unilateral emphysema). Abnormality of ciliary movement occurs in patients with the Immotile or Dysfunctional Cilia Syndrome. Cystic fibrosis is an inherited disorder in which there is excessive production of mucus resulting in obstruction of the airways.

The Immotile Cilia Syndrome is an interesting condition which has been described recently. Patients with this syndrome have a defect in the the ultrastructure of the respiratory cilia which cause the cilia to be immotile or beat in a non effective manner. This in turn leads to impairment of mucociliary transport and eventually infection of the sinuses and bronchial tree. The most common abnormality noted is the absence of dynein arms on the outer microtubular doublets and this defect is also seen in the tails of spermatozoa in male patients who may give a history of sterility in addition to chronic respiratory infection. Kartagener's syndrome is thought to be a variant of the Immotile Cilia Syndrome and comprises the triad of sinusitis, situs inversus and bronchiectasis.

Immune deficiency occurs in patients with congenital hypogammaglobulinaemia and selective IgA deficiency. Recurrent infection leads to bronchiectasis.

Acquired Bronchiectasis

In the vast majority of patients, bronchiectasis is acquired either in childhood or adult life.

Childhood Infections

Many adult patients can trace their symptoms back to childhood after an attack of infection due to measles or whooping cough.

Damage to the bronchi may be due to the primary lung infection or a secondary bacterial infection. Bronchiectasis affecting the right middle lobe may be due to primary tuberculosis occurring in childhood. The enlarged tuberculous lymph node may compress the middle lobe bronchus causing obstruction and atelectasis followed by infection and bronchiectasis. Another cause of bronchial infection in childhood is inhalation of a foreign body.

Adults

In adults, any disease causing bronchial obstruction or destruction of the lung can lead to bronchiectasis. Examples include post primary tuberculosis, lung adenoma, chronic bronchitis and suppurative pneumonia. Proximal bronchiectasis is caused by allergic bronchopulmonary aspergillosis. However this disorder is not common in Singapore.

Clinical Features

The 2 main symptoms are cough and sputum. Cough is usually worse in the morning when secretions have accumulated in the lung overnight. The sputum is yellow in colour because of bacterial infection; sometimes it is blood stained. The sputum has a foul odour when there is infection

by anaerobic organisms. Dyspnoea or wheezing may occur in patients with co-existing disease such as chronic bronchitis or when complications have occurred eg collapse of the lung. Patients may also present with recurrent pneumonia or pleurisy.

Physical sign

Coarse crepitations can be heard over the lung bases. Localised crepitations may be heard distal to the site of bronchial obstruction. Clubbing is seen in patients with long standing or severe bronchiectasis.

Chest X ray

The chest x-ray may look normal in mild disease or it may show ring or tubular shadows. Sometimes cystic shadows can be seen with fluid levels in them.

Investigations

Sputum should be taken and sent for examination by direct smear and culture for pyogenic organisms and also for tubercle bacilli if indicated. Sputum cultures should be done frequently as the bacterial flora may change with repeated antibiotic treatment.

When immune deficiency or cystic fibrosis is suspected, the following investigations should be done:- serum electrophoresis, serum immunoglobulin levels, sodium concentration in sweat. In the Immotile Cilia Syndrome, a nasal biopsy or examination of spermatozoa or both should be done.

Bronchogram

It is an unpleasant procedure and is not without risk. It should be ordered for patients who may require surgical treatment. A bronchogram can show up the site and extent of bronchiectasis in the affected lobe and also the state of the bronchi in the other lobes of the lung.

Management

It must be emphasised that drainage of lung secretions is very important in treatment. Initially postural drainage should be done under the supervision of a physiotherapist as the patient has to be instructed on how to position his body to allow the secretions to flow out from the affected lobe. The frequency of drainage will depend on the amount of secretions in the lung, ranging from 4-6 times a day during an exacerbation of infection to 2 times a day (morning and night) when most of the secretions have come out.

Antibiotics

Infection is controlled by giving a broad spectrum antibiotic, (ampicillin or tetracycline) as it is usually due to Haemophilus influenzae or pneumococcus. Occasionally, infection is more serious and is due to Staphylococcus aureus or Pseudomonas aeruginosa. When this occurs, the patient should be admitted to hospital and given a course of Cloxacillin or gentamicin. It is not advisable to give patients continuous long term antibiotics as this will lead to the emergence of drug resistant organisms and superinfection by more virulent organisms or fungi.

Bronchodilator and steroid

Patients with wheezing may benefit from bronchodilator therapy. Thus it is useful to assess the patient's ventilatory function and the response to bronchodilator therapy.

When bronchiectasis is due to allergic bronchopulmonary aspergillosis, further bronchial damage to the bronchial wall can be prevented by giving long term prednisiolone.

Surgical Treatment

Surgery is indicated in patients who have severe symptoms not responding to medical treatment and in patients with recurrent haemoptysis. A patient is suitable for surgery when he has localised disease and his lung function is normal. Surgery is contraindicated when there is bilateral bronchiectasis or extensive disease and lung function is impaired by co-existing disease such as chronic bronchitis. Patients with severe hemoptysis who are unfit for resectional surgery may benefit from interventional radiologic therapy in which angiography is done (either pulmonary or selective bronchial arteriography or both) followed by embolisation of the enlarged bronchial artery by gelfoam.

Complications of Bronchiectasis

Patients may develop pneumonia, pleurisy, hemoptysis (which can be massive). Rarely brain abscess or amyloidosis may occur. The late complications of bronchiectasis are cor pulmonale and respiratory failure.

Prevention

As the genesis of bronchiectasis starts in childhood, any infection or respiratory complications should be vigorously treated. Children should be immunised against measles and whooping cough and any inhaled foreign body should be removed as early as possible.

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Pathophysiology of asthma

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Introduction

Asthma is a chronic pulmonary disease characterised by increased irritability of the tracheobronchial tree and manifested by recurrent episodes of more or less generalised airway obstruction. The condition is usually reversible either spontaneously or following administration of appropriate therapy.

The majority of laymen have little difficulty in knowing whether they have asthma. It is the physician who has the conflict and the main cause of this is the contemporary feeling equating asthma with airway hyperreactivity[1]. This will be discussed in greater detail later.

The current understanding of the pathogenesis of asthma is dealt with by considering allergic and nonallergic asthma although component factors of each may be operative in any one patient.

Allergic Asthma

Several surveys suggest that extrinsic antigens play an important aetiologic role in the majority of patients with asthma. In Munro Ford's review of 11551 cases of asthma in Australia[2], 53.7% of the asthmatics (and 63.4% of the asthmatics over age 14) were predominantly extrinsic as determined by history and skin testing. A study made in Tucson by Burrows et al[3] showed that 71.6% of adults with asthma had positive skin tests while in Singapore, positive skin reactions to house dust mite, feathers and human hair were high in 69% of asthmatics tested[4].

(1) Mediator release

Interaction between allergen and specific IgE antibody fixed to the surface of mast cells causes an interaction between IgE receptors and transmethylation of membrane phospholipids followed by movement of calcium ions into the cells. This triggers a chain of intracellular biochemical events resulting in the release of histamine, a kallikrein, eosinophil chemotactic factor of anaphylaxis (ECF-A), neutrophil chemotactic factor of anaphylaxis (NCF-A), other mediators and slow reacting substance (SRS-A). Phospholipase A activation results in liberation of arachidonic acid from phospholipids, which is then acted upon by cyclooxygenase forming prostaglandin G_2 and H_2 and from the latter are derived various other prostaglandins and thromboxane A_2[5]. The various prostaglandins all have different functions. PGD_2 and PGF_{2a} and thromboxane A_2 all cause broncho-constriction whereas PGE_2 is a bronchodilator.

Recent advances about the lipoxygenase pathway show that it metabolises arachidonic acid to give leukotrienes and SRS-A is now known to be a mixture of leukotrienes C, D and E. These three leukotrienes are potent bronchoconstrictors with relatively greater action on peripheral airways than on central airways. Leukotriene D by inhalation resulted in tightness and wheezing but this bronchoconstriction was slightly delayed in onset and was substantially prolonged as compared with that elicited by histamine[6].

(2) Dual response

Patients hypersensitive to an allergen on direct bronchoprovocation may show a dual response; the first phase is an immediate bronchoconstriction which occurs within minutes after exposure to antigen and subsides within one to two hours. Bronchoconstriction then recurs 4 to 6 hours after exposure and may remit again at two to five hours or more later. Such a reaction was observed in 8 or 15 asthmatics in one study[7]. The pathogenesis of this response has been clarified. Biphasic increases in circulating neutrophil chemotactic activity (NCA) coincide with the dual bronchoconstrictive response suggesting release of mediators from mast cells during both the early and late responses[8]. Earlier work[9] implicated the involvement of type III reaction in the late asthmatic response and late reactions also occurred in the absence of precipitins[7,10].

Therefore it would appear that type I immediate hypersensity (IgE-mediated) could cause both early and late bronchoconstriction via this circulating NCA and those without the late response lack the second peak of NCA in their blood. But the late response could still occur in the absence of this NCA if leukotriene D action or a type

III reaction took place in the airways. It has been suggested that the late airway obstruction (often more severe than the early) may have more relevance to the natural history of asthma than does the immediate response[10,11]. The late reactions last longer, are associated with marked lung hyperinflation, and are often more resistant to treatment than are the early responses; these features are more characteristics of "naturally-occurring" asthma[12].

Another important piece of work was the elucidation of allergen-specific IgG_4 antibodies associated with the late bronchoconstrictive response[13]. Patients with both specific IgE and IgG_4 antibodies developed a dual response on allergen bronchial provocation. Classically IgE antibodies mediate type I responses but an IgG antibody mediating the same is called the short-term sensitising (STS) IgG. It differs from IgE in that it is heat stable to 56°C, has a low affinity for mast cells, sensitises them for only two to six hours and is not capable of releasing as much of the mast cell histamine as IgE antibody. This STS-IgG was also reported to be associated with the late asthmatic response[14,15] and to belong to IgG_4 subclass[16].

NON-ALLERGIC ASTHMA

Also called intrinsic asthma, it is associated with infection, exercise, changes in weather (including cold temperature, variations in humidity and barometric pressure), cold air, emotional stress and inhalation of air pollutants such as sulphur dioxide or inert dust particles. Also included in nonallergic asthma are those caused by beta-adrenergic blocking drugs and aspirin sensitivity. Exercise-induced asthma and aspirin sensitivity will be explained in more detail.

(1) Exercise-induced asthma (EIA)

Exercise of one to two minutes' duration typically causes bronchodilation. If it is continued for five to eight minutes, bronchoconstriction may occur and is most extreme from five to ten minutes after completion of the exercise. Repeated exercise results in lessening of the bronchoconstrictive effect if the interval between exercise is less than one hour but not otherwise. This fact and the inhibitory effect of cromolyn on EIA support the importance of mediator release from mast cells in its pathogenesis. In those whose airway obstruction after exercise is in the small airways, cromolyn would be prophylactically useful whereas cholinergic blockade would prevent exercise-induced large airway obstruction[17]. Thus EIA may be mediated primarily by humoral agents in some asthmatics and largely by neurogenic mechanisms in others. Further it seems likely that cooling of the airways might cause nonspecific release of mediators from mast cells to provoke EIA[18].

(2) Aspirin sensitivity

Tied up with this are nasal polyps and bronchial asthma. This triad association was noted by Widal et al in 1922 and further, in 1937, in affected patients, the asthma was found to be often severe and unresponsive to treatment[19]. Between two and four per cent of asthmatics are sensitive to aspirin[20] and nasal polyps are present in a high proportion in such patients[21]. Patients with asthma who are aspirin-sensitive are commonly of the intrinsic nonatopic type[20] and this triad may be familial[22]. Other substances reported to produce similar reactions included morphine and morphine derivatives, codeine, amidopyrine, antipyrine and indomethacin[23] and also hydrazine yellow (tartrazine) used in food and drug colouring; paracetamol and phenylbutazone. The suggestion in 1975 that inhibition of prostaglanding synthesis may be important[24] is on firmer ground today now that minute doses of aspirin and indomethacin are known to block the cyclo-oxygenase pathway as discussed earlier in this paper.

BRONCHIAL HYPERREACTIVITY: The basic characteristic of asthma(?)

Bronchial irritability is even more characteristic of asthma than is allergy and can account for the bronchoconstrictive effects of many of the nonallergic causes of asthma. Bronchial irritability can be assessed by periodic inhalation of a bronchoconstrictive agent such as methacholine or histamine to determine the cumulative dose that elicits a decrease of at least 20% in forced expiratory volume in 1 second (FEV_1) as compared with the saline control[25]. Another bronchoconstrictive manoeuvre is the use of exercise[26] although this tends to be less sensitive than histamine or methacholine challenge[27].

Bronchial hyperreactivity is found in virtually all asthmatics and only 10 to 15 per cent of subjects with neither asthma nor allergic[28]. But in healthy subjects, increase in airway reactivity has been demonstrated following exposure to nonspecific airway irritants like cigarette smoke[29], viral infection[30] and air pollutants[31]. For asthmatics currently symptomatic, the degree of bronchial hyperreactivity is related to the severity of symptoms[32], the number of previous hospital

admissions[33], the ease with which asthma is induced by nonallergic and allergic stimuli, and can be correlated with the minimum amount of medication required to control symptoms[34,35].

Sensitised mast cells are present within the lumina of the airways. It is likely that release of mediators from sensitised mast cells within the airways opens up the tight junctions between the epithelial cells and facilitates access of mediators and antigen to the irritant receptors and mast cells found in profusion in the submucosa. Bronchial reactivity increases during the pollen season and following repeated experimental challenge by inhaled allergen[36]. Also the association between the onset of allergic asthma and recent viral respiratory infections in infants of allergic parents[37] support the above findings.

It is clear that whatever the pathogenesis, the preexisting level of nonspecific bronchial responsiveness of the host is a major determinant of the occurrence of early asthmatic responses to both allergic and nonallergic stimuli. And the effect of the provoking stimulus producing bronchospasm is dependent largely upon the preexisting "twitchiness" of the responding bronchi. The early asthmatic response to stimuli like that provoked by histamine testing, is not followed by an increase in nonspecific bronchial reactivity.

Late asthmatic responses are associated with the acquisition of increased bronchial reactivity which may extend over days and months. This has occurred on exposure to antigen[36], chemically reactive substances[38], to virus infection[39], and to ozone[40]. In effect these various conditions induce asthma as an acquired condition and raise the question as to whether asthma is generally acquired as a result of exposure to inducers, many as yet unknown.

There is now support for the theme that a variety of types of injury can have the common effect of an increase in nonspecific bronchial responsiveness. But there is also evidence to suggest that asthma is not just bronchial hyperreactivity but that hyperreactivity is a non-specific protective type of reflex which occurs secondary to injury of a membrane[41]. To this end it is thought that asthma is an inherited pathogenetic defect in the permeability of the mucous membrane to water and so acts as a sponge attracting water from the external environment. This supersaturation of the mucosa and submucosa of the asthmatic patient leads to progressive symptoms of asthma. That inhalation of distilled water induces bronchospasm in asthma but not in chronic bronchitis patients and normal subjects tends to support this hypothesis[42].

To blur the whole issue is the recent report that bronchial asthma can be present without increased airway reactivity[43]. Nine asthmatics were bronchoprovoked with acetylcholine and histamine but failed to show airway hyperreactivity. Is this fact or artifact is the subject of an editorial[44]

ROLE OF THE AUTONOMIC NERVOUS SYSTEM

Airway hyperreactivity could be due to increased parasympathetic or alpha-adrenergic activity or to decreased beta-adrenergic activity.

(1) Parasympathetic nervous system

Vagal nerves probably cause bronchoconstriction by release of acetylcholine at postganglionic endings on the muscle as attested to by this response being potentiated by acetylcholinesterase inhibitors[45,46] and blocked by atropine[45]. A mild degree of resting tone in the airway smooth muscles is present and maintained by vagal efferent activity. In patients with asthma and chronic bronchitis, atropine is reported to be equivalent in potency of bronchodilation to beta-adrenergic agonists[47,48]. Multiple receptors affect bronchomotor tone and include those in the nose, larynx, lungs and chemo- and baro-receptors located elsewhere. One of the most potent bronchoconstrictor reflexes is via stimulation of "irritant" receptors that lie immediately beneath tight junctions between epithelial cells in airways[49] by inhalation of dust, chemical irritants, drugs like histamine and by mechanical stimulation. Even irritation of the larynx and upper airway will cause reflex bronchoconstriction of the lower airways[50].

In asthmatics, the role of emotions on airways is assumed to be expressed through the parasympathetic nervous system and they may respond to psychological stimuli with bronchoconstriction[51-55].

The state of the airway epithelium may also be an important determinant of the vagal component[56] and damage to airway epithelium by infection, immunologic inflammation and irritants might sensitise airway sensory receptors and thus cause exaggerated reflex responses.

2) Sympathetic nervous system

Generally, sympathetic innervation to the airway smooth muscle is sparse although a small degree of sympathetic dilator tone normally exists in the airways. This bronchodilation is abolished with beta-adrenergic antagonists and in animals the

propranolol-induced mild bronchoconstriction can be relieved by vagotomy or atropine[57]. In healthy human subjects, after systemic or inhaled propranolol, no or very mild bronchoconstriction may develop[58-60]. In contrast patients with asthma may develop severe bronchoconstriction after propranolol[61,62] and unopposed parasympathetic activity is the main cause since atropine can antagonise this propranolol effect[62,63].

There is evidence that beta-adrenergic antagonists can modify airway smooth muscle responses and based on animal models, one theory of the pathogenesis of asthma proposes that the disease represents a diminished responsiveness of the beta-adrenergic receptors[64]. Although there is some evidence for adrenergic control of airway smooth muscle responses in humans, it appears that beta-blockade by itself is insufficient to cause bronchial hyperreactivity. The in vitro function of leukocytes from subjects with allergic asthma show a diminished response to beta adrenergic agonists[65]. To support this further, it has been demonstrated that asthmatic subjects only on theophylline preparations (and no beta-adrenergic agonists) had less lymphocyte beta-adrenergic receptors as compared to controls[66]. Also it was found that there was a relationship between greater severity of disease and less such lymphocyte receptors. In vitro and in vivo studies have reported receptor inactivation and/or beta-adrenergic unresponsiveness following the use of beta-adrenergic agonists but hydrocortisone may cause an increase in the numbers or restore the sensitivity to these agents[66].

Another recent discovery is the presence of autoantibodies to beta-2-adrenergic receptors in patients with asthma and allergic rhinitis[67] and this is proposed as another possible cause of adrenergic hyporesponsiveness in these patients. Since adrenergic hyporesponsiveness is also present in nonasthmatic atopic eczema patients untreated with beta-adrenergic agents, the hyporesponsiveness is not always due to adrenergic drugs but it is also not specific for asthma.

There are reports of alpha receptors in human airway smooth muscle that produce constriction when stimulated[68] and patients with allergic asthma were found to have significantly enhanced alpha-adrenergic responses when compared both to normals and patients with allergic rhinitis[69] but further study is required. Increased bronchial smooth muscle alpha-1-receptor activity is not the primary abnormality producing the variability between asthmatics in nonspecific bronchial hyperreactivity[70].

SUMMARY

While the major portion of this paper has focussed on the more recent trends in thinking about and research in asthma, the elementary basic principles of asthma pathophysiology are no less important. In brief, its pathology involves excess mucus, inflammation and bronchospasm in causing obstruction of airways; the control of airway calibre is dependent on mechanical, neural and humoral factors; and lung function tests exhibit air flow obstruction, hyperinflation, disordered gas exchange and an abnormal breathing pattern of hyperventilation and hypocapnia.

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Occupational Lung Diseases

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There are two main groups of occupational diseases of the lungs:

I. Pneumoconiosis
II. Others

I. PNEUMOCONIOSIS

This is a generic name referring to a group of lung diseases caused by the inhalation of different kinds of dust. The International Labour Organization defines pneumoconiosis as "the accumulation of dust in the lungs and the tissue reaction to its presence". Many of these dusts produce fibrosis in the lungs and disability. Some others cause radiological opacities without symptoms.

Patho-physiology

When dust enters the respiratory system, the coarse particles are trapped in the hairs of the nostrils. Finer particles are expelled from the trachea or bronchi by the action of the cilia which line the mucous membranes of these structures. Some of these particles are trapped in the mucous secretions of these tubes and coughed out as phlegm.

The finest particles, of five microns or less in diameter, reach the lower parts of the bronchial tree and alvecli.

The most dangerous of such particles are those between one to five microns. Those below one micron in size diffuse readily through the alveolar wall and are removed by lymphatic drainage from the lungs. However, particles of one to five microns require phagoytosis by the macrophages in the alveoli, around the respiratory bronchioles or in the regional lymphatic ductules or nodes. Under certain circumstances, as we shall describe in this chapter, fibrosis develops in the lungs.

The development of disease after dust inhalation depends on the following characteristics of the dusts:

(1) Chemical nature. Some dusts are virtually harmless; others are very fibrogenic.
(2) Size (as already discussed above).
(3) Shape. Dust in the form of fibres, e.g. asbestos, may be harmful to the lungs.
(4) Concentration. Other things being equal, a greater concentration of fine dust will produce more damage than a smaller concentration.
(5) Duration of exposure. The onset and extent of disease depend largely on the duration of exposure to the dust and its concentration.

Lung function tests

Lung function tests mainly test one or more of the following functions of that organ:

(1) **Expansibility of compliance** (bellows-like action of the lungs)
Examples: Vital capacity, forced vital capacity (FVC).
The forced vital capacity is defined as the maximum volume of air that can be expelled with maximal effort after full tropiration. Fibrosis in the lung parenchyma for example, may cause a decreased capability of the lungs to expand.
(2) **Expiration** (the expulsion capability of the lungs)
Example: Forced expiration volume timed over one second ($FEV_{1.0}$) or over ¾ second ($FEV_{0.75}$). The FEV is defined as the maximum volume of air that can be expelled in a given time with maximum effort after filling the lungs completely.
(3) **Exchange of gases** (diffusion across the alveolar membrane)
Examples: The concentrations of oxygen and carbon dioxide in the blood.

Kinds of pneumoconiosis

The common forms are due to inhalation of mineral dust, and include:

(1) Silicosis
(2) Asbestosis
(3) Coal workers' pneumoconiosis (coal dust pneumoconiosis)

Less common forms include:

(4) Graphite lung
(5) Talcosis
(6) Pulmonary siderosis
(7) Pneumoconiosis due to non-fibrous silicates

(1) **Silicosis**

Silicosis is probably the commonest pneumoconiosis in the world. In developed countries, it is uncertain whether silicosis or asbestosis is more common. In developing countries however, there is little doubt that silicosis is far more common than asbestosis, as asbestos has not been extensively used for many years there.

Exposed workers

Siliceous matter is widespread. Most of the earth's crust is composed of free silica. The sand on the seashore and in the desert is made up of it. The sand found in nature, however, is too coarse to be retained in the lungs. It is Man who has produced the danger of silicosis for himself. There are many processes which cause the breaking up of sand or other material containing silicon dioxide into minute particles of less than five microns. These include granite quarrying, sandblasting, fettling of metal castings and making of pottery and china.

Patho-physiology

Silicosis is caused by the inhalation of fine particles of crystalline free silica also called silicon dioxide (SiO_2). These particles are engulfed by the phagocytes in the alveoli and carried to the lymphatics around the alveoli and the hilar lymp nodes. Many of the phagocytes die after ingesting the silicon dioxide particles. Initially, a fibrogenic response develops in the minute lymph follicles adjacent to the terminal bronchioles. Eventually, large areas of the lungs may develop fibrosis.

Under the microscope, the typical lesion in silicosis is the onion-like whorled appearance of a silicotic nodule. This is formed of free particles of silica surrounded by dense concentric layers of collagen fibres which often become hyalinised, mixed with a scattering of reticulin fibres. The nodule is usually well-defined. Later on, the nodules may coalesce.

Emphysema and lymph nodes hardened by fibrosis are common. Sometimes calcification in the lesions occurs. The pleurae are characteristically thickened and adherent.

In advanced stages, enormous and enlarging masses of fibrous tissue are present (progressive massive fibrosis).

The pathogenesis of all these changes is uncertain. Various theories have been put forward, to explain the action of silicon dioxide on the lung tissues, whether it is mechanical (physical), chemical or immunological. It is probable that there is at least partially an immunological basis.

Clinical aspects

A period of time, usually five to ten years, lapses between the first exposure and the onset of symptoms. This is called the latent period, and its duration varies with such factors as the concentration of dust, the silica content of the dust and the susceptibility of the person.

Symptoms

There may be no or few symptoms until there are widespread areas of fibrosis involving a large proportion of the lungs. Then breathlessness and sometimes chest pain may occur. Cough, wheeze and excessive sputum are usually not prominent without an associated bronchitis.

Signs

There are surprisingly few signs, even when many nodules and masses are apparent on the X-ray, unless there is a concomitant bronchitis. They may be signs of emphysema. Cyanosis is a late sign. Deviation of the trachea due to enlarged lymph nodes may occur. There are usually no adventitious sounds unless chronic bronchitis or tuberculosis is present. Finger clubbing is absent.

Complications

(1) **Tuberculosis** is a common and important complication.
Silicosis seems to predispose to tuberculosis. Often many of the symptoms and signs in silicotic patients are due to tuberculosis, which is a common cause of death in these people.

(2) **Chronic bronchitis** is fairly common in the advanced stages of silicosis, leading to emphysema and sometimes spontaneous pneumothorax.

(3) **Cor pulmonale.** Right ventricular failure may supervene.

(4) **Rheumatoid arthritis** may be present. Sometimes, there may be the presence of the rheumatoid factor without any arthritis.
Silicosis does not seem to predispose to lung cancer.

X-Ray changes

Early on, there are fine, discrete nodules. Later there are miliary shadows ("snow storm" appearance) when the whole of the lung fields are affected. Large shadows appear on coalescence of individual nodules. Calcification may be present in the lungs or lymph modes in the hilar or peritracheal areas.

Lung function tests

There are no characteristic features. If X-Rays show only partial involvement of the lungs, lung function tests usually give normal results, unless there is an accompanying bronchitis.

When there is progressive massive fibrosis, there can be both a reduction of the vital capacity and the expiratory volume. These finds are due to a combination of the restrictive effect of the severe fibrosis and the obstructive effect of the emphysema from a complicating bronchitis.

Laboratory tests

It is advisable to do a sputum test to exclude complicating tuberculosis. An electrocardiogram should also be done to ascertain if the right ventricle is affected.

Management

There is no specific treatment of any proven benefit for silicosis. Appropriate treatment should be given for tuberculosis, bronchitis or other complications. Otherwise only symptomatic treatment is possible.

Prevention and control of silicosis

As for other dust diseases, the prevention of silicosis depends mainly on the use of wetting methods and ventilation. In biological monitoring X-Rays and lung function tests are very useful.

(2) Asbestos

Asbestos is widely used, especially in industrialized countries. Asbestos has a lot of useful properties, such as resistance to heat, fire and acids.

Asbestos is used in the form of sheets, pipes, ducts, tiles, jointings, gaskets, textiles, paints, moulds, brake linings of vehicles and other articles. The construction industry consumes fifty percent or more of all asbestos used.

Chemical composition of asbestos

Asbestos is the name given to a group of fibrous silicate compounds, largely containing magnesium but often other elements as well. There are two main types of asbestos:

(1) **Chrysotile** (white asbestos)
(2) **The amphiboles —**
  (a) Crocidolite (blue asbestos)
  (b) Amosite (brown asbestos)
  (c) Anthrophyllite
  (d) Tremolite
  (e) Actinolite

The different amphiboles contain elements such as iron and calcium, in addition to magnesium.

Chrysotile accounts for ninety to ninety-five percent of all asbestos used. All the kinds of asbestos are composed of long fibres, ten to fifty microns in length.

Many cases of illness and death have been caused by the inhalation of fine asbestos dust. In many developed countries, the morbidity from asbestos exceeds that from silicosis. This is not true yet of most tropical and other developing countries.

Exposed workers

Many kinds of workers are exposed to asbestos. They include those mining asbestos, making asbestos products, and handling them in lagging and other operations. Lagging activities are widespread in the making, repairing and breaking of ships and the construction industry. Workers transporting, unpacking and disposing of asbestos products may also suffer from asbestos exposure.

Patho-physiology

The known ill effects from the inhalation of fine asbestos dust are:

(1) Fibrosis of the lungs (called asbestosis)
(2) Lung cancer
(3) Malignant mesothelioma of the pleura and peritoneum.

Asbestosis

Long fibres of asbestos can get into the fine bronchioles and alveoli, as long as their diameter is small enough. Some of them are phagocytosed. The fibres penetrate into the peribronchiolar spaces or are carried there in the cytoplasm of the alveolar phagocytes. The alveolar walls and peribronchiolar areas develop extensive fibrosis. There is destruction of the normal tissue architecture and interference with the blood supply to the alveoli. The thickened alveolar membranes interfere with the gaseous exchanges across them.

As the disease progresses, there is fibrous thickening of the interalveolar and interlobular septa and pleura. The fibrosis is characteristically most marked in the lower lobes. Fibrous or calcified plaques develop quite commonly in the pleura, pericardium and diaphragm, usually many years after onset of exposure.

Microscopically, peribronchiolar nodules, extending into the adjacent alveoli, are seen. The fibrosis of the bronchiolar and alveolar walls is diffuse. The nodules do not have dense and hyaline collagenous fibres as in silicosis. They are

more cellular, due to a greater infiltration of mononuclear phagocytes. In contrast to silicosis, emphysema is uncommon and the hilar lymph nodes are not enlarged.

After inhalation of asbestos dust, **"asbestos bodies"** can often be found in the sputum in asbestos nodules or even in the abdominal cavity. They are golden yellow and elongated in structure, beaded along their length and bulbous at the ends. Each body consists of an asbestos fibre coated with an iron-protein complex. It must be pointed out that:

(1) Asbestos bodies do not have an invariable association with lung fibrosis, i.e. they indicate an exposure to asbestos and not necessarily asbestosis.
(2) Asbestos bodies require differentiation from similar bodies, formed as a result of inhalation of other substances, e.g. carborundum.

Lung cancer

There is an increase of risk to the squamous type of bronchogenic carcinoma. Most of the cases of lung cancer reported in asbestos workers were found in cigarette smokers. The combination of cigarette smoking and exposure to asbestos dust seems to have a summative effect in causing lung cancer, although asbestos exposure probably causes an increased risk of lung cancer even in non-smokers.

Mesothelioma

This is a malignant neoplasm of the serous cells of the pleura or peritoneum. About half the number of known cases have no history of exposure to asbestos. Exposure to crocidolite seems to be particularly dangerous. The peritoneal location is quite easily explained by the asbestos fibres having been transported there by either the lymph and blood vessels or via the retroperitoneal route. The tumour takes the form of huge whitish masses bulging from the pleura or peritoneum into the adjacent organs. It is possible that some other factor, in addition to asbestos, may be necessary for the development of mesothelioma.

Other cancers

There is some evidence to suggest that exposure to asbestos increases the risk of gastro-intestinal cancers.

Asbestos corns

Asbestos fibres may sometimes penetrate the skin and form corns or warts on the hands of forearms, which are usually symptomless. They merely indicate that the worker on whom they appear has handled asbestos.

Latent periods

As a very rough but useful "rule-of-thumb", the latent periods between first exposure to asbestos and subsequent development of symptoms are:

Asbestosis 10 years (more precisely five to ten years probably nearer five years than ten).

Lung cancer twenty years (more precisely fourteen to twenty-two years).

Malignant mesotholioma thirty years (more precisely thirty to forty-five years).

It must be stressed that cases of lung cancer or in workers exposed to asbestos do not necessarily have any pathological or clinical signs of asbestosis.

It should be noted, moreover, that the "latent period" is not necessarily the same as the period of exposure to asbestos. Lung fibrosis may progress even after withdrawal from all further exposure to asbestos. There is some evidence to suggest that sometimes very short exposures to asbestos, e.g. a few months, may lead to mesothelioma decades later.

Clinical aspects of asbestosis

The symptoms of asbestosis are insidious, the first being usually breathlessness on exertion. Cough occurs commonly and is dry or either slightly productive. Lassitude is common.

There is usually no chest pain. If there is, lung cancer or mesothelioma should be excluded.

Signs

Clubbing is frequent. Cyanosis is usually absent or light. In advanced cases, there is limitation of the expansion of the chest, especially at the lung bases. There may be an impaired percussion note at the bases. Fine crepitations occur early, either at the lung bases or in the axillae, and are heard best at the end of a deep inspiration.

Complications

Uncomplicated asbestosis does not usually shorten the life expectancy appreciably or cause gross disability. However, cor pulmonale may supervene and cause death.

Asbestosis does not appear to produce an increase in susceptibility to tuberculosis.

X-Ray appearances

Early cases present with fine mottlings or streakiness in the lower lung fields, especially in

the region of the costophrenic angles.

Advanced cases give a typically "shaggy heart appearance", due to the fibrosis of the pericardium and the overlying pleura. Calcified pleural plaques are common.

Lung functions

In early cases, there may be little or no impairment. In advanced cases, impairment of expansion of lungs, due to their encasement in dense fibrosis, may lead to reduction in the vital capacity. Alveolar fibrosis may cause interference of gas transfer.

Clinical aspects of lung cancer and mesothelioma

Very briefly, the symptoms and signs are those of malignancy, with cachexia, and localised signs due to pressure on or damage to affected structures. Any sudden change in the intensity or nature of symptoms or signs in a case of pre-existing asbestosis should alert the physician to the possibility of a superimposed malignancy.

Prevention and control of asbestos-related diseases

Many countries are now adhering to the limit of two fibres of asbestos per cm^3 of air (for all fibres above five microns in length) as a time-weighted average for an eight-hour day. However, a safe limit to prevent lung cancer or mesothelioma, may have to be much lower.

(3) Coal workers' pneumoconiosis

(anthracosis, coal dust pneumoconiosis or coalminer's lung)

Coal worker's pneumoconiosis is a lung condition due to exposure to coal dust. Coal is a mixture of carbon and inorganic materials. Coal deposits are usually found in siliceous formations. Therefore dust in coal mines commonly contains some free silica dust as well. However, recent epidemiological and experimental evidence indicates that pure carbon dust alone can cause coalwork's pneumoconiosis. The condition is more common in mines producing hard coal than soft coal.

Exposed workers

Coal-miners constitute the group of exposed workers.

Patho-physiology

There are two main forms of coalminer's pneumoconiosis:

(1) Simple
(2) Complicated

In general, the tissue response is not significant in simple pneumoconiosis, whereas it is considerable in the complicated variety.

(a) **Simple coalworkers' pneumoconiosis**

There are black indurated nodules usually in the upper portions of both the upper and lower lobes, with black dust particles, both lying free and in macrophages. Microscopically, the lesions commence as localized "macules" which are mainly peribronchiolar. The basic lesion is a star-shaped lesion with a profuse amount of dust but very little fibrosis. Each macule is made up of dust particles, macrophages and a slight proliferation of reticulin fibres. Collagen fibrosis is not conspicuous. Both types of fibres are randomly arranged, unlike the "onion skin" arrangement in a silicotic nodule.

The hilar lymph nodes may be black and slightly enlarged.

(b) **Complicated coalworkers' pneumoconiosis**

This condition develops in lungs already affected by simple pneumoconiosis. It is characterized by the development of a massive and increasing amount of fibrosis (progressive massive fibrosis, and occurs more in the upper halves and the posterior parts of the lungs. We have already mentioned that progressive massive fibrosis (PMF) can also develop in silicosis. However, contrary to what was thought a few years ago, free silica dust does not appear to be a pre-requisite for the development of PMF in coalworkers' pneumoconiosis. Other theories about predisposing factors, such as tuberculous infection or an auto-immune reaction, have also fallen into disfavour. One theory now held is that PMF is due to a loading of the lung with more coal dust than it can cope with, but this is still controversial. Severe emphysema may accompany PMF.

Clinical aspects

Symptoms

The simple form of pneumoconiosis is quite symptomless. The complicated form may cause cough and pain in the chest.

Signs

No characteristic signs occur in the simple variety. In PMF, these largely depend on the severity of the emphysema commonly present.

The trachea may be deviated and clubbing of the fingers may occur.

Complications

Pleural effusion or cor pulmonale may complicate PMF. The life expectancy does not seem to

be reduced in simple pneumoconiosis, but PMF may shorten life.

X-ray picture

In simple pneumoconiosis discrete and small shadows may be seen. In PMF large masses with gross distortion of the pulmonary architecture are present.

Lung functions

No impairment occurs in simple pneumoconiosis, but severe impairment can occur in cases of PMF.

Caplan's syndrome

It was found that rheumatoid arthritis has been associated with either type of coalworkers' pneumoconiosis. Rheumatoid nodules were found in the lungs. The syndrome of rheumatoid arthritis and pneumoconiosis was given the name of Caplan's syndrome, after its discoverer. Since then it has been found that exposure to a wide variety of dusts, including free silica and asbestos, can also cause this phenomenon.

(4) **Graphite lung**

The mining and processing of natural graphite can result in a kind of pneumoconiosis which may lead to disability after ten years or more. The presence of free silica dust, as occurs often in graphite mines, seems to aggravate the condition. No predisposition to tuberculosis appears to exist.

Some authorities consider it to be similar to coalworkers' pneumoconiosis. Recent work in Sri Lanka suggests that graphite dust does not cause severe disability, but some dyspnoea and cough in a proportion of workers after prolonged exposure.

(5) **Talcosis**

The pulmonary condition which arises from the inhalation of talc is called "talcosis". Talc is hydrous magnesium silicate.

The "talc" used in industry is of variable composition. Sometimes it may contain little or no talc as understood chemically. It may, for instance, contain some asbestos instead. In a tropical country, chemical analysis of the so-called "talc" used in the manufacture of powder to prevent materials, e.g. rubber sheets, from adhering together ("parting powder") showed a high content of quartz. Industrial "talc" is used in the rubber, textile, and paper industries; in the manufacturing of machines and rubber products, e.g. shoes and added to paints, ceramic tiles, cosmetics and other products. Hence large numbers of workers are exposed in the making or usage of "talc" powder.

Largely as a result of the wide interpretation of the word "talc" in industry, the study of the health effects of talc has been very difficult. "Industrial" talc dusts, when inhaled over several years have been known to cause fibrosis of the lung and an increased risk of lung cancer.

(6) **Pulmonary Siderosis**

After heavy exposure to pure iron dust for several years, a proportion of those so exposed may develop fibrosis of the lungs. Most workers with exposure to pure iron dust, however, will retain variable amounts of iron in their lungs without developing any fibrosis.

(7) **Lung diseases due to non-fibrous silicates**

Non-fibrous silicates include kaolin, fuller's earth and mica.

(a) Kaolin is largely a silicate containing aluminium. Pulmonary fibrosis has been reported, although it is uncertain whether it is due to kaolin itself or quartz and other contaminants.
(b) Fuller's earth contains calcium. Pure samples do not seem to produce pulmonary fibrosis. However, fuller's earth is often contaminated with quartz.
(c) Mica is the name given to a group of silicates. There is no definite evidence that mica can cause pulmonary fibrosis.

Radiological classification of the severity of pneumoconiosis

The I.L.O. (International Labour Organization) V/C classification is a system of recording the radiological changes resulting from the inhalation of all types of mineral dusts. It emphasizes the following features:

(1) Size of individual opacities.
(2) Profusion of opacities per unit area.
(3) Whether the opacities are rounded or irregular.
(4) Presence or absence of pleural thickening or calcification.

Even with all these criteria, there is often disagreement between experts as to which category a particular X-ray should fall into. Nonetheless, the classification is still useful for purposes such as the comparison of data obtained in studies from different countries.

II OTHER OCCUPATIONAL LUNG DISEASES

There are other lung diseases caused by inhalation of vegetable dusts but which are not usually placed in the group of diseases with the generic

name "pneumoconiosis". This is because there are no characteristic morbid anatomical features associated with the functional changes caused by these dusts. We shall now describe some of the more important ones of these diseases.

(1) **Byssinosis**

Byssinosis is a chronic disease of the lungs due to the inhalation of fine dust of cotton, flax or soft hemp. Fine dust of sisal can also probably cause the disease.

Exposed workers

Byssinosis is seen especially among cotton workers, particularly those engaged in the handling of raw cotton; for instance, in the ginneries, mixing and carding rooms and in bale pressing.

Patho-physiology

The aetiological agent in the dusts already mentioned is still not known. Many believe it to be a bronchoconstricting substance present in the pericarp of the leaves of the plant concerned. Others postulate the presence of a proteclytic enzyme from the plant. Cigarette smoking, respiratory infection and other atmospheric pollutants may play a contributory role.

No specific lesion has been found in the lungs. There is often pigmentation due to dust retention, infiltration of macrophages and other cells in the walls of the bronchi and bronchioles and the emphysena characteristic of chronic bronchitis.

Clinical aspects

In the early stages, there is typically a tightness of the chest, fever, breathlessness and an irritating cough. These symptoms tend to occur a few hours after onset of initial exposure. Thereafter, the symptoms subside despite continued exposure to the dust. However, a withdrawal of exposure for a few days, e.g. during a weekend, triggers off a relapse of symptoms, hence the name "Monday morning fever" also applied to it. It must be pointed out that there are other types of "Monday morning fever" unrelated to byssinosis.

Later, continued exposure may lead to the persistence of symptoms throughout the week. There may be a severe breathlessness and considerable disability. At this stage, byssinosis is indistinguishable from chronic bronchitis.

X-rays

There are no specific findings.

Lung function tests

In the early stages, the forced expiratory volume is reduced in the presence of symptoms. A continued decline in the FEV value may be an indication to remove a worker from all further exposure to the dust involved.

(2) **Farmers' lung**

Farmers' lung is an occupational pulmonary disease amongst dairy farmers. It arises through the inhalation of fungal spores in dust from hay, which is stored for the feeding of dairy animals. The storage occurs especially during winter months when fresh grass is not available. Therefore farmers' lung occurs most frequently in the winter season, and especially when hay is stored in poorly ventilated spaces. The usual conditions predisposing to farmers' lung do not prevail in tropical countries. In temperate countries throughout the world, however, this disease is one of the most frequent and disabling respiratory diseases among workers engaged in dairy farming.

(3) **Bagassosis**

Bagassosis is an occupational disease which affects workers inhaling the dust from the fibre of sugar-cane after sugar had been extracted (bagasse). Bagasse is used in the manufacturing of boarding for construction and insulation purposes. The disease manifests itself as an acute attack of fever with cough, dyspnoea and sometimes haemoptysis. It is believed to be a hyperimmune reaction to fungi in the bagasse.

(4) **Mushroom workers' lung**

Mushroom cultivation involves the process of making a compost. Mushroom workers may develop repeated attacks of upper respiratory illness. The acute signs and symptoms are very similar to those in farmers' lung. It is uncertain whether chronic illness can occur or not.

(5) **Suberosis**

Workers who inhale cork dust may develop an acute respiratory illness and sometimes chronic disease of the lungs. It appears that inhalation of dust from only mouldy cork would cause this disease, which is called suberosis.

(6) **Bird breeders' lung**

Persons exposed to the dust of the droppings of birds, such as chickens, parrots, pigeons and parakeets, may develop a respiratory illness with acute and chronic stages which resembles farmers' lung.

(7) **Other fungal diseases of the lungs**

Agricultural workers are liable to develop fun-

gal diseases, of which there are many kinds. Aspergillosis, which is caused by the **Aspergillus** group of fungi, usually occurs only in debilitated persons. Allergy to aspergillus antigens may occur and manifests itself as bronchial asthma. **Histoplasmosis** and **coccidioidomycosis** are two other examples of fungal disease which can cause pulmonary and other lesions.

(8) **Other vegetable dusts**

There is very little scientific work done on lung diseases which may be due to many kinds of vegetable dusts, especially those mostly prevalent in tropical countries.

(a) **Sisal dust**

Sisal is a hard fibre of the leaves of a plant, the American aloe, and is used chiefly for the making of ropes.

Inhalation of sisal dust may produce symptoms of bronchitis, with productive, cough, emphysema and sometimes breathlessness. Most authorities state that sisal dust does not produce lung fibrosis, but some others claim that it may do so.

(b) **Coir dust**

Coir is the fibre from coconuts, which are extremely common in tropical lands. It is used for a variety of purposes, including the making of mattresses, brushes and brooms.

To date there does not seem to be any definite evidence that coir dust causes any respiratory disease.

(9) **Occupational asthma due to vegetable dusts**

There are many wood and other dusts which can produce constriction of the bronchial tubes, thereby giving rise to the symptoms and signs of bronchial asthma.

FURTHER READING

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Differential diagnosis of diffuse pulmonary shadows

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The roentgenologic diagnosis of the chest begins with the identification of an abnormality on the chest film. The recognition of a pulmonary abnormality depends on the understanding of Absorption Co-efficient of body tissues, Radiological Contrast and Anatomy of the Thorax.

1) The Absorption Co-efficient of body tissues

In the human body, all tissues with the exception of calcium and fat, have the same atomic number and hence the same absorption co-efficient – that is about that of water. From this, it follows that all soft tissues of the lungs are equally radio-opaque, and that the difference in radio-opacity between one shadow and another, is not because the tissues casting them are different in nature, but because they are of different thickness in the line of the X-ray beam. Thus, faint shadows imply a thin layer, while a denser shadow will be a thicker mass of tissue.

2) Radiographic Contrast

As the soft tissues of the lungs have approximately the same Absorption Co-efficient, the outline of any structure can only be seen, or made out if it is adjacent to, or surrounded by, an environment casting a shadow of different radio-opacity – that is if there is adequate Radiographic Contrast between the structure casting the shadow and its environment.

3) Anatomy of the heart, lungs and pleura

Knowledge of the normal anatomy of the heart, lungs and pleura and the ways they respond physiologically to disease are essential in the interpretation of abnormal shadows in the chest film. It is useful to note that the pulmonary tissues can only respond to an insult in a limited number of ways; therefore, many diseases may look similar. Because of these limitations, clinical, laboratory and functional investigations have to be employed, together with past records, and old films are compared before a more accurate differential diagnosis of pulmonary shadows can be arrived at.

Clinical History

a) Symptoms of Respiratory Disease
- Cough and expectoration
- Shortness of breath
- Chest pain
- Haemoptysis

b) Past Illnesses and Personal History
- Family History
- Occupational and Residential History

Physical Examination

Presence of significant chest signs
Presence of extra-thoracic manifestations of pulmonary disease e.g. clubbing and pulmonary osteoarthropathy and cyanosis.

Bacteriology of Pulmonary disease

Smears and cultures
Animal Inoculation

Skin Tests

Biochemical Tests

Sputum, pleural fluid and blood or serum

Cytology

Malignant or non-malignant cells in pulmonary secretions and pleural effusions

Pulmonary Function Tests

Specialised Biopsy Procedures
- Bronchoscopic Biopsy
- Pulmonary Parenchymal Biopsy
- Pleural Biopsy
- Lymph Node Biopsy

Chest X-rays

A SUGGESTED APPROACH IN DIFFUSE PULMONARY SHADOWS

The following questions may be asked during the interpretation of a film while recognising a pattern of altered shadows in the lungs:

I) Is the disease multinodular?
II) Is the disease predominantly an air-space filling process?
III) Is the patient immune-suppressed, or on steroid therapy or has a known malignant neoplasm?
IV) Is the disease predominantly interstitial, reticular or granular?
V) Is the pulmonary arterial hypertension and/or pulmonary hyperinflation a dominant feature?

The Disease is Multinodular

Multinodular diseases are of two types — granulomatous and non-granulomatous.

Granulomatous lesions

Decision I — The shadows are usually approximately the same size (1 to 3 mm) and the margins are irregular. Are the granulomatous lesions due to any of the following list?
Miliary T.B.
Sarcoidosis
Silicosis
Fungal infection

Non-Granulomatous lesions

The shadows are larger, vary in size and usually have more regular margins.

Decision II — Are the non-granulomatous nodules metastatic?
— the nodules vary in size and rounded — so called 'cannon ball' shadows.

Decision III — Are the nodules due to uncommon non-granulomatous diseases?
e.g. Rheumatoid nodules
Wegener's granulomatosis
Eosinophilic granuloma
etc.

Decision IV — If the heart is enlarged and/or pulmonary vessels are congested, consider the nodular form of Congestive Cardiac Failure as a cause of non-granulomatous multinodular shadows.

Decision V — Is the patient very acutely ill, and febrile? Consider
1) Viral Infection
2) Septic Pulmonary Emboli.

The Disease is Predominantly an Airspace Filling Process

Decision I — Is pulmonary oedema present? Pulmonary oedema frequently shows as bilateral central or symmetrical shadows in the lungs, but dependent pulmonary oedema in one lung may occur. If the answer is yes, consider the following causes of pulmonary oedema:
a) Cardiac
b) Renal (uraemic pulmonary oedema)
c) Over-transfusion
d) Ingestion of water or toxic substances, e.g. drowning, paraquat poisoning
e) Heroin overdose
f) Neurogenic or 'Septic Shock Syndrome
g) Respiratory lung syndrome — oxygen intoxication
h) Fat emboli
i) Sudden ascent to high altitudes.

Decision II — Is the heart primarily responsible for the pulmonary oedema? There will be
a) Cardiomegaly
b) Pulmonary venous congestion and redistribution
c) Interstitial oedema (Kerley's lines)
d) Pleural effusion

Decision III — Is the heart failure superimposed on chronic obstructive disease? There will be in addition, superimposed nodular, reticular and mixed pattern shadows. Previous chest films and follow-up films are essential.

Decision IV — If the above causes of pulmonary oedema have been eliminated, then consider:
a) Pulmonary alveolar preteinosis
b) Haemosiderosis
(i) Secondary to mitral valvular disease

(ii) Primary haemosiderosis
c) Intrapulmonary haemorrhage
d) Eosinophilic lung syndrome (Type III immune response)

The Patient is Immune-Suppressed, or on Steroid Therapy or has a Known Malignant Tumour

Decision I — Is the disease due to the neoplasm itself?
Primary pulmonary carcinoma is always a localised process, which later causes local spread to the lungs. Other metastatic pulmonary diseases are the result of:
a) Haematogenous spread resulting in multiple nodules
b) Lymphangitic spread with linear shadows emanating from one or both hila and involving the peripheral septa.
c) Mixed multinodular and lymphatic spread type.

Decision II — Are the shadows due to reaction to a drug used to treat the malignancy?
Consider:
a) Methotrexate
b) Busulphan
c) Dilantin
d) Nitrofuradantin
e) Adriamycin

Decision III — Consider opportunistic infections in this group of patients
a) Pyogenic
b) Fungal
c) Viral
d) Pneumocystic Carinii
e) P.T.B.

Decision IV — Did the shadows develop after blood transfusion?
Transfusion reaction occurs particularly after white cell transfusion.

Decision V — Radiation reaction
Diffuse shadows develop after pulmonary radiation. The reaction may be early or late.

The Disease is Predominantly Interstitial, Reticular, Granular, Mottled or Linear but NOT Granulomatous.

Decision I — Are the shadows purely due to an interstitial pulmonary oedema from early heart failure?

Decision II — Could the patient be having a hypersensitivity reaction?
a) Type I immune reaction.
IgE binds with mast cells causing histamine release
e.g. extrinsic asthma,
anaphylactic reaction.
b) Type II immune reaction.
IgM – dependent or Arthus reaction type
e.g.
(i) Airborne – Extrinsic allergic alveolitis
– Farmer's lung
– Mushroom lung
– Bagassosis
– Bronchopulmonary aspergillosis
(ii) Blood borne – intrinsic allergic alveolitis
– Rheumatoid lung
– Lupus erythematosis
– Progressive systemic sclerosis
– Drug reaction.

The Disease shows Dominance of Pulmonary Arterial Hypertension and/or Pulmonary Hyperinflation.

Decision I — Is there emphysema?
Emphysema is present. Consider the types of emphysema.
a) Panacinar emphysema
b) Centrilobular type
i) Mitral stenosis
ii) Obliterative vascular disease
iii) Multiple pulmonary emboli
iv) Schistosomiasis
v) Idiopathic
c) Alpha 1 – antitrypsin deficiency type
d) Bullous emphysema.

Decision II — Is the patient asthmatic?
Asthma
– Mucous plugs cause areas of atelectasis in the lungs
– presence of pneumonitis precipitates asthma
– Allergic aspergillosis

Decision III — Is there chronic infection of the airways?
a) Chronic bronchitis – there are thickened bronchioles in the

chest films

b) Bronchiectasis – usually a local disease but may be generalised as part of an overall airway obstructive picture.
   i) Thickened irregular bronchial walls surrounding cystic spaces.
   ii) Pneumonia superimposed in bronchiectatic areas.

c) Cystic Fibrosis
   i) Young patient
   ii) Thickened bronchial walls
   iii) Localised hyperinflation
   iv) Superimposed, chronic infection and atelectasis.

CONCLUSION

And so the list of differential diagnoses goes on. It is apt to reiterate that the lung can only respond to insults of disease in a limited fashion. The result is abnormal opacities in the chest X-ray. Without the aids of patients' history, clinical examination, investigative procedures and results of previous chest X-rays, the diagnosis of diffuse lung shadows will remain inaccurate.

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Respiratory Emergencies

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It is useful to imagine the respiratory system to be a chain with many links, which aims to maintain tissue oxygenation and eliminate carbon dioxide. In the brain, the respiratory centres provide the drive and rhythm to breathing. These are transmitted through nerves to the respiratory muscles which in turn move the chest wall. The chest wall acts as a negative pressure pump which moves air in and out of the lungs. We can therefore envisage disruptions at various levels of the process:

1. Neuromuscular output
2. The Pump — Chest wall and Pleura
3. The Lungs — Airways
   — Alveolar interstitium
   — Blood vessels

We shall discuss four acute respiratory emergencies each involving a different level of the respiratory chain:

1. Acute Ventilatory Failure (Disease of neuromuscular output)
2. Spontaneous Pneumothorax (Disease of the respiratory pump)
3. Pulmonary Embolism and Infarction (Disease of the blood vessels)
4. Adult Respiratory Distress Syndrome (Disease of the alveolar interstitium)

Acute Ventilatory Failure (in patients with normal lungs)

Any diminution of the neuromuscular output to breathing may result in ventilatory failure. The causes include:

Barbiturate overdose	) Diminished Respiratory Centre Output
Opiate overdose	)
Encephalitis	)
Poliomyelitis	) Diminished Neuromuscular Transmission of Central Output
Guillain-Barre	)
Myaesthenia gravis	)
Respiratory muscle fatigue	)

Barbiturates reduce depth of breathing more than frequency, and opiates vice versa. In both Guillain-Barre and myaesthenia gravis acute ventilatory failure is a major complication which if undetected could result in death. Any patient who has been having prolonged laboured breathing from any cause risks developing respiratory muscle fatigue and subsequent ventilatory failure.

The effects of acute ventilatory failure are a raised arterial CO_2 tension ($PaCO_2$) from failure of CO_2 elimination, a reduced arterial oxygen tension (PaO_2), and a fall in pH. As renal compensation lags behind, the serum bicarbonate is normal or slightly raised. This is a typical blood gas picture of acute respiratory acidosis. The rapidity of onset is important, as the body can tolerate these changes better if they occur gradually. Cigarette smoking has an adverse effect, as raised carboxyhaemoglobin levels in smokers may prevent up to 20% of haemoglobin from participating in oxygen transport.

One may anticipate ventilatory failure when signs of respiratory muscle weakness and fatigue appear. The patient becomes tachypnoeic, is unable to take a deep breath, is unable to cough and may exhibit paradoxic inward motion of the abdomen during inspiration. The patient may also show alternating "rib cage breathing" and "abdominal breathing" much in the same way that a person carrying a heavy bag will use his right and left hands alternately to give the unused hand a rest. Once ventilatory failure sets in, clinical manifestations of hypoxaemia (mental confusion, tachycardia, systemic hypotension, cyanosis) and hypercapnoea (cerebral oedema) supervene.

The diagnosis is confirmed by arterial blood gas analysis, and the $PaCO_2$ is greater than 50 mmHg. Although raised $PaCO_2$ is the key feature to diagnosis, it is the accompanying hypoxaemia which is more life threatening. PaO_2 levels may fall below 60 mmHg. (while breathing air) partly due to hypoventilation, and partly due to secondary atelectasis resulting from shallow breathing and inability to clear secretions by coughing. In a patient at risk of developing ventilatory failure, one may monitor vital capacity* and maximal inspiratory pressures*, for these become abnormal before the blood gases. When vital capacity is less than 15 ml/kg body weight and maximal inspiratory pressure less than -40 cm H_2O, the patient's

ability to cough is impaired. When the vital capacity is less than 10 ml/kg and maximal inspiratory pressure less than −20 cm H_2O, ventilatory support is often required.

The moment ventilatory failure is recognised, oxygen should be administered freely while ventilatory support is being organised. As anaemia and hypotension jeopardise oxygen transport, transfusions with blood and fluids may be necessary. Endotracheal intubation is carried out with portex tubes (size 7-7.5 for females, size 7.5-8 for males) after anaesthetising the pharynx, and intravenous diazepam 10 mg if necessary. The cuff of the tube is inflated just enough to prevent back-leakage as higher pressures are associated with a higher incidence of tracheal damage. Pressure cycled ventilators may suffice if lung inflation is not made more difficult by airway narrowing (e.g. by secretions) or lung stiffness (e.g. from complicating pneumonia). However, as the concentration of delivered oxygen is not precise and the volume of each delivered breath may vary in pressure cycled ventilators, we prefer to use volume cycled ventilators. Tidal volume should be 10-15 m/kg and the frequency 12-20/min. The oxygen concentration delivered is the lowest level that will maintain PaO_2 at 70-100 mmHg. Arterial blood gases are monitored and the respirator settings adjusted accordingly. Chest x-rays are performed daily to check endotracheal tube position, and detect complicating pneumothorax or pneumonia. If stress gastrointestinal bleeding occurs, i/v Cimetidine 200 mg. six hourly is given. If ventilatory support is prolonged (> 2 weeks) a tracheostomy may be required. Adequate caloric intake with careful fluid and electrolyte balance must be maintained.

Weaning off the ventilator can be considered when

1. a trial period (20 minutes) of spontaneous breathing does not cause $PaCO_2$ to rise
2. minute ventilation during spontaneous breathing does not exceed 15 litre/min. (this degree of ventilation cannot be maintained in a patient who has had prolonged ventilatory support)
3. maximal inspiratory pressures exceed −10 cm H_2O
4. vital capacity is greater than 10 ml/kg or 1 litre

The process of weaning is first explained to the patient. Under close observation, the ventilator is removed and the patient put on humidified oxygen enriched air through a T-piece. If the pulse rises, the blood pressure falls, or the patient becomes distressed, the ventilator is re-connected. If the patient remains stable, blood gas analysis is done after 15-20 minutes of spontaneous respiration. If the blood gas results are satisfactory, the patient is further observed. It may be necessary to put the patient back on the ventilator at intervals and gradually prolong the periods of spontaneous breathing so as not to fatigue the respiratory muscles which have been accustomed to ventilatory support.

Spontaneous Pneumothorax

Spontaneous pneumothorax may be primary, when there is no known underlying lung disease, and secondary, when there is a known underlying disorder.

Primary spontaneous pneumothorax occurs typically in a tall asthenic young man. In the secondary type, the patient is generally over 40 years of age and has an underlying chronic obstructive lung disease or pulmonary tuberculosis. An interesting condition called catamenial pneumothorax occurs in women of reproductive age. Typically, the patient develops recurrent right side pneumothorax coincident with menstruation.

The commonest complaints are chest pain and dyspnoea. The physical findings include contralateral tracheal shift, increased percussion note and diminished breath sounds. It is important to remember that it may be impossible to detect these signs in a patient with airways obstruction where pneumothorax can only be excluded by a chest film. If the pneumothorax is small or indistinct on chest x-ray, an expiratory film will help make it more obvious.

In a young man with less than 20% lung collapse, chest tube insertion may be unnecessary, and the patient can be followed up until complete re-expansion occurs, usually in 2 to 4 weeks. Oxygen therapy may be used to accelerate pneumothorax resorption. If the pneumothorax is large, or when the patient's reserve is limited, intercostal tube drainage is indicated. The tube is inserted through the 2nd intercostal space at the mid clavicular line, and connected to an underwater seal. In the patient whose upper lobes are affected by pleural adhesions from previous tuberculosis, the tube is inserted in a lower space at the anterior axillary line. Check x-ray is performed to check tube position and show lung expansion. Re-expansion is occasionally complicated by ipsilateral pulmonary oedema. This may be asymptomatic but may produce hypotension in which case rapid intravenous infusion of crystalloids should be administered to restore circulation.

Generally, air drainage ceases in a day or two,

and the observation of a swinging water level in the tube during breathing and coughing confirms tube patency. If after a trial clamping of tube for a few hours the lung remains expanded on chest x-ray, the tube can be removed. Occasionally, one encounters a patient with a persistent pulmonary-pleural connection. This manifests itself as continual air leakage (> 5-7 days) when external suction is applied. Inspiratory-expiratory chest films may be helpful. When the leak is sealed, the expiratory film will show the pneumothorax to occupy a larger proportion of the hemithorax. If the leak is large, the expiratory film will show proportionate reduction in size of pneumothorax and lung. (See Fig. 1).

Persistent air leakage is often caused by underlying lung disease like emphysema or lung cysts. Two courses of management are possible here. One is surgical pleurodesis which is definitive. The other is continued and prolonged (up to several weeks) external suction (pressures from −5 to −25 cm H_2O) with or without chemical pleurodesis. This may be the only option for those who are unfit to undergo thoracotomy.

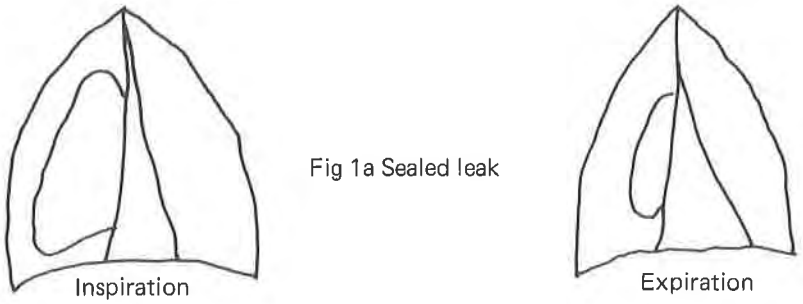

Fig 1a Sealed leak

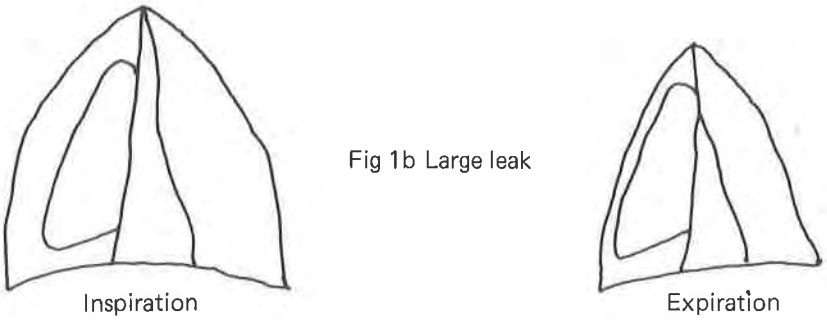

Fig 1b Large leak

Two situations which require special mention are tension pneumothorax and pneumothorax in the setting of positive pressure ventilation. Both may be rapidly fatal. Gas leaks into the pleural space with each inspiratory phase, but cannot leak back into the lung during expiration. Inspiratory-expiratory films will show contralateral mediastinal shift during expiration due to the valve effect. (See Fig. 2)

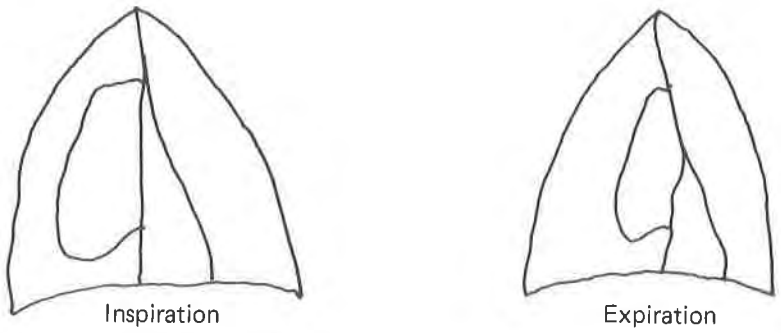

Fig 2

Avoid inflation pressures exceeding 80 cm H_2O during artificial ventilation, as chances of pneumothorax are high. Immediate intercostal tube drainage is mandatory and life saving.

About one quarter of pneumothoraces exhibit a small fluid level at the costophrenic angle or chest x-ray. This may be due to serous or sanguinous fluid and is of no clinical significance. However, in about 2% of pneumothoraces, significant haemothorax occurs and its early recognition is vital as patients can die of ex-sanguination. The treatment is restoration of circulation by blood transfusion, tube drainage of the haemothorax, and emergency thoracotomy to arrest the bleeding.

After a first episode of pneumothorax, a patient has a 30% chance of suffering another one on the same side. After a second episode, the chances of recurrence are so high that surgical pleurodesis should be performed.

Pulmonary Embolism and Infarction

Pulmonary embolism is often suspected on clinical grounds and treatment instituted on that basis. Predisposing factors include

1. chronically ill, elderly patients
2. immobilisation
3. recent surgery, especially pelvic
4. congestive cardiac failure
5. oral contraceptive use
6. thrombophlebitis
7. pregnancy and puerperium
8. malignancy

The patient complains of shortness of breath, chest pain and palpitations. Clinically there may be tachypnoea, tachycardia, loud P_2, hypotension, and evidence of deep venous thrombosis. Chest x-ray is usually normal but may show a raised hemidiaphragm, or enlarged pulmonary artery with distal oligaemia, or unilateral pleural effusion (which occurs only when infarction accompanies embolism). The pleural fluid is bloody and high in eosinophil count. Arterial blood gases show hypoxaemia ($PaO_2 < 60$ mmHg.) and hypocapnoea ($PaCO_2 < 40$ mmHg.) Electrocardiogram findings include tachycardia, right axis deviation, S-wave in Lead I, Q wave and T wave inversion in Lead III (S1Q3T3), and right ventricular strain pattern (S-T and T wave change in V1-V3). Ventilation-perfusion lung scans show loss of perfusion in the embolised area with preservation of ventilation to that area. Pulmonary angiography is most specific for diagnosis.

Pulmonary infarction occurs in only about 10% of patients with pulmonary embolism. These patients have pleurisy, gross haemoptysis, a pulmonary opacity on chest x-ray and a bloody pleural effusion (note that these features are absent in pulmonary embolism without infarction).

The treatment of pulmonary embolism is really the prevention of further embolic episodes. This is achieved by anti-coagulation. Intravenous heparin is used either as an infusion (approximately 1,000 units/hour) or as intermitten 4-6 hourly boluses (70-100 units/kg/dose). If the cause of embolism is reversible (e.g. post-surgery immobilisation) then a 10-14 day course of heparin will suffice. If however, there is a persisting predisposition toward recurrent embolism, then oral warfarin therapy is prescribed three days before heparin is discontinued. Warfarin anti-coagulation may be continued for 6-12 months.

Surgical removal of a major pulmonary embolism is a heroic measure and is only resorted to when the patient's chances of survival are considered slim on conservative management alone.

Adult Respiratory Distress Syndrome

This often appears as an unexpected onset of severe respiratory distress in a person with previously normal lungs. Typically, rapid shallow breathing progresses to frank dyspnoea, cough, crepitations and cyanosis. Chest x-ray shows interstitial and alveolar infiltrates (the picture of pulmonary oedema). Progressive enrichment of O_2 mixtures and assisted pressure cycled ventilation at higher and higher airway pressures become less and less effective in maintaining arterial oxygenation. The patient then dies of respiratory and circulatory failure. The initiating event may be trauma (and this may be non thoracic), septicaemia, acute pancreatitis, viral pneumonia or inhaled irritants. Increased pulmonary capillary permeability and exudation increase lung stiffness and ventilation is possible only with high inflation pressure. There is a profound impairment of oxygen exchange capability of the lungs, and this is largely due to shunting of blood through atelectatic non-ventilated alveoli. These patients therefore require volume cycled ventilators (which are capable of higher inflation pressure than pressure cycled ones). Positive end expiratory pressure (PEEP) may be necessary to prevent alveolar collapse and thus reduce shunting. This enables adequate oxygenation of the patient without using very high and potentially toxic levels of oxygen concentration ($>60\%$).

*Vital capacity is the total volume of gas that can be exhaled after a maximal inspiration. Maximal inspiratory pressure is the most negative pressure a patient can generate while attempting to suck in air from residual volume, and reflects inspiratory muscle strength. Both these tests can be performed at the bedside.

HOME STUDY PROGRAMME MCQs

MCQs on articles in Vol. IX No. 1 (January/March 1983)

Answers to these questions will be published in the next issue (Vol. IX, No. 3). If not fully satisfied with your answers and scores, why not read the articles and your books again?

1. In which of the following pulmonary diseases is finger clubbing a recognised feature:
   A Bronchiectasis
   B Asbestosis
   C Bronchial carcinoma
   D Primary pulmonary tuberculosis
   E Lung abscess

2. Clubbing **with cyanosis** is seen in:
   A Secondary polycythaemia
   B Chronic pulmonary fibrosis
   C Acute nitrite poisoning
   D Chronic emphysema
   E Congenital heart disease

3. Clubbing **without cyanosis** is seen in:
   A Ulcerative colitis
   B Compensated mitral stenosis
   C Subacute bacterial endocarditis
   D Occupational effect
   E Some healthy persons

4. Cyanosis
   A Due to lung disease is typically present in finger tips but not in the tongue
   B In the toes but not in the fingers is associated with a patent ductus arteriosus with a reversed shunt
   C Occurs more commonly in a person with polycythaemia than in a person with anaemia
   D Is usually present if the vital capacity is less than 80% of the predicted value
   E Is always associated with finger clubbing

5. Haemoptysis may be due to:
   A Bronchial adenoma
   B Bronchiectasis
   C Viral pneumonia
   D Mitral stenosis
   E Pulmonary embolism

6. Haemoptysis recurring at intervals over five years might well occur in:
   A Pneumoconiosis
   B Bronchial adenoma
   C Idiopathic pulmonary haemosiderosis
   D Sarcoidosis
   E Bronchiectasis

7. Breath sounds are diminished over a:
   A Consolidated lobe
   B Pneumothorax
   C Bronchiectatic lobe
   D Collapsed lobe with occluded bronchus
   E Pleural effusion

8. Which of the following statements is/are correct?
   A The left main bronchus is more vertical than the right
   B The right lung is divided into upper and lower lobes by the oblique fissure
   C The transverse fissue runs horizontally from the junction of the sixth costal cartilage with the sternum to join the oblique fissure at the tip of the scapula
   D The left upper lobe is largely in front of the lower lobe
   E The middle lobe is situated behind the right upper lobe

9. Cough
   A Is the most common of all respiratory symptoms
   B Is absent in laryngeal paralysis
   C Is usually a late symptom in bronchial carcinoma
   D Is one of the principal manifestations of left ventricular failure
   E May produce syncope

10. Chronic cough in children may be due to:
   A Recurrent bronchitis
   B Bronchial asthma
   C Chronic sinusitis
   D Inhaled foreign body
   E Psychological factors

11. Chronic cough in an adult with a normal chest X-ray may be due to:
    A Left-sided heart failure
    B Chronic bronchitis
    C Bronchiectasis
    D Gastro-oesophageal reflux
    E Bronchial asthma

12. The following associations are correct:
    A Epiglottitis and inspiratory stridor
    B Bronchiolitis and rhonchi
    C Lobar pneumonia and mucoid sputum
    D Chronic bronchitis and hypoxia
    E Pulmonary embolism and sudden dyspnoea

13. In acute laryngo-tracheo-bronchitis:
    A Symptoms come on rapidly
    B Severe dyspnoea is usual
    C Leucocyte count is always elevated
    D Prognosis is grave
    E If respiratory obstruction is imminent, oxygen and aminophylline is the treatment of choice

14. The following are likely to be associated with Streptococcal pharyngitis:
    A Hoarseness
    B Fever
    C Purulent tonsils
    D Enlarged cervical lymph nodes
    E All of the above

15. The physical signs of unilateral hydrothorax include:
    A Tracheal deviation to the affected side
    B Prominence of costal interspaces on the affected side
    C Dullness over the affected side
    D Diminished vocal resonance on the affected side
    E All of the above

16. In a patient with moderate obstructive pulmonasry emphysema, physical signs would include:
    A Distant heart sounds
    B Narrow subcostal angle
    C Prominent pulmonary second sound
    D Prolonged expiration
    E Hyper-resonance to percussion

17. Which of the following statements are true of chest X-rays:
    A A P-A film in maximal expiration is useful when a small pneumothorax is suspected
    B Tomography is of particular value in defining cavitated lesions of the lungs
    C Notching of the ribs indicates coarctation of the aorta
    D A lordotic A-P film is of value in suspected lingular consolidation
    E Lateral decubitus views can help identify small pleural effusions

18. In a P—A chest X-ray, the right hemidiaphragm is higher than the left in:
    A Normal persons
    B Paralysis of the right phrenic nerve
    C Right sub-phrenic abscess
    D Radiation fibrosis of the right lung
    E Right pleural effusion

19. Differential diagnoses in an adult female with an asymptomatic anterior mediastinal density found on routine chest X-ray include:
    A Substernal thyroid
    B Thymoma
    C Aneurysm of aortic arch
    D Bronchogenic cyst
    E Neurofibroma

20. Calcified lesions seen in a chest X-ray may be caused by:
    A Measles
    B Histoplasmosis
    C Psittacosis
    D Pertussis
    E Sarcoidosis

21. Percutaneous needle biopsy of the lung is contra-indicated in:
    A Pulmonary hypertension
    B Diffuse interstitial pulmonary disease
    C Bullous lung disease
    D Hamartoma
    E Respiratory insufficiency.

NEWS FROM THE COUNCIL

The Tenth WONCA World Conference on Family Medicine

The World Conference took place in Singapore from May 20 – 24, 1983, after 2½ years of meticulous planning and hard work by the Host Organising Committee. It was gratifying to note, from enthusiastic and warm response of delegates and accompanying persons, that the Conference was a success, in terms of both the Scientific Contents and the Social Programme. Meditech '83 too proved to be an unqualified success, in the view of delegates and participating exhibitors.

On-site Registration was overwhelming, swelling the number of delegates and accompanying persons to more than 1,800. It was heartening to note the registration of 80 local delegates. However there could and should have been more local participants.

For the Scientific Sessions there was a total of one keynote speaker, 14 plenary speakers and 115 free standing papers speakers. There was a larger than usual attendance for the open forums of the WONCA Standing Committees and enthusiastic response was noted for the special Seminar/Workshop on Cancer Prevention Programme, the Stress Management and the Balint Group. Attendance even on the last day of the Conference was very good.

The Opening Ceremony, officiated by the President of the Republic of Singapore, Mr C V Devan Nair, was witnessed by about 2,000 people. The "Curtain Raiser" on Family Medicine gave the Conference a beautiful start – the response from the participants was overwhelming. Delegates and guests were treated to a gastronomic spread and to the cultures and crafts of the East.

The Closing Banquet had to be held in two venues due to the large number of participants – the Neptune Theatre Restaurant and the Tropicana Restaurant. The President of WONCA, Dr David Game was installed at the closing ceremony.

Meditech '83, with 36 stalls, was successfully held despite competition. The exhibitors were very satisfied with the response from the visitors.

For the first time in the history of WONCA a book of abstracts and the Conference Proceedings were printed in time for the Conference. It was a real achievement for the Organising Committee and for the College A daily news bulletin was published for the five days of the Conference and again this was achieved only through hard work and dedication.

The Council thanks the Host Organising Committee and all members of the College who have given their best towards the success of the Conference – a significant milestone in the history of the College, and a recognised achievement in Singapore.

Annual General Meeting

The Twelfth Annual General Meeting of the College of General Practitioners Singapore will be held at the Academy Lecture Theatrette, Alumni Medical Centre, 4-A College Road, Singapore-0316, on Sunday, 26 June 1983, at 2.30 p.m. Office bearers for 1983-85 will be elected at this meeting. Lunch will be provided and it is hoped that many members will be present at the meeting.

Eleventh College Examination for Diplomate Membership

The Eleventh College Examination for Diplomate Membership will be held on:

Sunday, 13 October 1983 – Theory
Sunday, 20 October 1983 – Clinicals

The College Convocation and Annual Dinner and the Sixth Sreenivasan Oration will be held at the Hyatt Hotel, on Sunday, 6 November 1983.

Undergraduate Teaching Programme

Ten Didactic Lectures were held for the Third Year Medical Students at the Allen Lecture Theatre. The programme was as follows:

Lecture No.	Date	Topic	Lecturer
1	25.4.83	Opening Address	Prof E P C Tock, Dean, Faculty of Medicine
		General Introduction and Consultation.	Dr Moti H Vaswani
2	26.4.83	Human Development and Behaviour.	Dr Koh Eng Kheng
3	27.4.83	Health and Ill-Health in Relation to the Family and the Community.	Dr Leong Vie Chung
4	28.4.83	Disease Patterns in Family Medicine/General Practice.	Dr James Chang Ming Yu
5	29.4.83	The Whole Person of Medicine and the Whole Person in the Family and the Community.	Dr Frederick Samuel
6	3.5.83	Problems and Limitations in Family Medicine/General Practice.	Dr Lim Kim Leong
7	4.5.83	Health Maintenance and Preventive Medicine in Family/General Practice.	Dr Lee Suan Yew
8	5.5.83	Affective Skills in Family Medicine/General Practice.	Dr Victor L Fernandez
9	6.5.83	The Family Physician and the Law.	Dr Moti H Vaswani
10	9.5.83	The Family Physician and the Aged and the Dying.	Dr Alfred W T Loh

Students attachment to GP Clinics started from April 25, 1983. The following 33 general practitioners are the Clinical Teachers in Family Medicine for 1983:

1 Dr Paul Chan Swee Mong
2 Dr James Chang Ming Yu
3 Dr Gabriel Chiong Peck Koon
4 Dr Chong Tong Mun
5 Dr Chua Pong Kuan
6 Dr Chua Sui Leng
7 Dr Victor L Fernandez
8 Dr Ho Gien Chiew
9 Dr Goh Kiat Seng
10 Dr Goh King Hua
11 Dr Freddie Kee
12 Dr Patrick Kee Chin Wah
13 Dr Koh Eng Kheng
14 Dr Koh Kim Chan
15 Dr Kong Kum Leng
16 Dr Lee Suan Yew
17 Dr Leong Chee Lum
18 Dr Lim Chan Yong
19 Dr Lim Chong Sing
20 Dr Lim Chun Choon
21 Dr Lim Kim Leong
22 Dr Lim Lean Huat
23 Dr Alfred Loh Wee Tiong
24 Dr Loo Choon Yong
25 Dr Frederick Samuel
26 Dr Soh Cheow Beng
27 Dr Tan Hoi Hwa
28 Dr Reginald Tong Thean Seng
29 Dr Moti H Vaswani
30 Dr Victor Wee Sip Leong
31 Dr Wong Kum Hoong
32 Dr Wong Sin Hee
33 Dr Henry Yeo Peng Hock

New Members

The following have been accepted by Council from January/June 1983 to be members of the College:

Ordinary Membership	–	Dr Y Atputharajah
		Dr Zubeda Khatoon
		Dr Lee Sai Kiang
		Dr Asha Nair
		Dr Omar bin Saleh Talib
		Dr Seah Cheng Kiah
		Dr Wong Sin Hee
Associate Membership	–	Dr Chua Seng Chew
		Dr Lim Swee Hock
		Dr Prabhakaran s/o K Govinda
		Dr Tan Chek Wee
		Dr Yao Wan Hwa
		Dr Yeap Eng Hooi
		Dr Cristina Yelf
Overseas Membership	–	Dr Robert Tan Kin Kiat

We welcome them to the College and hope they will participate fully in all activities of the College.

SIF

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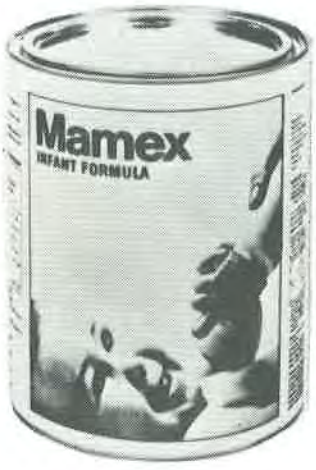
- added lactose, the natural nutrition of the beneficial intestinal lactobacillus, which provokes the acid intestinal reaction characteristic of breast-fed infants
- 14 vitamins and 11 minerals in adequate and balanced concentration
- low renal solute load with the use of whey solids in the form of demineralized whey
- low buffer capacity reducing the requirement of hydrochloric acid in the gastric juice



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Breast feeding is best and all mothers should be encouraged to breast feed their babies. However, when an Infant Formula is required as a supplement or an alternative to breast milk, the product of choice is Mamex Infant Formula.

SIFECS (MOH)/35/80

DUMEX

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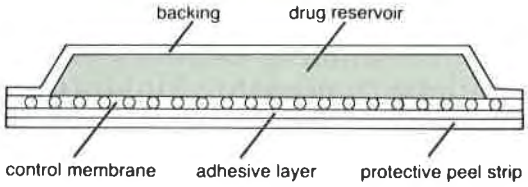

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Important notice

The World Health Organization (WHO) has recommended that pregnant women and new mothers be informed of the benefits and superiority of breastfeeding. Mothers should be given guidance on the preparation for, and maintenance of, lactation, the importance of good maternal nutrition and the difficulty of reversing a decision not to initiate, or to discontinue, breastfeeding. Before using an infant formula, mothers should be advised of the social and financial implications of that decision and the importance for the health of the infant of using the formula correctly. Unnecessary introduction of supplements, including partial bottle feeding, should be avoided because of the potentially negative effect on breastfeeding.*

* WHO - International Code of Marketing of Breast Milk Substitutes, WHA 34.22, May 1981.

SIFECS (MOH)/91/83

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