Medical Quiz: Diagnostic dilemma
Childhood Infections: Streptococcal Sore Throat
Health Education: Ensuring Safe Water
Student’s page: Diagnosis of Rheumatic Fever
TB: Laboratory Diagnosis of TB
Pregnancy: Vomiting in Pregnancy
Rural surgery: Disinfection at district level
Medical Emergency: Subcutaneous Extravasation
Parent/patient education: Rheumatic fever, smoking, lung cancer, x-rays, amniocentesis
Foods we take: Coffee
A leaf from the history of Medicine: Edward Jenner
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Editorial

The first two issues of the IJPD met an expectedly warm response. The doctors practicing in the field as well as medical students, both, have enthusiastically welcomed the issue. This has encouraged all those involved in the publication of the journal.

The journal, as promised, carries articles of direct relevance to the practising doctor. 

**Anaemia** is a common nutritional disorder particularly rampant in the developing world. Classification & typing is generally based on the results of complete blood count and peripheral blood smear. These parameters are easy to do and are within the reach of practitioners even in the remote areas of the country. One article has been put to highlight the role of CBC 7 PBF in diagnosis of anaemia.

Pregnancy is a special occasion, where the mother’s condition directly affects the child. The so-called **TORCH infections** are generally mild in the mother but can prove disastrous to the ofetus & the neonate. Accordingly the fetus may abort or the neonate may get congenital anomalies, rashes and CNS calcifications. **Rh incompatibility** between the mother and her foetus is an important cause of mortality among the neonate, and at the same time is so easy to prevent. One article on this not so infrequent condition has also been included. Als.

**Cold chain maintenance** is an area which is so crucial to the potency of vaccines yet is so easily overlooked. We have discussed this important aspect of immunization in detail.

**Influenza** is a tricky infection: it is one of the oldest scourges of mankind which runs the danger of attaining pandemic proportions every few years. Why despite all efforts to curb its epidemicity influenza virus still continues to baffle mankind has been fully dealt with. Viral hepatitis is a common infection across the world, and new viruses are emerging with passage of time. Some recent information on this hepatotrophic virus has been shared here.

Urinary tract infection in children is a common infection. Infants and younger children require aggressive therapy because they are prone to get pyelonephritis with renal scarring. Traditionally, patients of this tender age have been treated intravenously, but **cefeime** – a 3rd generation oral cephalosporin - has made treatment very convenient and cheaper. The role of cefeime in the treatment of urinary tract infection is discussed by our colleague from Andhra. **Fever in children** is one of the commonest symptoms, and may sometimes try the wits of any doctor. Management of paedriatic fever is dealt in with reference to use of antipyretics.

Foods we use are not only nutritionally important sometimes have benefits which only recently have been explored and documented. Our favourite beverage, **tea**, has been found to contain antioxidats and ingredients which could prevent atherosclerosis, heart disease and even cancers.

In a series of essays on establishing and maintaining surgical facilities in remote areas, we are carrying an article on **surgery room etiquette**. A surgical emergency, reduction of rectal prolapse also has been included.

A special supplement on **drug use in hepatic impairment** has been given as the annexure.

March 2005.

Bashir Gaash
(Executive Editor)
Contributors:

Thangan Menon, MD, is Professor, Department of Microbiology, Dr A.L.M Postgraduate Institute of Basic Medical Sciences, University of Madras, Chennai.

Imtiyaz Ali, MD, is the Professor & Head, Department of Community Medicine, SK Institute of Medical Sciences, Soura.

Ambu Mani, MD, is Assistant Professor, Sri Ramachandran Medical College & Research Institute, Chennai.

Naveen Thakoor, MD, is an otolaryngologist from Jaipur, Rajasthan.

G. Lahijanni, MD, is senior consultant, Adolescent Medicine, University of Adelaide.

Arvind Bhan, MD, is a consultant from Palampura, Himachal Pradesh.

Rehana Kausar, MD, Faculty Member, Regional Institute of Health, Kashmir, is a postgraduate from SK Institute of Medical Sciences, Soura, Srinagar.

Manzoor A Kadri: Before joining the Regional Institute of Health, Kashmir, worked as a teacher at the Government Medical College, Srinagar (Department of Microbiology).

Babina Bassaud, PhD, a Kashmir-born Clinical Psychologist, specializes in childhood psychological disorder and is currently working in Canada.

Niyaz A Jan, a Srinagar-based private practitioner, is a Fellow in Gastrointestinal Endoscopy from the Cleveland Clinic, Ohio, USA.

Rohini Bhan is a trainer at the Regional Institute of Health, Kashmir.

Shabnam Bashir works at the Indraprastha Apollo Hospital, New Delhi.

Bashir Gaash; an International Fellow in Tropical Diseases, and a postgraduate in Social & Preventive Medicine and Health Administration, is currently the Principal, Regional Institute of Health, Kashmir.

Muzaffar Ahmad; MD in General Medicine, has a Fellowship in Emergency Medicine from USA, and currently is the Director Health Services, Kashmir.
At the age of 11 years, a young girl presented to the OPD with joint pain and was not feeling well. She was treated for an episode of tonsillitis 6 weeks ago, which she recovered from completely but has felt tired ever since then. She has a rash on her back and lower limbs that looks like bruises. She finds it very difficult to sleep without using three pillows under her head, as her breathing is much better with these. Her father died from a brain tumour 3 years ago. Both her sister and brother are well. Her mother is a fulltime teacher, and two cats and three goldfish live in the house with them. She does not go to school any more as she is too tired. Upon expiration, a gallop rhythm with early diastolic murmur at the mid-sternal edge can be heard. A soft systolic murmur can also be heard at the apex. An ejection systolic click can be heard at the lower sternal edge. The liver measures 3 cm below the costal margin, and has a smooth surface. The test results are:

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>141 mmol/l</td>
</tr>
<tr>
<td>K</td>
<td>3.9 mmol/l</td>
</tr>
<tr>
<td>Urea</td>
<td>6.2 mmol/l</td>
</tr>
<tr>
<td>Creatinine</td>
<td>55 mmol/l</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.12 mmol/l</td>
</tr>
<tr>
<td>Albumin</td>
<td>36 g/l</td>
</tr>
<tr>
<td>Total protein</td>
<td>70 g/l</td>
</tr>
<tr>
<td>ALT</td>
<td>35 iu/l</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>280 iu/l</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>11.2 g/dl</td>
</tr>
<tr>
<td>TLC</td>
<td>6.9 x 10^9/l</td>
</tr>
<tr>
<td>Platelets</td>
<td>450 x 10^9/l</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>50 s</td>
</tr>
<tr>
<td>Antinuclear antibody)ANA</td>
<td>-ive</td>
</tr>
<tr>
<td>Rheumatoid factor</td>
<td>Negative</td>
</tr>
<tr>
<td>ESR</td>
<td>50 mm/hr</td>
</tr>
<tr>
<td>CRP</td>
<td>25</td>
</tr>
</tbody>
</table>

1) What are the most urgent investigations?
- ECG

2) Which 3 other investigations are appropriate?
- ASO titre
- Throat swabs
- Anti-DNase
- Blood film
- Blood culture
- Creatinine Kinase
- Sweat test
- Thyroid function test

An ECHO was performed which showed that the anterior mitral valve leaflet was thickened and the posterior leaflet was moving paradoxically. There is a mild mitral regurgitation. The aortic valve is bicuspid with AR flow velocity. The AR flow velocity was 3 m/s with a gradient of 36 mm. The peak systolic flow across the valve was 2 m/s.

3) What lesions are associated with the ECHO & the clinical features?
- Mitral valve stenosis
- Aortic stenosis
- Bicuspid aortic valve stenosis
- Mitral valve prolapse
- Mitral regurgitation
- ASD
- VSD
- Pulmonary stenosis
- Tricuspid regurgitation

4) What is the diagnosis?
- Acute rheumatic fever
- Endocarditis
- Congenital heart disease
- Myocarditis
- JCA
- SLE
Streptococcal infection

Bashir Gaash

Streptococci were first demonstrated in patients with erysipelas by Billroth in 1874, and isolated in 1883 by Fehleisen, but it was Rosenbach who, in 1884, applied the designation to the group. The name ‘streptococcus’ is derived from the Greek ‘streptos’ signifying a chain formed of links or a necklace of beads; ‘coccus’ meaning a berry. The organisms may occur in pairs or in chains of varying length. There are two main types: haemolytic & non-haemolytic. The haemolytic ones are further divided on whether they lead to alpha, beta or gamma hemolysis. In beta-haemolysis, a colony of organisms grown on blood agar is surrounded by a clear zone, 2-4 mm in diameter, within which the RBC are completely lysed.

Strep. pyogenes and other streptococci that show a similar phenotype are called β-haemolytic streptococci. They are pathogenic in man. In contrast, species that are typically part of the normal throat flora are either non-haemolytic, or they produce α-haemolysis, characterized by a zone of incomplete, often greenish clearing around colonies grown on blood agar.

The β-hemolytic streptococci grow poorly on ordinary nutrient agar but more readily on blood agar: the zones of beta-hemolysis are fully developed after 18 hours’ growth and very little enlargement occurs thereafter. The colonies do not exceed 1 mm in size. Although the organisms grow both aerobically & anaerobically, β-haemolysis develops more readily anaerobically. Although the growth is possible over a wider range of temperatures, the optimum temperature is 37°C.

Virulence factors & Toxins

a) Extracellular products:
Haemolysins (Streptolysins): Two types of haemolysins, O & S, which are at peak 8 hours after incubation at 37°C, are produced in broth culture. The O-haemolysin is readily oxidized (derives its name from oxygen-lability) while the S-hemolysin is oxygen-stable, (derives its S from being produced by streptococci growing in the presence of serum). The former is produced by all streptococci while as the latter is only produced by group A, C & G.

Streptolysin O & S are the proteins responsible for beta-haemolysis. They lyse RBC & WBC by making holes in their cell membrane. Streptolysins are toxic to the body; streptolysin O is antigenic and leads to production of antibodies (antistreptolysin O) while streptolysin S is either non-antigenic or too weakly antigenic. Antistreptolysin O (ASO) is a true antibody which rises as a result of streptococcal infection. That is why practitioners want to know its titre. It is an exceedingly useful indicator of recent streptococcal infection.

Anti-streptolysin ‘O’ is toxic to a variety of cells and cell fractions including polymorphonuclear
leukocytes, platelets, lysosomes etc. Previously it was thought that a separate haemolysin, leucocidin, destroyed white blood cells. Now we know that it is identical with streptolysin O.

Several extracellular factors serve to facilitate liquefaction of pus and the spreading of streptococci through tissue planes. Four antigenically different DNases (A, B, C & D) participate in the degradation of deoxyribonucleic acid. Previously, deoxyribonuclease used to be called Streptodornase. Streptokinase, another toxic product of GAS, leads to dissolution of clots by catalyzing the conversion of plasminogen into plasmin. Proteinase is another factor liberated by GAS. A spreading factor, hyaluronidase, is also found in streptococcal filtrates. These enzymes degrade the tissue and enhance the spread of infection. The tissue destruction in turn leads to inflammatory response which leads to various symptoms and signs.

**Streptococcal pyrogenic exotoxins (SPEs)** are a family of genetically similar exotoxins that cause severe streptococcal disease unrelated to strep throat. These diseases include scarlet fever, streptococcal toxic shock syndrome, and ‘flesh eating’ necrotizing fasciitis. These exotoxins are superantigens, which can lead to massive activation of T cells. The resulting uncontrolled release of cytokines is responsible for the seriousness of these infections. **Erythrogenic (Dick) toxin** belongs to this group. It is an SPE released from the infection site which enters the bloodstream, circulates throughout the body and leads to a redness of skin and whitish coating of the tongue.

Antibodies to five of extracellular products are used in the serodiagnosis of streptococcal infection. These are ASO, anti-DNase, antihyaluronidase, antinicotinamine adenine dinucleotidase, and antistreptokinase.

**b) Somatic Antigens:**

In addition to elaborating various toxins, Group A streptococci (GAS) possess different somatic protein antigens, which enable GAS to be further divided. This typing is of great value in the study of outbreaks of streptococcal infection. M protein causes the degradation of complement component C3b, an opsonin that would otherwise promote phagocytosis of the bacteria. M protein is essential for the virulence of GAS, because antibody to it prevents infection from occurring. Organisms which donot have M protein are avirulent. More than 90 different kinds of M protein exist and, unfortunately, antibody to one type does not prevent infection by a strain that has another kind of M protein.

Another protein antigen closely related to the M protein is the serum opacity-factor (OF). This is itself antigenic and type-specific; it is useful as an epidemiological marker in typifying GAS when they are not identifiable on the basis of M antigen. It has been found that immune response to M-protein is generally weaker after pharyngeal infection with OF-positive than with OF-negative strains.

Protein F of the cell wall mediates attachments of GAS to the throat by adhering to a protein found on the surface of the epitheloid cells.
Another cell wall constituent, lipoteichoic acid, plays a similar role in the first step in colonization ie adherence of GAS to fibronectin on the surface of human epithelial cells.

Protein G of the bacterium binds to the Fc segment of immunoglobulin G. The effect is to prevent phagocytosis mediated by specific antibody against the bacterium. There are other multifunctional surface proteins which have the ability to bind to host proteins, including immunoglobulin G & A.

C5a peptidase is an enzyme released by GAS, which destroys the C5a component of the complement system, which normally attracts phagocytes to the site of a bacterial infection. This cell-bound peptidase cleaves the C5a component of the complement and inhibits neutrophil chemotaxis.

Both, M antigen and the capsule, which contains mucopolysaccharide hyaluronic acid, are related to the virulence of the strain. M protein is the major somatic virulence factor, while hyaluronic acid capsule is an accessory virulence factor. Both retard phagocytosis of the microorganism by polymorphonuclear leukocytes and strains containing this, being resistant to phagocytosis, multiply rapidly in body and initiate disease.

**Virulent factors of Streptococcus pyogenes (GAS)**

- **C5a peptidase:** Inhibits attraction of phagocytes by destroying C5a
- **Hyaluronic acid capsule:** Inhibits phagocytosis; aids penetration of epithelium
- **M protein:** Interferes with phagocytosis by causing breakdown of C3b opsonin
- **Protein F:** Responsible for attachment to host cells
- **Protein G:** Interferes with phagocytosis by binding Fc segment of IgG
- **SPEs:** Superantigens responsible for scarlet fever, toxic shock, ‘flesh-eating’ fascitis
- **Streptolysin O & S:** Lyse leukocytes and erythrocytes
- **Tissue degrading enzymes:** Enhance spread of bacteria by breaking down DNA, proteins, blood clots, tissue hyaluronic acid.

**Epidemiology:** More than 95% of streptococcal infections in man are caused by group A streptococcus (GAS), which, based on the presence of M antigen, has more than 90 subtypes. Immunity is type-specific; a patient exposed to one subtype develops immunity to that type, but remains susceptible to other subtypes. Thus, he may continue to get repeated sore throat. Other serotypes as C & G are occasionally involved.

The disease occurs primarily in children aged 5-15 years old, with the peak incidence in first few years of the school. However, all age groups are susceptible, and children getting infection in school may infect any family member. There is no sex predilection,
and thus boys and girls are equally susceptible.

Infection is spread from person to person. Nasal carriers are much more infectious than throat carriers. Streptococci are present in large numbers in the saliva of patients for only about a week after the onset of the illness, though the organisms may remain on the throat for many weeks longer. Coughing, yelling, & dribbling by salivary carriers, and sneezing by nasal carriers, lead to dispersal of streptococci in the moist state. Nasal & salivary carriers are thus much more infectious than faucial carriers.

Wet handkerchiefs & wet swabs can transmit infection. When dried, streptococci lose their capability to infect.

More than the profuseness of bacilli in the throat or nose of carriers, it is the age and closeness of contact which determine spread of infection. Overcrowding, as occurs in schools, refugee camps and military or police barracks favours person-to-person spread, and also may enhance the virulence of organisms by natural selection. This is also an underlying cause for increased incidence in colder areas and colder months of the year.

Explosive food- and waterborne infections are well documented. A person preparing food may cough or sneeze on food (especially which is eaten raw) and infect the food. Outbreak consequent to use of salad has also been recorded. Infrequently, a human carrier may infect the milk, and lead to a milk-borne spread. Rarely a human carrier may lead to mastitis in cow, which in turn may lead to infection of milk. Outbreaks of tonsillitis and scarlet fever have been reported from consumption of cream.

Fomites have no significant role to play.

Carriers: GAS frequently colonizes the throats of asymptomatic persons. Pharyngeal carrier rates in normal children vary in different populations, at different seasons and in different conditions and geographical locations. A rate of between 6 & 8% is normally found in populations living in temperate climates; in school-going children these rates have mostly been found to be 15-20%, but may be as high as 50%. In adults, carriage rate is lower. No direct relationship has been observed between carrier rates and outbreaks of illness.

During convalescence GAS rapidly disappear from nose, and the number and virulence of organisms from throat declines. Thus, risk of infection from convalescent carrier is lesser than that from the acutely ill patient.

Clinical features

The usual incubation period is 2-4 days; the organisms multiply in tonsillopharyngeal region, and are present in large numbers in both the nose and throat. There is sudden onset of sore throat with malaise, fever (101°F or higher), and headache. Nausea, vomiting and abdominal pain are common in children. On examination, the posterior part of pharynx is red and oedematous and shows lymphoid hyperplasia. Hyperemic tonsils are studded with grayish-white exudate. Lymph nodes at the angles of mandible are enlarged or tender.
### Streptococcal Serogroups Most Frequently Involved in Human Disease

<table>
<thead>
<tr>
<th>Serogroup</th>
<th>Group specific Cell Wall Antigen</th>
<th>Usual Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Rhamnose-N-acetyl polysaccharide</td>
<td>Pharyngitis, tonsillitis, otitis media, sinusitis, scarlet fever, erysipelas, cellulites, impetigo, pneumonia, endometritis, septicemia. Delayed non-suppurative sequelae: acute rheumatic fever, acute glomerulonephritis,</td>
</tr>
<tr>
<td>B</td>
<td>Rhamnose-glucosamine polysaccharide</td>
<td>Chorioamnionitis, peuperal sepsis, neonatal sepsis, meningitis</td>
</tr>
<tr>
<td>C</td>
<td>Rhamnose-N-acetylgalatosamine polysaccharide</td>
<td>Upper respiratory infections</td>
</tr>
<tr>
<td>D</td>
<td>Glycerol teichnoic acid</td>
<td>Genitourinary tract infections, wound infections, endocarditis</td>
</tr>
<tr>
<td>G</td>
<td>Rhamnose-galactosamine polysaccharide</td>
<td>Upper respiratory infection, cellulites, septicemia, deep-tissue infections.</td>
</tr>
</tbody>
</table>

Total leucocyte count may exceed 12000/cmm with increased numbers of neutrophils. The level of C-reactive protein (CRP) is usually elevated. Throat culture is positive for β-hemolytic streptococci.

All patients don’t exhibit such full-blown syndrome. All don’t show tonsillar or pharyngeal exudates. Those who have undergone tonsillectomy experience a milder syndrome. Infants exhibit less localization to faucial areas, but instead get a generalized infection with rhinorrhea, suppurrative complications, low-grade fever, and a more protracted course. Exudative pharyngitis in children younger than 3 years rarely is streptococcal.

Fever abates within 3-5 days; all acute signs and symptoms subside within a week. It takes several weeks for tonsils and lymph nodes to return their normal size.

In untreated infection, organisms persist for many weeks in the throat. During convalescence the organisms decrease in number and disappear sooner from the nares than the throat. The M-protein content of streptococci, and thus their virulence, gradually decreases. In untreated cases, the organisms have been found die down within 18 days. Penicillin shortens the period of fever, toxicity and infectivity. This is readily appreciated if the drug is started within first 24 hours.

In patients who don’t receive drugs, type-specific antibodies are detectable in serum between 4 and 8 weeks. These antibodies protect the patient from subsequent attack with the same M-typable organism but not from others.
Sore Throat
Differential Diagnosis

Naveen Thakoor

Sore throats are common and affect all age groups but are most common in children & young adults. Most sore throats are part of the spectrum of viral upper respiratory infections but they may be caused by bacterial infection in younger patient. Prolonged pain in the throat in middle-aged or elderly adults is cause for concern, especially if they are heavy smokers or drinkers. In this situation one must consider the presence of a neoplasm. The complaint of sore throat demands a thorough physical examination of the oral cavity, oropharynx, hypopharynx, larynx, thyroid gland, and neck. Pain is sometimes referred to the throat from the oesophagus, stomach or heart.

When the diagnosis is not immediately obvious on physical examination, routine cultures for bacteria (and viruses if available), routine haematological studies, lateral x-rays of the neck, barium studies of the pharynx, oesophagus and stomach, and CT scanning of the neck may be required. If pain in the throat is initiated or accentuated by exertion, then cardiac assessment is needed. Frequent recurrent infections of the pharynx may be an expression of immunodeficiency as seen in diGeorge syndrome or AIDS. It is also seen in immunoglobulin deficiencies.

Causes of sore throat
Tonsillitis
   Bacterial-streptococcal
   Viral-mononucleosis
   Viral pharyngitis
   Rhinovirus
   Coxakie
   Epstein-Barr
   Adenovirus
   Herpes type I & II
   Vincent’s angina
   Fungal pharyngitis
   Candida
   Phycomycetes
   Blastomyces
   Syphilis
   AIDS
   Aphous ulcers
   Pemphigus
   Erythema multiforme
   Eagler’s syndrome
   Glossopharyngeal neuralgia
   Carotidynia
   Cervical spine pain
   Blood dyscrasias
   Thyroiditis
   Reflux Oesophagitis
   Lymphoma
   Leukemia
   Carcinoma
   Tonsil
   Tongue base
   Soft palate
   Supraglottic larynx
   Minor salivary gland tumours

The present thinking about tonsillitis is that there is a resident Epstein-Barr virus in the tonsil which is activated by physical factors. Then there occurs a viral tonsillitis which causes a mild malaise and mild sore throat but its importance lies in the fact that the tonsil no longer secretes immunoglobulin A and is, thus, prone to secondary bacterial infections. This may cause a bacterial tonsillitis, which presents with an elevation of temperature, pain in the throat, trismus, difficulty in eating and speaking, and is often accompanied by cervical lymphadenopathy. There is usually a good response to antibiotics. Tonsillitis may go on to spread outside the capsule of the tonsil, resulting in a peritonsillar abscess or even into the
The parapharyngeal space in the neck causing a parapharyngeal abscess.

There is a physiological increase in size of the tonsil between 4 and 6, and subsequently the tonsils shrink. In preschool age, tonsillitis is usually accompanied by enlargement of adenoids. This can lead to the sleep apnoea syndrome which can vary from snoring to respiratory obstruction. Large tonsil in young adults, especially if accompanied by malaise, are due to a glandular fever. The types of fever that cause the most marked tonsilar swelling are mononucleosis and especially toxoplasmosis. A peritoneal abscess means the infection has passed through the capsule of the tonsil so that there is oedema between the superior constrictor muscle and the capsule of the tonsil. This pushes the tonsil medially on one side and causes trismus, difficulty in speaking and marked elevation in temperature and malaise.

Viral pharyngitis can be caused by the influenza virus, herpes simplex virus, adenovirus, rhinovirus, coxsackie virus and Epstein-Barr virus. The patient usually complains of severe pain with comparatively mild clinical findings. There will probably be redness and oedema of pharynx, especially along the pillars of the tonsil and the posterior pharyngeal wall. Lymphoid aggregates in the pharyngeal mucosa swell, causing a nodular appearance on the posterior pharyngeal wall.

Vincent's angina, or trench mouth, is a contagious disease of the oral cavity and pharynx which is more frequent in young adults and is caused by Treponema dentium and a fusiform bacterium. There is extensive smelly, painful bleeding ulceration covered in grey, necrotic membrane along the margins of the gum.

In our place, diphtheria must always be considered in the differential diagnosis of sore throat at any age. The patient has sore throat, anorexia, low-grade fever and malaise. There is a pseudomembrane which may be seen in the posterior hypopharynx, and may extend into the larynx or trachea or upwards into the nose. This membrane is made of fibrin, cellular debris and bacteria, and may be white, gray or black, and is firmly adherent, resulting in bleeding if forcibly removed. Soft tissue swelling of the neck results in the so-called bullneck. In vaccinated persons, the membrane may be follicular or non-conflent.

Fungal pharyngitis due to candida infection is not uncommon in debilitated adults or in diabetic or immuno-suppressed patients. Less common is infection with the phycomycetes and blastomyces fungi and it is usually confined to selected geographic areas.

In candidiasis (thrush), curdlike material can be seen on tonsils or pharynx, which is easily scrapped. In diphtheria, the membrane usually begins on the tonsils or posterior pharynx, but may spread downwards (larynx/trachea) or upwards (nasopharynx). This membrane is firmly adherent to the underlying tissue, and removal leaves a raw bleeding surface. Diphtheria victims may be toxic & seriously ill, but subclinical cases and carriers are common.

In Kashmir, diphtheria outbreaks occurred twice in district Baramulla.
First outbreak started in Sheri, near Uri, where the patients contracted the infection while attending a marriage ceremony. Some invitees from Srinagar also had contracted diphtheria. The 2nd outbreak occurred in 2003 in two villages of Bandipora, and had started from nomads returning from pastures. All the patients were past 10 years of age. (Ed)

Aphthous lesions are painful superficial ulcers occurring on the mucosa of the oral cavity. They occur episodically and may be recurrent. They are often associated with regional ileitis and the patient must be investigated for this coexisting condition.

Eagle’s syndrome, stylagia, is a controversial symptom. It consists of pain in the tonsil or fossa which is usually unilateral and is presumed due to an elongated styloid process. It is difficult to distinguish it from glossopharyngeal neuralgia.

Acute or subacute thyroiditis can cause pain in the neck or throat. In the acute phase of the disorder, the patient has no difficulty in localizing the problem to the neck. Patients with subacute thyroiditis, however, frequently complain of a persistent soreness in the throat. Discomfort is constant and is aggravated by swallowing and is associated with the sensation of a lump in the throat. Patients are intolerant of constriction of the neck by shirt collars etc.

Reflux oesophagitis usually causes vague symptoms of soreness in the throat or a sensation of a lump in the throat. The patients may have chronic hoarseness or a constant feeling of wanting to clear his throat. The pain may be aggravated after meals or at night-time when the patient is recumbent.

Lymphoma rarely causes pain in the throat and usually presents as enlargement of one tonsil. This may or may not be accompanied by enlargement of the cervical lymph nodes. Carcinoma in the area of the base of tongue, tonsil, soft palate, or upper part of the larynx produces a deep pain in the throat which is quite often difficult to diagnose because it remains hidden and is usually of an ulcerative infiltrative variety. Most minor salivary gland tumours in this area are malignant, the adenoid cystic variety being the commonest.
Bio-typing of Group A Streptococci isolated from Normal School children in South India

Anbu Mani.N* & Thangam Menon**

Abstract: Throat swabs were collected from 250 children aged 5 – 15 years studying at Government High Schools, Nanganallur, Chennai. Group A Streptococci were isolated from 13/250 (5.2%) children. The isolates were biotyped using sugar fermentation tests (Mannitol, Cyclodextrin, Glycogen, Pullulan, Methyl β – D - glucopyranoside) and enzymatic hydrolysis of 4-methylumbelliferyl – β – D – glucuronide. Biotype 4 was the most commonly observed biotype.

Key Words: Group A Streptococci, Biotype.

Group A streptococci (GAS) are the important human pathogens causing acute pyogenic infections such as pharyngitis and pyoderma. They are also responsible for the non-suppurative sequelae such as rheumatic fever (RF), acute glomerulo nephritis (AGN) and rheumatic heart disease (RHD).

Infections (and sequelae) due to group A Streptococcus continue to be an important problem in India and other developing countries, therefore continued surveillance is imperative to monitor epidemiological trends. The purpose of the study was to determine the isolation rate and biotypes of group A Streptococci from throat cultures of normal school children in South India.

Materials and Methods:

Throat swabs were collected from 250 children aged 5 – 15 years studying at Government High Schools of Nanganallur, Chennai. Swabs were transported to the laboratory immediately and were inoculated on to tryptose blood agar plates. Plates were incubated overnight at 37°C, under 5-7% CO₂. β-hemolytic Streptococci were identified by colony morphology and grouping of strains was done by latex agglutination test using Streptex-Kit (Abbott- Murex). Group A Streptococci were biotyped using sugar fermentation tests and enzymatic hydrolysis of 4-methylumbelliferyl–β–D–glucuronide. Sugar fermentation reactions were performed using serum peptone water containing 1% sugars (Mannitol, Cyclodextrin, Glycogen, Pullulan, Methyl β–D-glucopyranoside). Glucuronidase activity was detected by impregnating Whatman filter paper (No.1) disc with the substrate (4-methylumbelliferyl–β–D-glucuronide) and incubating with the bacterial culture for 30 minutes at 37°C, after which the disc was examined for fluorescence under UV light.
Result and Discussion

Out of the 250 throat swabs collected from normal school-going children, 22 (8.8%) β-hemolytic Streptococci were isolated - 13 (59.09%) belonging to group A, 5 (22.72%) to group G, 3 (13.63%) to group C and 1/22 (4.54%) group B.

Table 1: β-hemolytic Streptococci isolated from normal school children

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Organism isolated</th>
<th>No. of Isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Group A Streptococci</td>
<td>13</td>
</tr>
<tr>
<td>2.</td>
<td>Group B Streptococci</td>
<td>1</td>
</tr>
<tr>
<td>3.</td>
<td>Group C Streptococci</td>
<td>3</td>
</tr>
<tr>
<td>4.</td>
<td>Group G Streptococci</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>22</td>
</tr>
</tbody>
</table>

The overall carriage rate of GAS was thus 5.2% (13/250). Biotyping of the 13 strains revealed that 4 belonged to biotype 4, 2 each to biotypes 2 and 8, 1 strain each to biotypes 3 and 9, while 3 strains were untypable (Table). Biotype 4 was the most commonly observed biotype.

Conclusion: Biotyping is a simple method of typing Group A Streptococci and may be a useful alternative in laboratories which are unable to do molecular typing.

References:


Diagnosis of Rheumatic Fever

S.M. Kadri

Rheumatic fever causes chronic progressive damage to the heart and its valves. Until 1960, it was a leading cause of death in children and a common cause of structural heart disease. The disease has been known for many centuries. Baillou (1538-1616) first distinguished acute rheumatic arthritis from gout. Sydenham (1624-1668) described chorea but did not associate it with acute rheumatic fever (ARF). In 1812, Charles Wells associated rheumatism with carditis and provided the first description of the subcutaneous nodules. In 1836, Jean-Baptiste Bouillaud and, in 1889, Walter Cheadle published classic works on the subject.

The association between sore throat and rheumatic fever was not made until 1880. The connection with scarlet fever was established in the early 1900s. In 1944, the Jones criteria were formulated to assist disease identification. These criteria, with some modification, remain in use even today. The introduction of antibiotics in the late 1940s allowed for the development of treatment and preventive strategies. The dramatic decline in the incidence of rheumatic fever is thought to be largely owing to antibiotic treatment of streptococcal infection.

- It must be remembered that diagnosis of ARF requires a high index of suspicion.

- Guidelines of diagnosis used by the American Heart Association include major and minor criteria (ie, modified Jones criteria). In addition to evidence of a previous streptococcal infection, the diagnosis requires 2 major Jones criteria or 1 major plus 2 minor Jones criteria.

- Guidelines of diagnosis used by the American Heart Association include major and minor criteria (ie, modified Jones criteria). In addition to evidence of a previous streptococcal infection, the diagnosis requires 2 major Jones criteria or 1 major plus 2 minor Jones criteria.

- Major criteria

  - Carditis: This occurs in as many as 40% of patients and may include cardiomegaly, new murmur, congestive heart failure, and pericarditis, with or without a rub and valvular disease.

  - Migratory polyarthritis: This condition occurs in 75% of cases and is polyarticular, fleeting, and involves the large joints.

  - Subcutaneous nodules (ie, Aschoff bodies): These nodules occur in 10% of patients and are edematous,
fragmented collagen fibers. They are firm, painless nodules on the extensor surfaces of the wrists, elbows, and knees.

- **Erythema marginatum:** This condition occurs in about 5% of cases. The rash is serpiginous and long lasting.

- **Chorea** (also known as Sydenham chorea and "St Vitus dance"): This characteristic movement disorder occurs in 5-10% of cases. Sydenham chorea consists of rapid, purposeless movements of the face and upper extremities. Onset may be delayed for several months and may cease when the patient is asleep.

**Minor criteria**

- Clinical findings include *arthralgia*, *fever* and previous history of ARF

- Laboratory findings include elevated acute phase reactants (eg, *erythrocyte sedimentation rate*, *C reactive protein*), a *prolonged PR interval*, and supporting evidence of *antecedent group A streptococcal infections* (ie, positive throat culture or rapid streptococcal screen and an elevated or rising streptococcal antibody titre).

**Lab Studies:**

- No specific confirmatory laboratory tests exist. However, several laboratory findings indicate continuing rheumatic inflammation. Some are part of the Jones minor criteria.

- Streptococcal antibody tests disclose *preceding* streptococcal infection.

- Isolation of group A streptococci via throat *culture*.

- Acute phase reactants (eg, erythrocyte sedimentation rate [ESR], C-reactive protein [CRP] in serum, and leukocytosis) may show an increase in serum complement, mucoproteins, alpha-2, and gamma globulins. Anemia usually is caused by suppression of erythropoiesis.

- ECG- PR interval prolongation is present in approximately 25% of all cases and is neither specific to nor diagnostic of ARF.

- Troponins have not been shown to be helpful in making the diagnosis since ischemia and necrosis are not the major cardiac problems.

**Imaging Studies:**

- Echocardiography may be helpful in establishing carditis.

- Synovial fluid analysis may demonstrate an elevated white blood cell count with no crystals or organisms.
References


Parent Education: Rheumatic Fever

Rheumatic Fever, a common acute inflammatory disease, is characterized by fever and pain, tenderness, redness, and swelling of the joints. Rheumatic fever can cause inflammation of the heart and damage to the heart valves. First attacks usually occur from the age of 5 to 15; recurrent attacks can occur throughout adult life. The mortality from the acute attack is low, and most cases subside spontaneously. Often, however, inflammation of the heart leads to scarring and deformity, causing the valves to malfunction. This strain on the heart muscle causes rheumatic heart disease, which can cause death in middle or later life.

Acute rheumatic fever is a complication of streptococcal infection, such as strep throat, scarlet fever, or erysipelas. It sometimes develops after infections so mild as to pass unnoticed. Rheumatic fever begins either insidiously or abruptly after a latent period of two to six weeks following the streptococcal infection. Aside from fever, malaise, and migratory arthritis, patients may develop nodules under the skin, skin rashes, abdominal pain, pleurisy, and chorea. The most serious aspect of the disease, however, is the involvement of the heart.

Treatment involves the use of penicillin to eradicate streptococci that may still be present, bed rest, and administration of salicylates or corticosteroids. It may take many weeks or months before the attack runs its course. Rheumatic fever has become relatively rare, probably due at least in part to the widespread use of antibiotics.
Surgery in the field
(From ‘Surgical Care at the District Hospital, World Health Organization, Geneva 2003)

Disinfection

Disinfectant solutions are used to inactivate any infectious agents that may be present in blood or other body fluids. They must always be available for cleaning working surfaces, equipment that can not be autoclaved and non-disposable items and for dealing with any spillages involving pathological specimens or other infectious material.

Needles and instruments should routinely be soaked in a chemical disinfectant for 30 minutes before cleaning. Disinfection decreases the viral and bacterial burden of an instrument, but does not clean debris from the instrument or confer sterility. The purpose of disinfection is to reduce the risk to those who have to handle the instruments during further cleaning.

Reusable needles must always be used with great care. After use, they should be placed in a special container of disinfectant before being cleaned and sterilized. Thick gloves should be worn when needles and sharp instruments are being cleaned.

There are many disinfectant solutions, with varying degree of effectiveness. In most countries, the most widely available disinfectant is sodium hypochlorite solution (bleach solution) which is particularly effective antiviral disinfectant solution.

To ensure effective disinfection, follow the manufacturer’s instructions or any other specific guidelines that have been given and dilute the concentrated solution to the correct working strength. It is important to use all disinfectant solutions within their expiry date as some solutions, such as hypochlorite, lose their activity very quickly.

- Cleaning removes debris
- Disinfection decreases the viral and bacterial burden of an instrument, but does not clean debris or confer sterility.
- Sterilization kills microbes

Disinfection must be performed before cleansing with detergent. There are many different disinfectants available and these act in different ways, so it is important to use the appropriate one in order to ensure effective disinfection. All disinfectants have what is known as contact time, which means that they must be left in contact with an infectious agent for a certain period of time to ensure that it is completely inactivated. However, some disinfectants are themselves inactivated by the presence of organic material and so higher concentrations of disinfectant and longer contact times must be used in certain situations, such as a large spilt of infected blood.

After disinfection, you clean with normal detergent and water to remove the inactivated material and the used disinfectant. Even if disinfection is performed correctly, all the waste material generated should be disposed off safely.

Take great care when using any disinfectants containing chlorine. In the presence of some chemicals, it is very easy to liberate poisonous chlorine gas from some chlorine-containing solutions.
(when bleach and acid are mixed, for example). If you have any doubts about the exact composition of a spilt mixture containing infectious agents, you can neutralize any acid present by adding a small amount of saturated sodium bicarbonate before adding bleach or hypochlorite solution.

Linen soiled with blood should be handled with gloves and should be collected and transported in leak-proof bags. Wash the linen first in cool water and then disinfect with a dilute chlorine solution. Then wash it with detergent for 25 minutes at a temperature of at least 71°C.

Before sterilization, all equipment must be disinfected and then cleaned to remove debris. Sterilization is intended to kill living organisms, but is not a method of cleaning.

Sterilization

The methods of sterilization in common use are:

- Autoclaving or steam sterilization
- Exposure to dry heat
- Treatment with chemical antiseptics

Autoclaving should be the main form of sterilization at the district hospital.

Autoclaving

Before sterilizing medical items, they must be disinfected and vigorously cleaned to remove all organic material. Proper disinfection decreases the risk for the person who will be cleaning the instruments. Sterilization of all surgical instruments and supplies is crucial in preventing HIV transmission. All viruses, including HIV, are inactivated by steam sterilization (autoclaving) for 20 minutes at 121°C-132°C or for 30 minutes if the instruments are in wrapped packs.

For efficient use, an autoclave requires a trained operator and depends on regular maintenance. The selection of a suitable autoclave requires serious consideration not only for its cost but also:

- Anticipated use
- Workload
- Size
- Complexity
- Power source

In general, the smaller the capacity, the shorter the whole process and the less damage to soft materials. It is often more practical to use a small autoclave several times a day than to use a large machine once.

Appropriate indicators must be used each time to show that sterilization has been accomplished. At the end of the procedure, the outsides of the packs of instruments should not have wet spots, which may indicate that sterilization has not occurred.

Dry heat

If items cannot be autoclaved, they can be sterilized by dry heat for 1-2 hours at 170°C. Instruments must be clean and free of grease or oil. However, sterilizing by hot air is a poor alternative to autoclaving since it is suitable only for metal instruments and a few natural suture materials.
Boiling instruments is now regarded as an unreliable means of sterilization and is not recommended as a routine in hospital practice.

**Antiseptics**

In general, instruments are no longer stored in liquid antiseptic. However, sharp instruments, other delicate equipment and certain catheters and tubes can be sterilized by exposure to formaldehyde, glutaraldehyde or chlorhexidine. If you are using formaldehyde, carefully clean the equipment and then expose it to vapour from paraformaldehyde tablets in a closed container for 48 hours. Ensure this process is carried out correctly.

Glutaraldehyde is a disinfectant that is effective against bacteria, fungi and a wide range of viruses. Always follow the manufacturer’s instructions for use.

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**Failure of normal methods of sterilization**

Failure of an autoclave or a power supply may suddenly interrupt normal sterilization procedures. If an extra set of sterile equipment and drapes are not available, the following ‘antiseptic technique’ will allow some surgery to continue.

1. Immerse towels and drapes for 1 hour in a reliable antiseptic such as chlorhexidine, wring them out and lay them moist on the skin of the patient.
2. Treat gauze packs and swabs similarly, but rinse them in diluted (1:1000) chlorhexidine solution before using them in the wound. From time to time during the operation, rinse gauze in use in this solution.
3. Immerse instruments, needles, and natural suture materials in strong antiseptic for 1 hour and rinse them in weak antiseptic just before use.
Health Education

Smoking is the act of inhaling the fumes from a burning substance, usually tobacco. The adverse effects of tobacco smoking totally outnumber those of other atmospheric pollutants. In the European Union, disease attributable to smoking accounts for approximately 15 per cent of all deaths. Tobacco smoking has consistently been referred to “as the single most important preventable cause of premature death”.

In most countries, the percentage of people who smoke is on the decline, but in recent years the number of young women who smoke, particularly in Western Europe, has been on the increase. There has also been an increase in children who smoke in the United Kingdom.

I  SMOKING AND DISEASE

Smoking can be divided into two categories: active (actively smoking oneself) and passive (inhaling smoke because of proximity to a smoker). Cigarette smoking is the prime, but not the only, culprit; pipe and cigar smoking, while less hazardous than cigarette smoking, are not without risk. Smokeless tobacco (chewing tobacco, tobacco pouches, and snuff dipping) has now emerged as a major cause of oral disease and death from oral cancer.

The average 20-a-day smoker is estimated to inhale tobacco smoke about 70,000 times a year. It is therefore not surprising that, with such abuse, a number of diseases, many of them fatal, are associated with smoking. These include cancer (particularly of the lungs, larynx, oral cavity, pharynx, oesophagus, pancreas, cervix, kidney, and bladder); coronary artery disease; cerebrovascular disease (stroke, intracerebral haemorrhages); and COAD (chronic obstructive airways disease, comprising chronic bronchitis and emphysema).

COMPONENTS OF TOBACCO SMOKE

The composition of tobacco smoke when inhaled has been the subject of much investigation. Even the presence of filter tips does not fully protect the smoker from the hazardous effects of the poisonous agents. Smoke contains over 4,000 chemicals; 43 of them are carcinogenic (cancer promoting). These include chemicals such as polycyclic hydrocarbons, beta naphthylamines, and also nitrosamines, which have been long recognized as carcinogenic in lower animals. It also contains cellular irritants such as ammonia, formaldehyde, and oxides of nitrogen. Carbon monoxide, which avidly binds to haemoglobin and reduces its oxygen-carrying capacity, is also present.

The major component, however, is nicotine, which has a variety of effects on the sympathetic nervous system in humans. It is highly addictive—comparable to heroin and cocaine—and produces an increased heart rate; raised
blood pressure; and increased discharge of sympathetic nerves in the autonomic nervous system.

III  CARDIOVASCULAR DISEASE

Coronary heart disease, particularly myocardial infarction (heart attack), is the primary cause of death related to cigarette smoking. This is caused by atherosclerosis (deposition of lipids, a type of fat, and fibrous tissue in the walls of arteries), one of the major risk factors for which is smoking. Although, cardiovascular deaths are declining in a number of countries, including the United Kingdom and the United States, they are dramatically increasing in the developing world.

IV  LUNG CANCER

This is closely followed by lung cancer, which is directly causally related to smoking. The disease carries a greatly elevated relative risk for development when comparing smokers and non-smokers. The risk of mortality is dose-related: the more pack-years (the number of packs smoked per day multiplied by the number of years of smoking), the higher is the risk of the disease developing.

Smoker's Lung Tissue
The lungs are made up of approximately 350 million tiny sacs called alveoli, where carbon dioxide from the body is exchanged for oxygen from the air. Various diseases that affect the lungs either destroy the alveoli directly, as does emphysema, or impair the alveoli’s ability to exchange gases. This picture shows the effects of emphysema (caused by smoking) on lung tissue.
V PERIPHERAL VASCULAR DISEASE

Peripheral vascular disease, which affects the feet and lower limbs, is more prevalent in smokers. Like coronary heart disease, it is caused by the development of atherosclerosis in the blood vessels of a limb. In severe cases, this may lead to amputation of a foot or digit because of gangrene (death of tissues). The risk of developing peripheral vascular disease is again dose-related, and may decrease once the patient stops smoking.

For routine operations, smokers are at a higher risk of developing post-operative complications, including deep venous thrombosis, which can lead to pulmonary embolism and sometimes death.

VI EFFECTS OF PASSIVE SMOKING

The effects of tobacco smoke on the unborn child are now well-catalogued. Many studies have shown an association between cigarette smoking and the increased incidence of babies having low birthweight, spontaneous abortions, stillbirths, and sudden infant death syndrome (SIDS). Furthermore, certain complications of pregnancy, some of which may be life-threatening (such as raised blood pressure), are also associated with smoking.

Involuntary smoking exposure (passive smoking) is detrimental to the health of adults and increasingly, children, who, according to recent studies, run a higher risk of developing lung problems. From the data to date, it appears that passive smoking carries a 1.5 relative risk of developing cancer of the lung as compared to non-smokers. There are also increased risks of heart attack and cerebrovascular diseases.

VII EFFECTS OF GIVING UP

Cessation of smoking is slowly followed by a reduction in excess mortality. After ten years the risk of premature death is more than halfway towards that of someone who has never smoked. The increased risk of lung and laryngeal cancer begins to decline after one to two years. There is a prompt reduction in the risk of developing a heart attack after one year of stopping smoking. It is also believed that the risk of developing COAD is reduced.

"Child abuse does not have to mean broken bones and black and blue marks. Young growing tissues are far more vulnerable to carcinogens than those of adults. Knowingly subjecting children to respiratory tract disease is child abuse."

William Cahan, Memorial Sloan Kettering Cancer Centre, USA, 1993)
The burning of tobacco produces a cocktail of dangerous chemicals. Almost half the world’s children (about 700 million) are exposed to smoke from burning tobacco and exhaled at home. Environmental tobacco smoke has particularly harmful effects on fetuses and young children, causing respiratory infections and other illnesses.

Children do not choose to inhale a mix of over 4000 chemicals, including carcinogens. In fact, the majority of children worldwide urge people to stop smoking in public spaces.

Table: Percentage of students aged 13-15 years who want ban on smoking in public places (1999-2003)

<table>
<thead>
<tr>
<th>Region</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Libya, Brazil</td>
<td>&gt; 75%</td>
</tr>
<tr>
<td>India, China</td>
<td>51-75%</td>
</tr>
<tr>
<td>Zambia, Zimbabwe</td>
<td>26%-50%</td>
</tr>
</tbody>
</table>

At home, it is the responsibility of parents to protect their children and stop smoking. Media campaigns, combined with smoking restrictions in public places and the workplace, can help make homes tobacco free. Other tobacco control measures include taxation, ban on tobacco advertising and health warnings on cigarette packs. The Framework Convention on Tobacco Control, an international treaty instigated by WHO, is currently in the process of signature and ratification.

Children whose parents and friends smoke are more likely to become addicted themselves; 250 million children alive today will be killed by tobacco if current consumption trends continue.

### Health Effects of Passive Smoking on Children
- Increased risk of sudden infant death syndrome
- Brain: possible association with brain tumours and longterm mental defects
- Ears: Middle ear infections (Acute Otitis Media)
- Lungs: Respiratory disease (including bronchitis & pneumonia)
- Asthma induction and exacerbation
- Chronic respiratory symptoms (Wheezing, coughing, breathlessness)
- Decreased lung function
- Heart: Adverse effects on oxygen uptake and arteries
- Blood: Possible association with lymphoma.
- Burns: From fires caused by tobacco
Lung cancer is a common cancer in developed countries; in the United states, it is the 2nd most common cancer in men and women. In 2001, there were some 170,000 new cases of lung cancer diagnosed in that country. Approximately, 80% of all are non-small cell lung cancers (NSCLC), adenocarcinoma forming 40% of this group. Squamous cell carcinoma comprises of 30-35% and large cell carcinoma 5-15%. Incidence and deaths are decreasing in men, but increasing in women. In Scotland and northern England it has already overtaken breast cancer as the primary cause of cancer death in women. Lung cancer is most common in individuals over 50 years of age. The highest incidence is in the 65 to 79 age-group.

Adenocarconimooa is the most common form of lung cancer in non-smoking persons, is often located peripherally to bronchi and tends to metastasize to the brain, adrenals, sand bone. Squamous cell carcinoma usually occurs centrally near the main stem bronchi and is usually associated with smoking. Large cell carcinoma is often located peripherally to bronchi.

Small cell carcinomas start in the hormonal cells in lungs (in 95% cases in the central part of the chest) and proliferate rapidly. Its subtypes include oat-cell or lymphocytic, intermediate and combined varieties. Vast majority of patients (97%) have history of smoking.

Although the 2nd most common cancer in incidence, lung cancer is the most common cause of cancer death in the United States. It accounted for 31% deaths in males and 25% in females during 2001. In the United Kingdom, there were about 34,960 deaths in 1998. There lung cancer is the most common cancer in men, with about 25,690 cases in 1996 and 21,850 deaths in 1998. In British women, it is the third most common cancer, with about 15,000 cases in 1996, and ranks second in cancer deaths, with about 13,110 reported in 1998.

<table>
<thead>
<tr>
<th>Lung Cancer Incidence &amp; Deaths in USA (2001)</th>
</tr>
</thead>
<tbody>
<tr>
<td>New cases diagnosed</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Men</td>
</tr>
<tr>
<td>Women</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

Survival rate is poor even countries like United States. Overall, the five-year survival rate is 15.8%. This low rate is attributable to the fact that the majority of new cases are diagnosed at a regional or distant stage, for which survival is very poor. If the disease is diagnosed while still localized, approximately 50% of patients survive 5 years. Quite unfortunately, even in such a resource-rich country, only 16% of new diagnoses are classified as localized, and fewer than 25% are asymptomatic at the time of diagnosis.

Risk factors: Lung cancer is one of the few deadly cancers where risk factors are identifiable, specific and modifiable.

A) Modifiable Risk Factors
1) Smoking is the major cause of lung cancer today; epidemiological studies show that, at least, 78% of lung cancers are related to smoking. Incidence is directly related to the quantity and duration of smoking. Evidence suggests that lung cancer has a multistep carcinogenesis, and genetic damage occurs with tobacco use.
2) Passive (2nd hand) smoke is also an important cause of lung cancer.
3) **Air pollutants**: Both indoor and outdoor air pollutants are suspected carcinogens. Radon is especially dangerous to underground miners. House hold combustion devices are important indoor carcinogens. 

4) **Building materials**, especially asbestos, have been incriminated.

5) **Occupational hazards**: Coal gasification, coke production, exposure to soot, and aluminium production are involved. Exposure to nickel, arsenic, uranium and chromium are other hazards.

6) **Tuberculosis** may predispose to lung cancer, especially developing in the scar tissue. The risk of lung cancer can be 5 times higher in men and 10 times in women who have contracted tuberculosis.

7) Past reports suggest that patients with COPD can have a 4-6 fold higher risk of lung cancer independent of their smoking history. This could be a confounding finding.

7) Certain **foods** are protective: carotenoids, vitamin C & E, and selenium can scavenge the free radicals produced by tobacco smoke, solvents, and pollutants.

B) **Non-modifiable risk factors**: 

1) **Genetic factors** play a definitive part in cancer. **Proto-oncogenes** are encoded proteins which influence cellular proliferation and differentiation, such as growth factors, growth factor receptors, signal transducing proteins, and nuclear regulatory proteins. Damage to these genes can lead to point mutations, chromosomal translocations, and gene amplification, which in turn results in cellular proliferation and thence to tumorigenesis. It has been estimated that at least 10-20 genetic mutations have occurred by the time a lung cancer is clinically evident.

**Tumour suppressor genes** exert a negative effect on regulation, and thus they regulate normal cellular growth. Inactivation of these genes, which may be from their inability to receive signals or inability to accurately process the signals received, leads to loss of function and this contributes to malignancy. Such tumour suppressor genes involved in the genesis of lung cancer include TSG 101 and DMBT1. The former permits proteolysis and cell-cycle progression, and is involved in 90% of SCLC, while the lack of the expression of the latter tumour suppressor gene has been found in 40% of NSCLC & 10% of SCLC. Various other tumour suppressor genes are also involved.

**Screening in lung cancer**

In spite of the fact that lung cancer is the number one killer among males & females in the developed world, no guidelines for screening have been laid anywhere. This is because it has not been proved that screening with x-ray or sputum cytology saves lives. Therefore, there are no official recommendations for screening for lung cancer, even in high risk populations.

**Chest x-ray**: Chest xray is one of the most valuable tools in the diagnosis of lung cancer. The sensitivity of the screening test, however, for detection of lung cancer depends on various underlying circumstances including the size and location of the lesion, quality assurance factors related to image quality, and the skill of the interpreting physician. Moreover, the mediastinum, ribs, and other aspects of chest structure may obstruct the lesion, so that a big lesion also is missed. Then, as in any other test, it is the interpretor’s error which may play a part.

Generally speaking, chest xrays are not sensitive at detecting lesions smaller than 2 cm.

**Sputum cytology**: Sputum cytology was once believed to have a potential for the
early detection of lung cancer, but showed little added advantage over chest-xray in the National Cancer Institute Trials. It is the least invasive means of obtaining a specific diagnosis in a patient who is suspected of having lung cancer, but can suffer because of a need for rigorous specimen collection and preservation technique. Average overall sensitivity is 64%, but drops to 40% in peripheral lung lesions.

**Low-radiation CAT:** It is the most promising new tool for early lung cancer detection. It has a potential for detecting 75-80% of lung cancers in Stage I. The newer low-radiation CAT scans whole chest in single breath-hold within 15 seconds. It is much more sensitive than plain chest xray in detecting small pulmonary nodules, mediastinal adenopathy, small pleural effusions, and the ability to detect abnormalities below the diaphragm. Lesions.

Currently, an Early Lung Cancer Project is on to determine if spiral Ct can be used as screening tool to reduce the lung cancer mortality by early detection and prompt management. Currently, what is needed is that practitioners aggressively pursue all cases presenting with dyspnoea, cough, haemoptysis, chest discomfort, hoaraseness and other related symptoms.

**Further Reading:**

Lung Cancer is the growth of malignant cells affecting, initially, the lung. Cancer forms when a lung cell undergoes alterations to its DNA (Nucleic Acids), leading to uncontrolled cell growth. Proliferation continues until a tumour forms.

The process of metastasis (distant spread) takes place when cells break off from the tumour and travel via the blood or lymphatic system, lodging in other organs. There they begin to multiply, forming other tumours.

Lung cancer falls into two groups: small cell and non-small cell carcinomas. Small cell lung cancer (SCLC) is also called oat cell carcinoma because of the cells’ shape. About 25 per cent of lung cancers are of this type.

Non-small cell lung cancer (non-SCLC) comprises squamous cell (or epidermoid) cancer, which arises from cells that line the airways of the lung, and is the most common form of lung cancer; Adenocarcinoma, which arises in mucus-producing cells which line the upper airways of the lung; and large cell carcinoma.

**SYMPTOMS**

The symptoms of lung cancer are a persistent hacking cough, blood in the sputum, wheezy breathing, pain in the shoulder or chest, neck or facial swelling, and recurring pneumonia or bronchitis.

<table>
<thead>
<tr>
<th><strong>Symptoms of lung cancer:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent hacking cough,</td>
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<tr>
<td>Blood in the sputum,</td>
</tr>
<tr>
<td>Wheezy breathing,</td>
</tr>
<tr>
<td>Pain in the shoulder or chest,</td>
</tr>
<tr>
<td>Neck or facial swelling, and</td>
</tr>
<tr>
<td>Recurring pneumonia or bronchitis.</td>
</tr>
</tbody>
</table>

**II CAUSES**

A huge proportion—90%—of lung cancers is caused by smoking. About 6 per cent may be linked to a naturally occurring radioactive gas, radon. Other causes are passive smoking (breathing in smoke from other people’s cigarettes, cigars, or pipes) and exposure to certain substances such as asbestos. Diet may also be involved; studies have shown that a diet low in fresh fruit and vegetables may increase risk.

**III SMOKING LINK**

The conclusive link between smoking and lung cancer was proven in 1950 at the end of an extensive study conducted by Richard Doll and Austin Bradford Hill. In the 1940s, there was a huge increase in lung cancers and the researchers showed that it was a direct result of the large increase in smoking that had begun 40 years before.

Cigarette smoke contains about 4,000 chemicals and at least 50 of them are carcinogenic (cancer-causing).
Evidence suggests that tar contains the carcinogens that cause lung cancer, so low-tar cigarettes may carry a lower risk. Risk is directly related to the number of cigarettes a person smokes and the number of years they continue to smoke.

While smoking has been decreasing in Western countries, it has become more widespread in some developing countries. China, for instance, now has an estimated 350 million smokers and is likely to see a large increase in lung cancers in the future; researchers estimate that about 50 million of today’s children in China will eventually die as a result of smoking. About half these deaths are likely to be from cancer.

Overall, 50 per cent of people who smoke are killed by their habit. However, it is now well established that giving up smoking before developing lung cancer or any of the other diseases linked to it (heart, circulatory, or other types of lung disease) eliminates most of the risk of dying from the habit.

IV DIAGNOSIS

Diagnosis of lung cancer is made by a chest X-ray, analysing sputum cells, and examining the bronchi using a bronchoscope, a fibre-optic instrument that is now sufficiently flexible to reach previously inaccessible bronchi.

V TREATMENT

Lung cancer is difficult to control with existing treatments. Less than 10 per cent of patients survive for five years after diagnosis. This is mainly because the cancer has usually spread before it is diagnosed and surgery is therefore not an option. Radiotherapy and chemotherapy can help improve a patient’s quality of life and in some cases extend life.

Small cell lung cancer spreads rapidly and is sensitive to drugs, so chemotherapy can control it for a while. However, the cells may become drug resistant and therefore the periods of remission become shorter.

Surgery may be possible in patients with non-SCLC if the cancer is confined to the original tumour site and has not metastasized. Unfortunately, it is common for this type of cancer to return. Treatment for advanced cancer may include radiotherapy, chemotherapy, or laser therapy.

VII POTENTIAL NEW TREATMENTS

New methods of giving chemotherapy are being devised to find out whether side effects can be lessened and survival of sufferers improved. For instance, one recent trial gave SCLC patients the drug etoposide by continuous infusion (via a drip into a main vein over several days). Initial results were encouraging, with patients reporting that they found the treatment more tolerable than the normal tablet method. Also, administering etoposide in this way eliminates the need for other anti-cancer drugs that can have debilitating side effects.

There are several new agents under clinical trial at present, including
the anti-cancer drugs taxol, taxotere, topotecan, and vinorelbine, and biological therapies including interleukin-2, interferon, and granulocyte colony stimulating factor. Cytokines (proteins vital to the workings of the body’s immune system) are also being used in cancer treatment to enhance the effects of anti-cancer drugs. Trials so far have shown a high response when giving the antiviral protein interferon and cisplatin together. Interferon, which occurs naturally in white blood cells, can be synthesized by genetic engineering. Alpha-interferon is also being used in combination with cisplatin for treating non-SCLC.

There are a number of gene therapies under trial, mainly in the United States. One approach is to reactivate the p53 gene, which suppresses tumours. Mutated p53 genes are involved in a large number of cancers, including lung cancer.

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Many patients wrongly believe that x-ray examination can reveal any underlying ailment. In view of the hazards posed by radiation, doctors should discourage all unnecessary x-rays even if the patients or attendants seek this diagnostic test.

Also, the extra danger posed by old, ill-maintained machines should be borne in mind. (Ed)

Health Education

X-Rays

X-Ray, a penetrating electromagnetic radiation and having a shorter wavelength than light, is produced by bombarding a target, usually made of tungsten, with high-speed electrons. X-rays were discovered accidentally in 1895 by the German physicist Wilhelm Conrad Roentgen while he was studying cathode rays in a high-voltage gaseous-discharge tube. Despite the fact that the tube was encased in a black cardboard box, Roentgen noticed that a barium platinocyanide screen, placed nearby by chance, emitted fluorescent light whenever the tube was in operation. After conducting further experiments, he determined that the fluorescence was caused by invisible radiation of a more penetrating nature than ultraviolet radiation. He named the invisible rays “X-rays” because of their unknown nature. Subsequently, X-rays were designated as Roentgen rays in his honour.

I  NATURE OF X-RAYS

X-rays are electromagnetic radiation ranging in wavelength from about 10 nm to 0.001 nm (1 nm, or nanometre, is $10^{-6}$ mm, or 40 billionths of an inch). The shorter the wavelength of the X-ray, the greater is its energy and its penetrating power. Longer wavelengths, near the ultraviolet-ray band of the electromagnetic spectrum, are known as soft X-rays. The shorter wavelengths, closer to or overlapping the gamma-ray range, are called hard X-
rays. X-rays forming a mixture of many different wavelengths are known as “white” X-rays, as opposed to “monochromatic” X-rays, which represent only a single wavelength. Both light and X-rays are produced by transitions from orbit to orbit of electrons in atoms: light by the transitions of outer electrons and X-rays by the transitions of inner electrons.

X-rays are produced whenever high-velocity electrons strike a material object. Much of the energy of the electrons is lost in heat; the remainder produces X-rays by causing changes in the target's atoms as a result of the impact. The X-rays emitted can have no more energy than the kinetic energy of the electrons that produce them. Moreover, the emitted radiation is not monochromatic but is composed of a wide range of wavelengths with a sharp lower wavelength limit corresponding to the maximum energy of the bombarding electrons. This continuous spectrum is referred to by the German name bremsstrahlung, which means “braking”, or slowing down, radiation, and is independent of the nature of the target. If the emitted X-rays are passed through an X-ray spectrometer, certain distinct lines are found superimposed on the continuous spectrum; these lines, known as the characteristic X-rays, represent wavelengths that depend only on the structure of the target atoms. In other words, a fast-moving electron striking the target can do two things: it can excite X-rays of any energy up to its own, or it can excite X-rays of particular energies, which are dependent on the nature of the target atom.

II X-RAY PRODUCTION

The first X-ray tube was the Crookes tube, a partially evacuated glass bulb containing two electrodes, named after its designer, the British chemist and physicist Sir William Crookes. When an electric current passes through such a tube, the residual gas is ionized and positive ions, striking the cathode, eject electrons from it. These electrons, in the form of a beam of cathode rays, bombard the glass walls of the tube and produce X-rays. Such tubes produce only soft X-rays of low energy.

An early improvement in the X-ray tube was the introduction of a curved cathode to focus the beam of electrons on a heavy-metal target, called the anticathode, or anode. This type generates harder rays of shorter wavelengths and of greater energy than those produced by the original Crookes tube, but the operation of such tubes is erratic because the X-ray production depends on the gas pressure within the tube.

The next great improvement was made in 1913 by the American physicist William David Coolidge. The Coolidge tube is highly evacuated and contains a heated filament and a target. It is essentially a thermionic vacuum tube in which the cathode emits electrons because it is heated by an auxiliary current and not because it is struck by ions as in the earlier types of tubes. The electrons emitted from the heated cathode are accelerated by the application of a high voltage across the tube. As the voltage is increased, the minimum wavelength of the radiation decreases.

Most of the X-ray tubes in present-day use are modified Coolidge
tubes. The larger and more powerful tubes have water-cooled anticathodes to prevent melting under the impact of the electron bombardment. The widely used shockproof tube is a modification of the Coolidge tube with improved insulation of the envelope (by oil) and grounded power cables. Such devices as the betatron are used to produce extremely hard X-rays, of shorter wavelength than the gamma rays emitted by naturally radioactive elements.

III PROPERTIES OF X-RAYS

X-rays affect a photographic emulsion in the same way that light does. Absorption of X radiation by any substance depends upon its density and atomic weight. The lower the atomic weight of the material, the more transparent it is to X-rays of given wavelengths. When the human body is X-rayed, the bones, which are composed of elements of higher atomic weight than the surrounding flesh, absorb the radiation more effectively and therefore cast darker shadows on a photographic plate. Radiation consisting of neutrons is now used in some types of radiography and produces almost opposite results. Objects that cast dark shadows in an X-ray picture are almost always light in a neutron radiograph.

A Fluorescence

X-rays also cause fluorescence in certain materials, such as barium platinocyanide and zinc sulphide. If a screen coated with such fluorescent material is substituted for the photographic films, the structure of opaque objects may be observed directly. This technique is known as fluoroscopy.

B Ionization

Another important characteristic of X-rays is their ionizing power, which depends upon their wavelength. The capacity of monochromatic X-rays to ionize is directly proportional to their energy. This property provides a method for measuring the energy of X-rays. When X-rays are passed through an ionization chamber, an electric current is produced that is proportional to the energy of the incident beam. In addition to ionization chambers, more sensitive devices, such as the Geiger-Müller counter and the scintillation counter, can measure the energy of X-rays on the basis of ionization. In addition, the path of X-rays, by virtue of their capacity to ionize, can be made visible in a cloud or bubble chamber.

IV APPLICATIONS OF X-RAYS

The principal uses of X radiation are in the fields of scientific research, industry, and medicine.

A Research

The study of X-rays played a vital role in theoretical physics, especially in the development of quantum mechanics. As a research tool, X-rays enabled physicists to confirm experimentally the theories of crystallography. By using X-ray diffraction methods, crystalline substances may be
identified and their structure determined. Virtually all present-day knowledge in this field was either discovered or verified by X-ray analysis. X-ray diffraction methods can also be applied to powdered substances that are not crystalline but that display some regularity of molecular structure. By means of such methods, chemical compounds can be identified and the size of ultramicroscopic particles can be established. Chemical elements and their isotopes may be identified by X-ray spectroscopy, which determines the wavelengths of their characteristic line spectra. Several elements were discovered by analysis of X-ray spectra.

A number of recent applications of X-rays in research are assuming increasing importance. Microradiography, for instance, produces fine-grain images that can be enlarged considerably. Two radiographs can be combined in a projector to produce a three-dimensional image called a stereoradiogram. Colour radiography is also used to enhance the detail of X-ray photographs; in this process, differences in the absorption of X-rays by a specimen are shown as different colours. Extremely detailed information is provided by the electron microprobe, which uses a sharply defined beam of electrons to generate X rays in an area of specimen as small as 1 micrometre (about 1/25,000 in) square.

**B Medicine**

X-ray photographs, called radiographs, and fluoroscopy are used extensively in medicine as diagnostic tools. In radiotherapy, X-rays are used to treat certain diseases, notably cancer, by exposing tumours to X radiation.

The value of radiographs for diagnostic purposes is a consequence of the penetrating properties of X-rays. Within a few years of their discovery, X-rays were being used to locate foreign bodies, such as bullets, within the human body. With the development of improved X-ray techniques, minute differences in tissues were revealed by radiographs, and many pathological conditions could be diagnosed by this means. X-rays provided the most important single method of diagnosing tuberculosis when that disease was prevalent. Pictures of the lungs were easy to interpret because the air spaces are more transparent to X-rays than the lung tissues. Various other cavities in the body can be filled artificially with contrasting media, either more transparent or more opaque to X-rays than the surrounding tissue, so that a particular organ is brought more sharply into view. Barium sulphate, which is highly opaque to X-rays, is used for the X-ray examination of the gastrointestinal tract. Certain opaque compounds are administered either by mouth or by injection into the bloodstream in order to examine the kidneys or the gallbladder. Such dyes can have serious side effects, however, and should be used only after careful consultation. The routine use of X-ray diagnosis has in fact been discouraged in recent years as of questionable usefulness.

A recent X-ray device, used without dyes, offers clear views of any part of the anatomy, including soft organ tissues. Called the body scanner, or computerized axial tomography (CAT or CT) scanner, it rotates 180° around a
patient's body, sending out a pencil-thin X-ray beam at 160 different points. Crystals positioned at the opposite points of the beam pick up and record the absorption rates of the varying thicknesses of tissue and bone. These data are then relayed to a computer that turns the information into a picture on a screen. Using the same dosage of radiation as that of the conventional X-ray machine, an entire “slice” of the body is made visible with about 100 times more clarity. The scanner was invented in 1972 by the British electronics engineer Godfrey Hounsfield, and was in general use by 1979.

Answers to Clinical Quiz V:

Answers:
1) ECG, echo & chest x-ray
2) ASO titres, throat swab & blood culture ( 3 times)
3) Mitral regurgitation & bicuspid aortic valve stenosis
4) Acute rheumatic fever

(See page: )

Acute rheumatic fever causes significant morbidity and mortality in the pediatric age group. It is relatively rare in developed countries but common in developing countries. Carditis is the commonest manifestation, which will cause permanent damage. There will be a history of throat infection or tonsillitis 2-3 weeks prior to this. Polyarthritis moves from one joint to another, usually the large joints such as the knee, ankles, and elbows. Chorea, subcutaneous nodules and erythema marginatum are other major manifestations. The minor criteria include arthalgia, fever, a high CPR or ESR, leukocytosis, a prolonged PR interval and previous rheumatic fever. rheumatic heart disease. Diagnosis of acute rheumatic fever should be expected in patients with two major or one major and two minor signs. Carditis affects at least 2/3rd of patients. The appearance or worsening of a heart murmur, pericarditis and heart failure is an indication of carditis. The apical systolic murmur is one typical murmur that indicates mitral regurgitation. A short apical mid-diastolic murmur will indicate mitral regurgitation and, less commonly, the high-pitched decrescendo, early diastolic murmur will indicate aortic regurgitation. Pericarditis can be recognized by pericardial friction rub and is always associated with myocarditis and valvular lesions. ECG may show a prolonged PR interval, complete heart block, and flattened or inverted T-wave on V4-V6, characteristic of pericarditis. Echocardiography will help to determine the state of the myocardium and any pericardial effusions. It is important that the affected child have bed rest. Use of oral penicillin, salicylate, and corticosteroids for 10 days should only follow severe carditis or heart failure. Prophylaxis should be given to all patients with previous rheumatic fever. The prognosis is worse if there is aortic valve involvement or progressive cardiac dilatation.
Endoscopy in Oesophageal Disease

Niyaz A Jan

Radiographic study and endoscopy are the two main diagnostic techniques used to evaluate oesophageal symptoms. Oesophagoscopy has certain definite advantages over x-ray study: we can take biopsy and also undertake certain therapeutic interventions. Flexible endoscopy has emerged as the preferred method for examination of the oesophagus for many indications. Video endoscopy can provide excellent image quality, permit multiple viewing, and allow storage as well.

In the USA, and many other countries, patients are usually premedicated with an intravenous narcotic and a benzodiazepine, with or without topical pharyngeal anaesthesia. Studies have shown that many patients can tolerate endoscopy with topical anaesthesia alone. Use of small caliber endoscopes which can even be passed intranasally has the potential to decrease the use of intravenous sedation in future. In our place, we use.

Diagnostic endoscopy is an appropriate initial evaluation for many patients with oesophageal symptoms. It is also an appropriate procedure to further evaluate abnormalities seen on radiologic studies of oesophagus. Sequential oesophagoscopy may be indicated for surveillance of Barret’s oesophagus or to follow up endoscopic variceal therapy.

Diagnostic Indications for Oesophagoscopy
Dysphagia and odynophagia

Certain patients with GERD (not all)
Patients with suspected infectious oesophagitis
Acute or chronic bleeding
Abnormal results of a radiologic examination
Surveillance in Barret’s Oesophagus
To evaluate for varices in certain patients with suspected portal hypertension or following endoscopic variceal therapy
To assess acute injury after caustic ingestion

The major advantage of endoscopy over barium oesophagram is that biopsies can be obtained and a variety of therapeutic interventions can be performed. Therapeutic capabilities include stricture dilatation, removal of foreign body, treatment of varices and other bleeding lesions, and treatment of chalasia. Oesophageal malignancies can be palliated with dilatation, laser therapy, photodynamic therapy, and insertion of metal stents. Endoscopic therapy for the ablation of Barret’s oesophagus and endoscopic interventions for the treatment of GERD are currently being evaluated.

Therapeutic Indications for Oesophagoscopy
Dilatation of strictures, benign and malignant
Removal of foreign bodies
Treatment of bleeding varices
Treatment of achalasia
Palliative treatment of malignancy
Removal of selected polypoid lesions

In general, upper endoscopy can be performed safely in almost all patients. It should not, however, be performed in uncooperative or moribund patients. It should also not be performed in patients with a known or suspected perforated viscus. The nonsterile instrument and the air insufflation needed to perform a procedure may aggravate mediastinal or peritoneal
contamination. In unstable patients, endoscopy should only be performed if the benefits of endoscopy are judged to outweigh the risks to the patient at that time.

**Contraindications to Oesophagoscopy**

- **Absolute:** Lack of patient cooperation
- Moribund patient
- Known or suspected perforated viscus

- **Relative:** Unstable cardiovascular or pulmonary status.

Complications of upper gastrointestinal endoscopy are rare. The most common complications are reactions to the sedative medication. Hence, the use of pulse, blood pressure, heart rhythm, and oxygen saturation monitoring is encouraged. Although oesophageal perforation can occur with diagnostic oesophagoscopy, it is more likely to result from therapeutic procedure.

**Complications of Oesophagoscopy (%)**

- Medical reactions: 0.5%
- Perforation: 0.03-0.1%
- Bleeding with biopsy: 0.03%

**Comparison of Diagnostic Accuracy of Radiology & Endoscopy in Oesophageal Disease:**

Endoscopy is superb for mucosal lesions and has biopsy and therapeutic advantages. On the other hand, the role of endoscopy in the diagnosis of motor disorders is limited. For this reason, in patients with dysphagia to both solids and liquids, it may be more appropriate to obtain an oesophagram as the initial diagnostic procedure. An oesophagram also enables the clinician to rule out orophagesal causes of dysphagia that can not be assessed by endoscopy.

<table>
<thead>
<tr>
<th>Oesophageal Disease</th>
<th>Radiology</th>
<th>Endoscopy</th>
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<tbody>
<tr>
<td>Orophageal dysphagia</td>
<td>Excellent</td>
<td>Poor</td>
</tr>
<tr>
<td>Cervical web</td>
<td>Excellent</td>
<td>Fair</td>
</tr>
<tr>
<td>Extrinsic compression</td>
<td>Excellent</td>
<td>Poor</td>
</tr>
<tr>
<td>Oesophageal diverticula</td>
<td>Excellent</td>
<td>Good</td>
</tr>
<tr>
<td>Carcinoma</td>
<td>Excellent</td>
<td>Excellent</td>
</tr>
<tr>
<td>Acute caustic injury</td>
<td>Poor</td>
<td>Excellent</td>
</tr>
<tr>
<td>Pill-induced injury</td>
<td>Fair</td>
<td>Excellent</td>
</tr>
<tr>
<td>Foreign body</td>
<td>Good</td>
<td>Excellent</td>
</tr>
<tr>
<td>GERD</td>
<td>Fair</td>
<td>Good</td>
</tr>
<tr>
<td>Peptic stricture</td>
<td>Excellent</td>
<td>Excellent</td>
</tr>
<tr>
<td>Barretts epithelium</td>
<td>Fair</td>
<td>Excellent</td>
</tr>
<tr>
<td>Lower oesophageal mucosal ring</td>
<td>Excellent</td>
<td>Good</td>
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<tr>
<td>Infectious esophagitis</td>
<td>Good</td>
<td>Excellent</td>
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<td>Varices</td>
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<td>Excellent</td>
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<td>Achalasia</td>
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<td>Excellent</td>
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<tr>
<td>Diffuse oesophageal spasm</td>
<td>Fair</td>
<td>Poor</td>
</tr>
<tr>
<td>Nonspecific oesophageal motor dysfunction</td>
<td>Fair</td>
<td>Poor</td>
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</tbody>
</table>

**Diagnostic sensitivity of Oesophagram & Oesophagoscopy in Oesophageal Diseases**
Coffee is the name given to the seeds (beans) of a genus of evergreen trees of the madder family, and to the beverage made from them. Of the 40 species of the genus, only 3 are commercially important: arabica or Arabian, robusta or Congo, and Liberian. The shrub (small tree), 15 to 20 ft high at maturity, bears shiny green, oval leaves that persist for three to five years and white, fragrant flowers that bloom for only a few days. During the six or seven months after the appearance of the flower, the fruit develops, changing from light green to red and, ultimately, when fully ripe and ready for picking, to deep crimson. The mature fruit, which resembles a cherry, grows in clusters attached to the branches by very short stems, and it usually contains two seeds, or beans, surrounded by a sweet pulp.

Coffee makes up the genus Coffea of the family Rubiaceae. Arabica or Arabian coffee is classified as Coffea arabica, robusta or Congo coffee as Coffea canephora, and Liberian coffee as Coffea liberica or Coffea excelsoides.

The coffee species are indigenous to Africa and adjacent islands, but have been introduced elsewhere. Today coffee grows well on the islands of Java, Sumatra, and Papua New Guinea, and in the Caribbean, Africa, Arabia, India, and South and Central America. The Americas, where arabica coffee is grown, produce approximately two thirds of the world’s supply.

**HISTORY**

Exactly where and when coffee was first cultivated is not known, but some authorities believe that it was grown initially in Arabia near the Red Sea in about AD 675. Coffee cultivation was rare until the 15th and 16th centuries, when extensive planting of the tree occurred in the Yemen region of Arabia. The consumption of coffee increased in Europe during the 17th century, prompting the Dutch to cultivate it in their colonies. In 1714 the French succeeded in taking a live cutting of a coffee tree to the island of Martinique in the Caribbean. This single plant was the genesis of the great coffee plantations of Latin America.

Because of the economic importance of coffee exports, a number of Latin American countries made arrangements before World War II to allocate export quotas so that each country would be assured a certain share of the United States coffee market. The first coffee quota agreement was arranged in 1940 and was administered by an Inter-American Coffee Board. The idea of establishing coffee export quotas on a worldwide basis was adopted in 1962, when an International Coffee Agreement was negotiated by the UN. During the five-year period in which this agreement was in effect, 41 exporting
countries and 25 importing countries acceded to its terms. The agreement was renegotiated in 1968, 1976, and 1983. Participating nations failed to sign a new pact by the end of 1997.

Throughout its history, coffee has stimulated ideas, debates, commerce and development, and numbed & exposed people to exploitation. It has helped to subvert cultures, social systems, and governments. Coffeehouses operate as centres of a bourgeois lifestyle for literati and businessmen alike, as well as meeting place for those who agitated for democratic politics. One can trace history if one attempts to retrospectively follow path of coffee for the last 6 centuries.

**PRODUCTION**

The soil in which coffee is grown must be rich, moist, and absorbent enough to accept water readily, but sufficiently loose to allow rapid drainage of excess water. The best soil is composed of leaf mould, other organic matter, and disintegrated volcanic rock. Although coffee trees are damaged easily by frost, they are cultivated in cooler regions that are prone to frost. The growing temperatures range from 13° to 26° C. Altitudes of coffee plantations range from sea level to the tropical forest level, about 6,000 ft. Robusta coffee and Liberian coffee grow best at altitudes below 3,000 ft; arabica coffee flourishes at the higher altitudes. The seeds are planted directly in the field or in specially prepared nurseries. In the latter case, selected young plants are transplanted later to the fields. Commercial fertilizers are used extensively to promote the growth of stronger, healthier trees with greater yields. Intercropping with nitrogen-fixing species, and shading with tall species, which provide leaf litter, are practised in various parts of the coffee-growing world. Both the trees and the fruit are subject to insect infestation and microbial diseases, which may be controlled by spraying and proper agricultural management. In the 1990s the use of integrated pest management and various environmentally aware methods have increased. Various hybrids, some of which are disease-resistant, have been developed, and traditional methods of selection are being replaced by genetic-engineering techniques to incorporate commercially useful traits, such as dwarf habit, disease resistance, or the absence of caffeine.

**A Harvesting**

The coffee tree produces its first full crop when it is about five years old. Thereafter it produces consistently for 15 or 20 years with careful pruning and good husbandry. Some trees yield 0.9 to 1.3 kg of marketable beans annually, but 0.45 kg is considered an average annual yield. Two methods of harvesting are used. One is based on selective picking; the other involves shaking the tree and stripping the fruit. In some areas mechanical harvesting has been introduced to do this. Beans picked by the first technique are generally processed, if water is available, by the so-called wet method, in which the beans are softened in water, de-pulped mechanically, fermented in large tanks,
washed again, and finally dried in the open or in heated, rotating cylinders. This method can generate serious water pollution problems and considerable efforts are being made to convert the pulp to commercially valuable animal feed or mulch. The so-called dry method, used generally for beans harvested by the second technique, entails only drying the beans and removing the outer coverings. In either case the final product, called green coffee, is sorted by hand or machine to remove defective beans and extraneous material, and is then graded according to size, and sometimes colour, using electronic equipment.

Various accusations have been leveled at coffee, including that it causes heart attacks, high blood pressure, pancreatic cancer, peptic ulcers, hypoglycemia, foetal malformations, cystic breast disease, and nervousness; and that it shortens life. But research has refuted all these accusations, and now we know that coffee has more benefits for human life and health. Some 3-4 cups of coffee (equivalent to 200-300 mg caffeine) per day is a mild stimulant helpful in relieving minor fatigue and boredom, with little risk of any harmful effects.

A nationwide survey, conducted by the US Department of Health & Human Services, concluded that “There is no evidence that heavy coffee drinking -5 or more cups per day - is related to poor health.
## Commercial Crops

The major types of commercial coffee are the arabicas and the robustas. Arabicas are produced mainly in the Americas and East Africa and are usually wet processed. The dry-processed beans are described as “Brazils”, and the wet-processed arabicas are generally referred to as “milds”. Robustas are produced mostly in west Africa and Asia and are mostly dry processed. The Brazils consist principally of Santos, Paraná, and Rio, named after the ports from which they are shipped. Milds are identified by the names of countries or districts in which they are grown, such as Medellín, Armenia, and Manizales coffees from Colombia. Robustas and other arabicas are similarly identified.

Usually several varieties of green coffee are blended and roasted together to produce the tastes, aromas, and flavours popular with coffee drinkers. As a rule the beans are heated in rotating, horizontal drums that provide a tumbling action to prevent uneven heating or scorching. The roasted beans are cooled rapidly. A very slow-roasting process has also been developed, which exploits cheap waste materials for fuel. Roasted coffee may be packaged and shipped to shops, which grind it for the customers on purchase, or it may be ground in plate- or roller-type grinding mills before shipment.

Ground coffee loses its unique flavour within about a week unless it is specially packaged. Plastic-and-paper combinations are popular packagings that afford protection to freshly roasted and ground coffee. Hermetically-sealed vacuum, or pressure, cans keep coffee fresh for up to three years if unopened. Once opened, exposure to oxygen and moisture in the air accelerate the deterioration, but this can be retarded by sealing tightly and chilling in a refrigerator. Whole roasted beans contain a lot of carbon dioxide, which provides an inert atmosphere and acts as a preservative for the delicate aroma. Ground roasted beans contain less, but still a significant amount.

### CHARACTERISTICS

Coffee contains a complex mixture of chemical components, some of which are not affected by roasting. Other compounds, particularly those related to the aroma, are produced by partial destruction of the green bean during roasting. Chemicals extracted by hot water are classified as non-volatile taste components and volatile aroma components. Important non-volatiles are caffeine, trigonelline, chlorogenic acids, amino acids, carbohydrates, and minerals. Important volatiles are organic acids, aldehydes, ketones, esters, amines, and thiols (sulphur compounds also known as mercaptans). The principal physiological effects of coffee are produced by caffeine, an alkaloid that acts as a mild stimulant.
In recent years controversy has arisen over the possibly harmful effects of coffee. With the modest consumption of coffee, caffeine is of little concern to most, and any effects are relatively short-lived. There has been concern, however, that heavy consumption during pregnancy may harm the foetus.

During the 1990s a fat-soluble diterpene called cafestol, for which coffee is the only known source, was identified as the substance that causes a reversible increase in cholesterol. Diterpene only enters coffee when it is prepared by boiling ground beans in water as commonly practised in France, Italy, and Scandinavia. It does not enter filtered coffee or instant coffee powder. The risk factors for heart disease in humans, however, remain uncertain.

Green beans contain trigonelline, which, during roasting, is partially converted to niacin, one of the B vitamins. In some areas of the world, the coffee drink may be a very important source of this vitamin. Unless sugar, milk, or cream is added, coffee is low in calories.

FORMS OF COFFEE

A Instant Coffee

Instant or soluble coffee is an important product of the coffee industry. At one time it was manufactured only in the consuming countries, but there are now large commercial plants in the coffee-growing countries. In its manufacture an extract is prepared by mixing coarsely ground, roasted coffee with hot water under pressure. The extract is concentrated and dried by various methods, including the use of spray dryers. In freeze-dried coffee the concentrated coffee extract is frozen, and the water is removed by sublimation under high vacuum. The product is packed under vacuum in sealed jars or in cans. Only the addition of hot water is required to make the beverage. The extraction rate—that is, the amount of instant powder that can be made from a given amount of green bean—is controlled by legislation.

B Decaffeinated Coffee

Caffeine can be removed from coffee by treating the green beans with an ester or chlorinated hydrocarbon solvents. The decaffeinated beans are roasted by ordinary procedures after the removal of the solvents. More recently, supercritical carbon dioxide (carbon dioxide under great pressure) has been used commercially in Europe and the United States to remove the caffeine. This process is attractive as it leaves no solvent residues in the extracted beans, but it has the disadvantage of being more expensive. Decaffeinated coffee is used by people who prefer to avoid the caffeine present in ordinary coffee.

C Coffee Substitutes

The use of substitutes for coffee is limited and controlled by legislation. The most important substitute is roasted chicory, although chicory is usually used as an extender. Roasted dandelion root, figs, and extracts of various cereals are
also used. In most countries, the addition of chicory or any other substance must be clearly stated on the brand label.

**Consumption:**

Worldwide, humans drank about 380 billion cups of coffee in 1991 or about 76 cups for each man, woman and child on the planet.

Although poor Yemenis were the first to taste coffee 600 years ago, now coffee is associated with nations, societies and families with a higher socioeconomic status.

Coffee is still very much a commodity consumed in rich countries and produced by poor ones – the per capita consumption is related to the per capita income in the consuming country. Americans are the world’s biggest consumers of coffee, with an average per capita of more than 2 kg per year. Yet in 1992, most of the world’s top 10 coffee consuming nations were in Northern Europe (in order Finland, Sweden, Denmark, Norway, Netherlands, Switzerland, Austria, Germany, France & Belgium). In Sweden, an average person drinks as much as 5 cups per day. Coffee producing countries increased their consumption from less than 10% of the total produce in early 20th Century to as high as ¼ towards the beginning of the 21st Century. Costa Ricans have the greatest taste for their own beans, Brazilians being the second. In Africa, only Ethiopians like to drink some of their own coffee; others don’t even taste their own produce.

Coffee consumption is on the rise throughout the world, more rapidly in countries with a higher or progressively increasing national income. Thus, countries in Asia with no historic connection to coffee plantation or consumption (Japan, Korea, Singapore) are rapidly increasing the per capita consumption.
Caffeine

The beneficial medicinal effects of caffeine (in tea) were recognized by the Chinese Emperor Shen Nung as early as 2737 BC; however, it was first isolated from green coffee beans in Germany in 1820 AD, subsequently was isolated from mat and kola nuts. The complete synthesis of caffeine was reported by Fischer and Ach in 1895. When theobromine (dimethyltheobromine) was methylated in 1861 it changed into caffeine, which led to the discovery that caffeine was structurally trimethylxanthine. Caffeine was discovered in coffee in 1820. In 1838 it was established that theine, discovered in tea in 1827, is identical to caffeine. Caffeine is produced commercially chiefly as a by-product in making caffeine-free coffee.

Caffeine, an alkaloid \((C_8H_{10}O_2N_4\cdotH_2O)\) found in coffee, tea, cacao, and some other plants. It is also present in most cola beverages. Caffeine, as other xanthines, is derived from a core pure purine structure, which forms the parent compound for nucleic acids including adenosine. This describes the CNS effect of caffeine. The behavioural effects (CNS stimulation) of caffeine falls in between theobromine (weak) and theophylline (Strong).

Methyxanthines inhibits phosphodiesterase, the enzyme which metabolizes cyclic adenosine monophosphate (cAMP), causing an accumulation of cyclic nucleotide. However, the concentrations required for this effect are much in excess of those found in the blood. The current body of knowledge suggests that the therapeutic effect of methyxanthines involves blockade of adenosine receptors, some of which have been characterized at the molecular and physiological levels.

Caffeine increases the blood pressure, stimulates the central nervous system, promotes urine formation, and stimulates the action of the heart and lungs. Caffeine is used in treating migraine because it constricts the dilated blood vessels that are believed to be involved in the causation of migraine. It also increases the potency of analgesics such as aspirin, and it can somewhat relieve asthma attacks by widening the bronchial airways.

Methyxanthines may cause gastric irritation; they also lead to cardiovascular effects as palpitation, headaches, insomnia, and dizziness. The effect is more pronounced with theophylline which is the most potent of methyxanthines. Caffeine has been suggested as a possible cause of cancer or of birth abnormalities. No studies, however, have yet confirmed any of these charges. People who stop drinking coffee do sometimes experience withdrawal headaches.
VOMITING IN PREGNANCY

Rehana Kausar

Vomiting in pregnancy is a common yet mild complaint though it could be manifestation of a severe underlying disease. Typically it commences between the first and second missed menstrual period and continues till the time of the fourth missed period. Nausea and vomiting are particularly worse in the morning but may continue throughout the day.

The causes of vomiting of pregnancy can be classified as:

- **Related to pregnancy:**
  a) Simple vomiting or morning sickness / emesis gravidarum
  b) Hyper emesis gravidarum
  c) Acute fulminating preeclampsia

- **Associated with pregnancy:**
  a) Urinary tract infection
  b) Hepatitis
  c) Appendicitis
  d) Peptic ulcer
  e) Intestinal obstruction
  f) Cholecystitis
  g) Intestinal obstruction
  h) Uremia
  i) Diabetic ketoacidosis
  j) Gynecological causes like twisted ovarian cyst or red degeneration of fibroid

**Morning sickness or emesis gravidarum** is so common in pregnancy that it is regarded as an inevitable symptom of pregnancy. Chorionic gonadotrophin has been implicated in its genesis, on the basis that its levels are high at the same time that nausea and vomiting are most common. In fact women with hydatidiform mole who typically have high levels of HCG in pregnancy complain of severe nausea and vomiting in pregnancy. Emotional factors have an important role to play in the severity of gestational nausea and vomiting. Morning sickness appears 2 weeks after a missed period and diminishes by 14 weeks in up to 50% of women. Morning sickness has been known for thousands of years; Egyptians described these symptoms as early as 2000B.C.

**Treatment:** There is no effective treatment for nausea and vomiting in pregnancy. Reassurance, advice to move the limbs before moving out of bed, taking dry toast before rising from bed and avoidance of fatty foods are enough to relieve the symptoms in majority. If these simple measures fail, antiemetics like trifluperazine, 1mg twice daily are effective. Patient is advised to take lots of fluids – at least 2.5 litres daily.

**HYPEREMESIS GRAVIDARUM**

Hyperemesis gravidarum is a severe type of vomiting of pregnancy, which has got deleterious effect on the health of mother and incapacitates her.

The incidence of hyper-emesis has reduced in the last 30 years; it is now encountered 1 in 1000 pregnancies. This is because of improved antenatal care and potent antihistaminics being available.
Occurs commonly in first trimester
Is more common in first pregnancy
There is strong family history: mothers and sisters suffer from same complication
Is more prevalent in hydatidiform mole and twin pregnancy
Is common in unplanned pregnancy

The primary clinical manifestation of hyperemesis is sustained and frequent vomiting, usually 4—8 weeks in duration and resulting in significant weight loss and dehydration. Other signs of starvation that usually develop are
  o Metabolic acidosis,
  o Ketosis
  o Oliguria
  o Hemoconcentration
  o Constipation
  o Hypokalemic alkalosis

Severe forms of hyperemesis involving weight loss of greater than 5% of pre-pregnant weight have been associated with poor fetal growth and poor outcome. Continued vomiting can lead to brainstem lesions resembling that characteristic of Wernicke’s encephalopathy.

Various theories of causation have been put forward:

1. **Hormonal** - excess of hCG and hyperthyroidism.
2. **Deficiency** of vitamin B6 owing to a change in protein metabolism.
3. **Impaired functioning of the adrenal cortex**
4. **Psychopathological** and emotional factors
5. **Alteration in gastrointestinal physiology**
6. **A hypersensitivity reaction**
7. **Poor nutrition**

Other causes of vomiting must be ruled out especially if vomiting starts after 12 weeks of gestation.

Investigations in a case of hyperemesis gravidarum include
- Temperature
- Blood pressure
- Weight on admission and daily weight
- Urinalysis
- Strict fluid assessment (intake and output)
- Electrolyte status (potassium, sodium, chloride)
- Blood urea nitrogen
- Blood Glucose
- Serum creatinine

Maternal complications
- Hemorrhagic retinitis
- Rupture of the esophagus
- Aspiration pneumonitis
- Electrolyte depletion
- Acid–base balance disruption
- Dental erosion

Treatment:

- The principal underlying treatment of hyperemesis is to prevent starvation and dehydration and recognizing any psychological component that may be present.
• Treatment includes hospitalization in a quiet room and a complete history so that emotional problems are ruled out. Parenteral nutrition is needed in some cases to maintain an anabolic state. Parenteral nutrition has moved on from its previous regimen of sodium chloride and dextrose to infusions of amino acids, electrolytes, carbohydrates, fat emulsions and trace elements. Vitamin B complex, vitamin C, and vitamin B₆ (100mg) should be added to intravenous solutions.

• Gastric rest, which means not allowing any oral intake, is helpful.

• Antiemetics or mild sedatives like promethazine 25 mg, prochlorperazine, trifluperazine, and phenobarbitone 16-32-mg one hour before meals or bedtime should be used. One study identified steroids as a successful management of hyperemesis. However, some drugs may be teratogenic to the fetus and their use should be scrutinized.

• As the patient begins to respond to intravenous therapy (as evidenced by cessation of vomiting, return of electrolyte balance to normal and an increase in urine output), small sips of water should be given. As soon as the patient can tolerate food, frequent, small meals consisting of fairly dry, easily digested, high-energy foods are given. Similarly, antiemetics and sedatives are slowly withdrawn or tapered.

• A psychiatric consultation should be arranged.

There appears to be no correlation between hyperemesis and delivery of small–for–gestational age baby except in the event of severe hyperemesis. Similarly, there appears to be no increase in the risk of congenital anomalies associated with the condition. Ketonuria, however, should be prevented because fetal anomalies are increased if ketonuria persists.

Complications of antiemetic therapy
- Jaundice
- Irregular jerky movements with opisthotonus with phenothiazines
Subcutaneous extravasation

Although peripheral intravenous administration of fluids and drugs is now very common, it is fraught with danger of extravasation, that is, unintentional instillation or leakage of agents into the perivascular and subcutaneous spaces during their administration. Cannulation devices are easily misplaced or once correctly placed run the danger of getting displaced. This is commoner in our place where the patient or the attendants may fiddle with the needle or the tubing. This is also possible that despite correct placement of the cannula, intimal inflammation occurs because of the infused drug or fluid and leads to narrowing of the vessels which increases the local pressure and eventually leads to extravasation. Injury to local tissues (skin or underlying structures) may occur if the agent or carrier fluid is directly cytotoxic or if an innocuous agent creates adverse local conditions as elevated tissue pressure and ischaemia.

The extent of injury varies anywhere from minimal discomfort to extensive necrosis. Various drugs which may lead to local damage on extravasation are enumerated in the Table.

Table: Extravasation of drugs and their hazards

<table>
<thead>
<tr>
<th>Nature of drug</th>
<th>Representative drugs</th>
<th>Effect on extravasation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irritant drugs</td>
<td>Cyclophosphamide</td>
<td>Local pain &amp; irritation</td>
</tr>
<tr>
<td></td>
<td>Carmustine/BCBNU</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dacarbazine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Streptozocin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Teniposide</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thiotepa</td>
<td></td>
</tr>
<tr>
<td>Vesicant drugs</td>
<td>Actinomycin D (Daunorubicin; Doxorubicin)</td>
<td>Blistering &amp; ulceration</td>
</tr>
<tr>
<td></td>
<td>Anthracyclines (Daunorubicin; Doxorubicin)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chromomycin A3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cisplatinum</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Carboplatinum</td>
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<tr>
<td></td>
<td>Epirubicin</td>
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<tr>
<td></td>
<td>Fluorouracil</td>
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<tr>
<td></td>
<td>Ifosfamide</td>
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<tr>
<td></td>
<td>Mechloethamine</td>
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<tr>
<td></td>
<td>Methotrexate</td>
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<tr>
<td></td>
<td>Mithramycin</td>
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<tr>
<td></td>
<td>Mitomycin C</td>
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<tr>
<td></td>
<td>Mitozantrone</td>
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<td></td>
<td>Mustine</td>
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<tr>
<td></td>
<td>Streptozocine</td>
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<tr>
<td></td>
<td>Vinblastine</td>
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<tr>
<td></td>
<td>Vincristine</td>
<td></td>
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<tr>
<td></td>
<td>Vindestine</td>
<td></td>
</tr>
<tr>
<td>Ischaemia-producing drugs</td>
<td>Dobutamine</td>
<td>Produce local ischaemia of tissues</td>
</tr>
<tr>
<td></td>
<td>Dopamine</td>
<td></td>
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<tr>
<td></td>
<td>Epinephrine</td>
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<tr>
<td></td>
<td>Metaraminol</td>
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<tr>
<td></td>
<td>Norepinephrine</td>
<td></td>
</tr>
</tbody>
</table>

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Diagnosis of extravasation is clinical; some drugs are however retained in the tissues and thus can be detected by laboratory tests. Such drugs include contrast agents, ethylenediamine tetracetic acid (EDTA), and anthracyclines.

Symptoms include local pain, burning sensations, and other paresthesias, and pressure at the site. Various signs of extravasation are erythema, immediate swelling with extravasated infusion solution and later swelling in the reactive tissue oedema, induration, slowing of infusion rate, and failure to aspirate blood from the catheter.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local pain,</td>
<td>Redness</td>
</tr>
<tr>
<td>Burning sensations,</td>
<td>Swelling</td>
</tr>
<tr>
<td>Other paresthesias,</td>
<td>Induration</td>
</tr>
<tr>
<td>Pressure at the site.</td>
<td>Slowing of perfusion rate</td>
</tr>
<tr>
<td></td>
<td>Failure to aspirate blood from catheter</td>
</tr>
</tbody>
</table>

Induration that persists for more than 24 hours may be an indicator of impending necrosis and ulceration, especially with drugs like anthracycline. Severe extravasations may occur in patients who are unable to feel or complain of symptoms (of pain & pressure).
Extravasations occur even in the best institutions of the developed world. Children are more at risk, as are hysterical and anxious patients. But in our hospitals and health centres the incidence of extravasations is very high. The prime reason is the faulty training imparted to the nursing staff. This, coupled with lack of dedication and commitment to the patient and the noble service, is responsible for extravasations in case intravenous drugs and solutions and abscess formation with intramuscular injections.

**Complications:** The outcome of extravasation injury is generally favourable, but severe complications as median and ulnar neuropathy, limb shortening, disabling joint stiffness and amputation have been recorded.

Necrosing ulcers may appear within 14-21 days, tend to be lazy, full-thickness and painful, and may expand over weeks if the offending drug remains active at the site. Such phenomena can follow extravasation of toxic drugs like adriamycin. This may require plastic surgery.

Huge and deep extravasations and those with severe disruption of local vasculature, may increase pressure in a limited space which in turn compromises circulation and function of tissues in that space (Acute Compartment Syndrome). More prone are the structures (muscles, nerves, blood vessels) tightly enveloped by fascia and bone, and various compartments of the forearm, hand, leg and foot are more commonly affected areas. The earliest sign in the acute compartment syndrome is diminution of two-point discrimination (>1 cm) in the distal distribution of the affected peripheral nerve. Symptoms that follow include weakness, severe resting pain increased by passive stretch of involved musculature, tenseness of overlying skin, and distal absence/feebleness of pulse. Acute compartment syndrome can lead to widespread necrosis and therefore, requires immediate attention of a specialist.

**Prevention of extravasation**

Australian hospitals have set up guidelines for peripheral intravenous therapy of antimitotic drugs with a grave extravasation injury potential.

1) **Staff:**
   - Experienced in techniques of drug preparation
   - Sound knowledge of properties and side effects of drugs
   - Acquainted with a protocol for management of extravasation injuries
   - Working in quiet environment

2. **Administration:**
   - **Veins:**
     - Vein preference sequence: Large forearm > dorsal hand > wrist > antecubital fossa
     - Away from joints, in areas of deep soft tissue
     - Avoidance of obstructed veins (Draining areas of super vena cava syndrome, surgical obstruction, peripheral vascular disease, radiation sites)
     - Minimal attempts at venipuncture
     - Venodilation of small veins 5-10 minutes before cannulation (Local heat; topical glyceryl trinitrate paste)
     - Maintained venodilation if necessary (Topical glyceryl trinitrate paste, 8 hourly)
Cannulae:
  Shortterm infusion via 21-gauge butterfly set.
  Longterm infusions via small-gauge flexible synthetic catheter
  Prolonged infusions most safely delivered via centrally placed catheter
  Implanted venous access devices (Monitored for needle/port and catheter continuum)
  Patency assessed by free flow of normal saline or Dextrose 5%, and easy blood return

Delivery systems:
  Free flow infusion or volumetric cassette pumps (20 pounds per sq inch or less)
  Avoidance of manual push infusions and high pressure pumps.

3) Drugs:
  Appropriate dilution of drug
  Infusion over shortest period consistent with patient’s venous capacity
  Vesicants given first sequence or last
  Minimum 10 ml normal saline or dextrose 5% flush after drug infusion to dilute local drug concentration
  Removal of cannula and application of pressure to site after drug infusion.

If all these precautions are taken, peripheral intravenous drug or fluid administration should not be fraught with dangers of injury to the patient and litigation to the practitioner.
The aetiological agent of TB is *Mycobacterium tuberculosis*. Other mycobacteria occasionally produce disease clinically indistinguishable from tuberculosis (TB), which are identifiable only through culture.

Transmission is mainly through air by inhalation of droplet nuclei. The initial infection usually goes unnoticed. Tuberculin sensitivity appears within a few weeks of infection. Initial lesions commonly heal leaving no residual changes except occasional pulmonary or tracheo-bronchial lymph node calcifications (primary complex). Approximately 95% of those initially infected enter this latent phase from which there is life-long risk of reactivation. In approximately 5%, the initial infection may progress directly to pulmonary TB or by lympho-haematogenous dissemination of bacilli, to pulmonary, miliary, meningeal or other extra-pulmonary involvement. The initial infection has a serious outcome more frequently in infants, adolescents and young adults.

Extra-pulmonary TB is much less common than pulmonary TB. It may affect any organ or tissue and includes TB meningitis, miliary TB, involvement of lymph nodes, pleura, bones, joints, intestines, pericardium, kidney, skin, etc.

Progressive pulmonary TB arises from endogenous reactivation of latent foci which remained dormant since the initial infection, or exogenous reinfection which, if untreated, leads to death within 2–3 years in at least half the patients.

**Magnitude:** The disease occurs worldwide, with a higher incidence in developing countries. In India, the estimated prevalence of sputum-positive patients is 0.4% (3.5 million cases). Under the National TB Control Programme, approximately 1.5 million total cases were detected and put on treatment every year. An estimated 0.5 million deaths from TB occur every year.

A person infected with *M. tuberculosis* who is not infected with HIV has approximately a 10% lifetime risk of developing tuberculosis disease; 50%–80% of this risk is in the first two years after infection with *M. tuberculosis* in these HIV-negative patients. Persons infected with *M. tuberculosis* who are also HIV infected have at least a 50% lifetime risk of developing tuberculosis, with an annual risk of developing disease of approximately 7%–10%, which is many times higher than that of HIV-negative patients. In developed countries, the mortality and morbidity from TB was declining over the last few decades but since the 1980s morbidity has increased especially in areas or population groups with high prevalence of HIV.

The prevalence of infection detected by tuberculin testing increases with age and in India it is more than 40% in adults.

**Forms of tuberculosis**

Tuberculosis is most commonly transmitted by inhalation of infected
droplet nuclei which are discharged in
the air when a patient with untreated
sputum positive TB coughs or sneezes.
If the bacillus succeeds in infecting a
person, active disease results in only
about 5%–10% of those who had
primary infection. Infection occurs
almost exclusively through the
respiratory route. Tuberculosis then
spreads from the primary lung lesion to
other parts of the body via the blood
stream, lymphatic and bronchial systems
and may thus affect any organ.

Various types of tuberculosis
encountered are:

**Pulmonary TB**: Tuberculosis affects the lungs
in more than 80% of cases. Pulmonary TB
which is sputum smear-positive is highly
infectious and should receive topmost
priority for treatment. Cases which are only
sputum culture-positive but smear-negative,
are much less infectious than those which
are smear-positive.

**Extra-pulmonary TB** can affect any part of the
body, such as the lymph nodes, bones and
joints, the genito-urinary tract, the nervous
system (meningitis), intestines, etc.

**Diagnosis**
is often difficult and it should be made by a
physician. Patients with extra-pulmonary
TB
(without concomitant pulmonary TB) hardly
ever spread the disease to others.

**Tuberculosis in children**: Sputum usually
cannot be obtained from children and, in
any case, it is often negative even on culture.
The diagnosis of TB in children therefore
rests largely on clinical history, contact
history, X-ray examination and tuberculin
testing. The decision whether or not to treat
the child for TB should be made by a
physician. Generally, any tuberculin-
positive child under 5 years of age who is a
contact of an adult sputum-positive case and
has signs or symptoms suggestive of TB
should be regarded as having active TB and
given a full course of treatment, regardless
of whether or not he has been vaccinated
with BCG.

**When should tuberculosis be
suspected?**
The most common symptoms of
pulmonary TB are persistent cough
(usually with sputum, sometimes blood-
stained), fever and chest pain for 3
weeks or more. Constitutional symptoms
like lethargy, lassitude, loss of appetite
and weight loss may be associated. In
extra-pulmonary TB, symptoms depend
on the organs involved, for example:
swelling, occasionally with pus
discharge when lymph nodes are
affected;
pain and swelling of the joints if these
are involved; headache, fever, stiffness
of the neck and mental confusion when
there is tuberculous meningitis.

Health education is a critical
component of tuberculosis control. The
different target groups which need to be
addressed are patients, their relatives,
medical and paramedical professionals
and the community. It should reinforce
positive attitudes and eliminate
negative ones. It must be emphasized
that TB is almost 100% curable with
adequate treatment. Directly Observed
Treatment with standardized Short-
Course Chemotherapy (DOTS) as per
the recommendations is the key to
curing TB. If treatment is not taken as
per recommendations it is likely that the
disease may become incurable (drug-
resistant). Such a patient can spread an
incurable form of the disease in his
household and the community. The
public should be made aware of the
risks of irregular or incomplete
treatment. Health professionals, the
community and paramedics should be
made aware that TB is best diagnosed
by sputum examination and that almost 100% of patients are cured by DOTS. The general public should be taught the importance of reporting at a health facility at the earliest if they have chest symptoms, especially productive cough persisting for 3 weeks or more. Patients with these symptoms should undergo a sputum examination at the nearest health facility. People should be informed of the location and facilities available for managing TB at the community level.

A “case” of tuberculosis

A case of pulmonary TB is a patient who is sputum-positive for Acid-Fast Bacilli (AFB) or if found sputum-negative is considered by a physician to be suffering from the disease on the basis of clinical and radiological evidence. A case of extra-pulmonary TB is a patient who is considered by a physician to warrant complete treatment based on clinical, histological, or other evidence. All cases of tuberculosis should be registered.

Classification of tuberculosis cases

It is important to classify cases of TB in order to determine the correct combination of drugs and duration of treatment. Classification of pulmonary cases should be based on 3 sputum smear examinations. Sputum should also be examined for cases of suspected extra-pulmonary TB if pulmonary symptoms are present.

1) Pulmonary tuberculosis, smear-positive

TB in a patient with at least 2 initial sputum smear examinations (direct smear microscopy) positive for AFB, Or: Tuberculosis in a patient with one sputum examination positive for AFB and radiographic abnormalities consistent with active pulmonary TB as determined by the treating Medical Officer, Or: Tuberculosis in a patient with one sputum specimen positive for AFB and culture positive for M. tuberculosis.

2) Pulmonary tuberculosis, smear-negative

TB in a patient with symptoms suggestive of TB with at least 3 sputum examinations negative for AFB, and radiographic abnormalities consistent with active pulmonary TB as determined by a Medical Officer, followed by a decision to treat the patient with a full course of anti-tuberculosis therapy, Or: Diagnosis based on positive culture but negative AFB sputum examinations.

3) Extra-pulmonary tuberculosis

TB of organs other than the lungs, such as the pleura (TB pleurisy), peripheral lymph nodes, abdomen, genito-urinary tract, skin, joints and bones, tubercular meningitis, tuberculoma of the brain, etc. Diagnosis should be based on one culture-positive specimen from an extra-pulmonary site, or histological evidence, or strong clinical evidence consistent with active extrapulmonary TB followed by a Medical Officer’s decision to treat the patient with a full course of anti-tuberculosis therapy. Pleurisy is classified as extra-pulmonary TB. A patient diagnosed with both pulmonary and extra-pulmonary TB should be classified as a case of pulmonary TB.

Types of Cases

New case: A patient who has never had treatment for tuberculosis or has taken anti-tuberculosis drugs for less than one month.
**Relapse:** A patient declared cured of TB by a physician, but who reports back to the health service and is found to be bacteriologically positive.

**Transferred in:** A patient who has been received into a Tuberculosis Unit/District, after starting treatment in another unit where he has been recorded.

**Treatment after default:** A patient who received anti-tuberculosis treatment for one month or more from any source and who returns to treatment after having defaulted, i.e., not taken anti-TB drugs consecutively for two months or more.

**Failure case:** Smear-positive patient who is smear-positive at 5 months or more after starting treatment. Failure also includes a patient who was initially smear negative but who becomes smear positive during treatment.

**Chronic case:** A patient who remains smear-positive after completing a retreatment regimen.

**“Other” case:** Patients who do not fit into the above-mentioned categories. Reasons for putting a patient in this category must be specified.

### Case-finding methods

All the following increase the yield, should be undertaken for the success of the control programme.

1. Examination of sputum of patients with symptoms suggestive of TB (productive cough for 3 weeks or more with or without haemoptysis, fever, chest pain, weight loss or night sweats), who present on their own initiative at health facilities;

2. Promotion of awareness in the community, the medical profession and all medical staff regarding respiratory symptoms, notably persistent productive cough for 3 weeks or more, and the need to obtain and examine 3 sputum specimens for the diagnosis of TB;

3. Examination of household contacts (especially children below 5 years) of smear-positive TB patients; and

4. Examination of the sputum of a patient who, for any reason, has had an X-ray of the chest which has shown abnormality consistent with active TB.

### Diagnosis

Microscopic examination of sputum is, as a rule, the only way by which the diagnosis of pulmonary TB can be confirmed. Whenever TB is suspected, at least 3 specimens of sputum should be collected and examined by microscopy. If possible, they should be obtained over 2 days.

**First visit to the microscopy centre:** A spot specimen is collected; this is a specimen obtained on the spot after coughing and clearing the throat, under supervision of a staff member. After collection of first specimen, the patient is then given a sputum container for collection of an early morning specimen and instructed to come with this sputum sample on the next working day.

**Second visit to the microscopy centre:** The early morning collection of sputum specimen (second specimen) brought by the patient is received and a further spot specimen is collected (third specimen).
All specimens should be examined in the nearest microscopy laboratory at the earliest.

**Zeil-Neelsen Staining Method**

1. Select a new unscratched slide and label the slide with the Laboratory Serial Number.
2. Spread sputum on the slide using a broomstick.
3. Allow the slide to air dry for 15–30 minutes.
4. Fix the slide by passing it over a flame 3–5 times for 3–4 seconds each time.
5. Pour filtered carbol fuchsin to cover the entire slide.
6. Gently heat the slide with carbol fuchsin on it until vapours rise. Do not boil.
7. Leave carbol fuchsin on the slide for 5 minutes.
8. Gently rinse the slide with tap water until all free carbol fuchsin stain is washed away.
9. Pour 25% sulphuric acid onto the slide.
10. Let the slide stand for 2–4 minutes.
11. Rinse gently with tap water. Tilt the slide to drain off the water.
12. If the slide is still red, reapply sulphuric acid for 1–3 minutes and rinse gently with tap water.
13. Pour 0.1% methylene blue onto the slide.
14. Leave methylene blue on the slide for 30 seconds.
15. Rinse gently with tap water.
16. Allow the slide to dry.
17. Examine the slide under the microscope using x40 lens to select the suitable area and then examine under x100 lens using a drop of immersion oil.
18. Record the results in the Laboratory Form and the Laboratory Register appropriately as per the table given below:

<table>
<thead>
<tr>
<th>Examination Result</th>
<th>Grading</th>
<th>No. of fields to be examined</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than 10 AFB per oil immersion field</td>
<td>Pos 3 +</td>
<td>20</td>
</tr>
<tr>
<td>1–10 AFB per oil immersion field</td>
<td>Pos 2 +</td>
<td>50</td>
</tr>
<tr>
<td>10–99 AFB per 100 oil immersion fields</td>
<td>Pos 1 +</td>
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</tr>
<tr>
<td>1–9 AFB per 100 oil immersion fields</td>
<td>Scanty;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Record exact number</td>
<td>200</td>
</tr>
<tr>
<td>No AFB in 100 oil immersion fields</td>
<td>Neg/ 0</td>
<td>100</td>
</tr>
</tbody>
</table>

19. Store all positive and negative slides until instructed by the supervisor.
20. Disinfect all contaminated material before discarding.

If the first spot specimen is positive by microscopy and the patient does not return for the second sputum test, an immediate search must be made to find the patient to prevent dissemination of infection in the community. In the interest of the patient, second and third specimens of sputum must be collected and examined. To facilitate this it is important to note down the complete address of all symptomatic patients who are being evaluated.

If required, a course of symptomatic treatment or antibiotics...
suitable for non-tuberculous infection (but not streptomycin or rifampicin) may be given while awaiting the laboratory smear reports on the specimens. If a smear-negative patient fails to respond to this treatment and remains ill, the patient should be referred for further investigation (clinical and radiological). The extra-pulmonary cases with cough should also be examined by sputum smear to exclude pulmonary TB.

Treatment for TB shall be started as soon as two positive laboratory reports of smear examination are received. Treatment for TB in patients with a single positive laboratory report should be determined by a Medical Officer. Treatment will usually not be started in the absence of a positive laboratory report unless it is prescribed by a physician on the basis of the clinical examination, chest X-ray film suggestive of TB and at least 3 negative smear results.

On each supervisory visit to the health centre, the health professional in charge of TB control will check the documents of all patients who have been diagnosed as suffering from TB since the last visit, including those with either a single or no positive laboratory report. Treatment of smear-negative patients should not be started without 3 sputum samples having been examined for AFB.

X-ray: The diagnosis of TB by X-ray is unreliable, because other chest diseases can resemble TB on an X-ray, and because pulmonary TB may show various types of radiographic abnormalities. It must be stressed that the determination of clinical activity of TB by X-ray is totally unreliable. Moreover, the cost of X-ray examination is relatively high in relation to case-finding by smear microscopy. Consequently, the diagnosis of TB in adults must, as a rule, be confirmed by smear examination.

X-ray examination can undoubtedly be helpful in clinical work-up when investigating patients with symptoms suggestive of TB who have negative AFB smears, contacts of infectious cases, and in patients suffering from miliary or extra-pulmonary TB. In patients with chest symptoms in whom all 3 smears for AFB are negative, a course of antibiotics for one to two weeks should be tried before taking a chest X-ray.

Tuberculin Test: The tuberculin test has limited value in clinical work, especially in countries like India which have a high prevalence of TB infection. A “positive” tuberculin test (10 mm or more induration after 48 hours with 1 TU of PPD) is merely an indication of infection and is infrequently followed by disease. A “negative” tuberculin test does not necessarily exclude active TB. Moreover, a “positive” tuberculin test may be due to infection with mycobacteria other than M. tuberculosis or due to BCG vaccination. However, the tuberculin test is important in clinical work with children in whom a positive test is more likely to reflect recent infection with TB and indicates a much higher risk of developing disease.

Diagnosis of extra-pulmonary TB can generally be made by a physician.
Diagnosis in children is made by a physician on the basis of clinical symptoms, a positive Mantoux tuberculin skin test, chest X-ray and history of contact with a case of TB.

**Aims of the laboratory service**

The aims of the laboratory service are: (i) the diagnosis of cases, and (ii) monitoring of treatment. The TB laboratory service consists of a network of laboratories throughout the country which carry out, as part of their work, microscopic examination of sputum smears stained by the Ziehl-Neelsen method, and also includes Reference Laboratories for Tuberculosis at the State and Central levels.

The Reference Laboratory of Tuberculosis should be capable of training and supervising the staff of the network of microscopy centres. It should provide quality control services for smear microscopy. Some reference laboratories should have facilities for culture and sensitivity tests. Culture and sensitivity tests are not done as a matter of routine for diagnosis and are primarily of value for drug sensitivity studies in cases of treatment failures and for research purposes.

Efficient peripheral laboratories play a crucial role in the success of the case-finding programme based on the detection of smear-positive cases. Microscopy centres for examination of sputum for detecting tubercle bacilli are usually located in hospitals and health centres.

**Smear examination: Diagnosis & follow-up**

**Sputum-positive cases**

For the diagnosis of a new case, 3 sputum samples must be tested. During follow-up, 2 smears have to be tested each time:

(a) at the end of the intensive phase (2 months for new cases and 3 months for retreatment cases), at the end of 4 months (5 months for retreatment cases) and at the end of treatment.

(b) If the smear is positive at the end of the intensive phase, it should be tested again at 3 months in new cases and at 4 months in retreatment cases.

All smear-positive slides must be cross-checked by the Laboratory Supervisor and later they may either be broken, disinfected and disposed of like any other glass scrap. All negative slides after laboratory cross-check may be washed thoroughly and can be reused, but not for TB work.

**Sputum-negative cases**

During follow-up, 2 smears must be tested at the end of 2 months and also at the end of treatment.

**Culture**

Direct smear examination has the highest priority. If available, culture of tubercle bacilli may be helpful, although in sputum-negative cases a clinical decision to treat for TB based on X-ray findings and lack of response to broad-spectrum antibiotics would be more practical and also ensure prompt treatment. Culture and sensitivity testing is valuable for epidemiological surveillance, planning and management of resistant/failure cases.
Patient Education: Amniocentesis

Amniocentesis is a prenatal screening procedure in which a small quantity of the amniotic fluid surrounding the foetus is removed from the uterus of a pregnant woman to allow the chromosomes of the foetus to be examined. The test is usually performed between 16 and 20 weeks of pregnancy in women considered to be at high risk for having a baby with chromosomal anomalies, although some centres are now performing it as early as 10 to 14 weeks.

Amniocentesis is most commonly used in pregnant women over the age of 35 to detect chromosomal abnormalities, particularly Down’s syndrome (trisomy 21). The risk of chromosomal defects is known to increase with maternal age, and generally after age 35 the risk of bearing a child with a chromosomal abnormality is greater than the risks connected with the procedure. Amniocentesis cannot determine the severity of Down’s syndrome or any other defect, however.

A woman younger than 35 might consider amniocentesis if any earlier screening procedure such as an alpha-fetoprotein test or ultrasound indicates the possibility of a genetic abnormality. Amniocentesis is also sometimes used later in pregnancy to determine the lung maturity of babies at risk because of premature labor. Although the procedure provides an opportunity for women to anticipate many potential defects, any woman considering it should also bear in mind that in rare cases it can reveal abnormalities about which little is known, thus leaving her to make painful decisions about whether to carry the fetus to term.

How is amniocentesis performed?

A woman undergoing amniocentesis lies on an examination table with her abdomen uncovered. With the aid of ultrasound, the physician guides a thin needle (usually about 3Â inches long) through the abdomen into the uterus and amniotic sac. About 1 ounce of amniotic fluid, which contains cells sloughed off by the foetus, is then withdrawn by a syringe attached to the needle. The actual procedure takes about 5 minutes and can be performed on an outpatient basis in either a doctors office or a hospital. It is relatively painless.

The fluid is sent to a laboratory, where the fetal cells are cultured and analyzed for chromosomal defects through a procedure known as karyotyping. At the same time, the level of alpha-fetoprotein (AFP) in the amniotic fluid is measured to determine
the possibility of a neural tube defect.

It generally takes about 2 weeks for results to come in, depending on what is being measured. Some new techniques not yet widely available require only 24 to 48 hours. AFP results are usually available within a few days of the test. Most women are anxious to hear from their physician as soon as possible, especially if they are considering terminating the pregnancy in the event of an abnormality.

**How accurate, reliable, and safe is the test?**

Amniocentesis is associated with a risk of miscarriage in 1 of 200 pregnancies as well as an even smaller risk (minimized with ultrasound guidance) that the needle may puncture the foetus. Other complications, though rare, can include cramping, vaginal bleeding, and the leaking of amniotic fluid. Thus, amniocentesis is normally offered only to women whose risk of having a genetically abnormal fetus is greater than 1 in 200. All women over the age of 35 run such a risk, as do women who know that they and their partner are carriers for genetic defects such as sickle cell anemia, Tay-Sachs disease, one of the thalassemias, or cystic fibrosis, or who have family members with a history of genetic disease. Amniocentesis is not necessary if only one member of a couple is a carrier.

Amniocentesis, even when performed as early as 10 weeks, is very reliable at detecting conditions such as Down’s syndrome which result from chromosomal abnormalities; but other malformations can occur during foetal development owing to non-genetic causes, and these would not be picked up by amniocentesis.
Evaluation of patient with stroke

Rohini Bhan & Arvind Bhan

Stroke is a major public health problem. It is the third leading cause of death and the leading cause of serious adult disability in the United States. It has an incidence rate of about 600,000 cases per year (including new and recurrent stroke), and a total mortality rate of about 158,000 per year. A consistent decline in stroke death rate has been occurring over the years without a significant decline in stroke incidence. This is resulting in an increasing stroke prevalence and an increase in the total number of stroke deaths. Currently, there are about 4 million stroke survivors in the United States. The majority of these survivors live with varying degrees of disabilities and the economic cost of stroke in the USA is estimated to be $43 billion per year. The first step in decreasing the impact of stroke on public health is decreasing stroke incidence rate by identifying stroke risk factors and modifying them. The second step in reducing the impact of stroke on the individual and the public is by developing safe and efficacious therapies for stroke. Until recently, prevention, supportive care and rehabilitation were the only modes of management of the stroke patient. While these approaches remain the mainstay of management, new therapeutic approaches, such as thrombolysis and neuroprotection, are being explored.

Risk Factors for Stroke

Identifying risk factors for stroke is important in stroke prevention and understanding its pathophysiology. Risk factors are usually divided into those that are modifiable and those that are non-modifiable.

Non-modifiable Risk Factors

1. **Age.** Age is the single most important risk factor for stroke. The majority of stroke patients are over the age of 65 years. Only 28% of stroke patients are under the age of 65 years. In the younger population, stroke occurs in newborn infants who suffer from asphyxia or brain hemorrhage, in young adults with valvular heart disease who develop embolic strokes, and in drug abusers who are at risk of developing embolic or hemorrhagic strokes. The incidence of stroke doubles each decade after the age of 55 years, and it is estimated that 40% of people over the age of 80 years suffer from multi-infarct dementia. As the population ages the prevalence of stroke will continue to increase, unless stroke incidence rates are decreased by effective stroke prevention.

2. **Gender.** Stroke occurs more commonly in men than in women. In general, the incidence of stroke is about 20% greater in men than in women; under the age of 65 years the incidence is about 30% greater in men. Although stroke incidence rate is greater in men, stroke deaths are
higher in women due to longer life expectancy and higher prevalence rate.

3. **Race.** Stroke incidence and mortality rates are about 2.5 times higher among African-Americans than among whites. Hispanics and native Americans have lower stroke death rates than whites in the age group 65 years and over. The reasons for these differences are not clear, although the prevalence of modifiable risk factors among these groups could account for some of these variations.

4. **Heredity.** The contribution of heredity as a risk factor of stroke is complicated by the presence of multiple risk factors in the same family. However, it seems that the risk of stroke is greater for people who have a family history of stroke, even after adjusting for other risk factors.

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**Modifiable Risk Factors**

1. **High blood pressure.** High blood pressure is one of the most important modifiable risk factors for stroke. It is the most common and potent precursor of thrombotic ischemic stroke. The increased risk of developing a stroke is proportional to the degree of increase in blood pressure. This risk can be 7 times higher in hypertensive patients compared to normotensive patients. The wide prevalence of hypertension (about 50 million in the United States) and the presence of effective therapy for it makes it an ideal target for therapy and stroke prevention.

2. **Carotid stenosis, transient ischemic attack (TIA), and previous stroke.** Carotid stenosis, TIA, and previous stroke are risk factors for the development of ischemic stroke, in a manner proportional to the degree of carotid stenosis and the severity of TIA or previous stroke. Antiplatelet therapy and carotid endarterectomy are effective in reducing the risk of new or recurrent stroke, although carotid endarterectomy is superior in patients who have symptomatic or asymptomatic carotid stenosis in excess of 60% and whose general health make them good candidates for elective surgery.

3. **Cardiac disease.** Coronary artery disease increases the risk of stroke about threefold, congestive heart failure by about 4 times, and atrial fibrillation by about 5 times. The coexistence of other cardiac disease can significantly increase the risk of stroke. In particular the association of valvular heart disease with atrial fibrillation can increase the risk of stroke by about 20 times. Mitral annular calcification increases the risk of stroke by about 2 times while uncomplicated mitral valve prolapse does not significantly increase this risk. Antithrombotic, but not antiplatelet, therapy reduces the risk of stroke in patients with atrial fibrillation. Results of the European Atrial Fibrillation Trial Study Group indicate that to achieve optimal levels of anticoagulation with the lowest
risk in patients with atrial fibrillation and a recent episode of cerebral ischemia, the target value for the International Normalized Ratio (INR), a standardized prothrombin time ratio, should be set at 3.0, and values below 2.0 and above 5.0 should be avoided.28

4. **Cigarette smoking.** Cigarette smoking increases the risk of stroke in a dose-dependent fashion, and the risk is slightly greater for women than men. Regardless of the starting age and the number of cigarettes smoked per day, cessation of smoking reduces this risk to levels comparable with non-smokers within 2-5 years.

5. **Diabetes mellitus.** Diabetes mellitus increases the risk of atherosclerotic and microangiopathic vascular disease resulting in an increased risk of large-vessel thrombotic strokes and small-vessel lacunar infarcts. Blood glucose levels greater than 150 mg/dl are associated with increased risk of stroke by about 2 times.

6. **Dyslipidemia.** Increased levels of blood cholesterol and low density lipoprotein and low levels of high density lipoprotein accelerate atherosclerosis. Hypercholesterolemia increases the risk of ischemic stroke in a progressive fashion. Lowering the level of cholesterol is effective in reducing the risk of stroke.

7. **Hypercoagulable states.** Hypercoagulable states, such as protein S and C deficiency, cancer, pregnancy, sickle cell anaemia and increased hematocrit may increase the risk of stroke. The stroke Prevention in Sickle Cell Disease (STOP) trial is aimed at determining the value of chronic blood transfusion in reducing the incidence of stroke in high risk children.

**Evaluation of the Patient with Acute Ischemic Stroke**

Thrombolytic therapy for acute ischemic stroke was recently approved by the United States Food and Drug Administration (US FDA). Current evidence indicates that in order to be safe and efficacious this therapy must be administered within three hours from the onset of stroke. This time limit is introducing new aspects and challenges into patient's evaluation and management.

The appellation of stroke is being changed into **brain attack** in order to enhance public awareness for the need to activate the Emergency Medical Services System (EMS) when symptoms suggestive of stroke are noted. These symptoms include:

- a sudden onset of one-sided weakness
- a sudden onset of one-sided numbness
- a sudden decrease in the level of consciousness
- a sudden onset of severe (cataclysmic) headache
- A sudden onset of difficulty in speaking
- A sudden onset of visual abnormalities such as loss of vision or parts of the visual field or the development of double vision
- A sudden onset of imbalance (ataxia), which may be associated with nausea and vomiting.

The Cincinnati Prehospital Stroke Scale has been developed to aid health personnel in identifying stroke patients. Three major neurological signs are sought: facial droop, arm drift and speech abnormalities.

- **Facial droop**: The patient is asked to smile or show teeth. Facial symmetry is considered normal and asymmetry abnormal.
- **Arm drift**: The patient is asked to close their eyes and hold out their arms. Equal arm movements or lack of movements is considered normal. One arm drift is considered abnormal.
- **Speech abnormalities**: In the West, the patient is asked to repeat a sentence "you can't teach an old dog new tricks." Ability to do so correctly is considered normal. Slurred, inappropriate words or inability to speak is considered abnormal.

In the USA, stroke teams, stroke codes and protocols, are being developed to provide standardized emergency evaluation and treatment of the stroke patient. The emergency evaluation consists of immediate general assessment followed by an emergency neurological assessment.

The immediate general assessment includes assessment of vital signs, establishing intravenous access and obtaining baseline laboratory tests (complete blood count, coagulation studies, electrolytes and blood glucose level), and alerting the stroke team. Ideally, the immediate general assessment should be completed within 10 minutes of arrival in the Emergency Department.

The emergency neurological assessment includes reviewing the patient's history, with particular attention to the time of onset of symptoms; performing a general physical examination including auscultation for heart murmurs and carotid bruits; an electrocardiogram (ECG); a neurological examination, preferably, by a neurologist, utilizing the National Institute of Health Stroke Scale (NIHSS); and a non-contrast CT scan of the head. Ideally this neurological assessment, including the CT scan, should be performed within 25 minutes, and the CT scan should be read within 45 minutes of arrival in the Emergency/Casualty Department. If there is a history of trauma or it is suspected in a comatose patient, a lateral cervical spine x-ray should be performed to rule out cervical spine injury. If subarachnoid hemorrhage is strongly suspected clinically but not detected by CT scan, a lumbar puncture should be performed. The aim of this neurological assessment is to determine the type, location, severity, and the differential diagnosis of stroke.

**Type of Stroke**

Strokes are generally divided into ischemic and hemorrhagic. Ischemic
strokes are caused by the occlusion of a blood vessel and hemorrhagic strokes are caused by the rupture of a blood vessel. The majority of strokes are ischemic and the majority of these ischemic strokes are large-vessel thrombotic strokes; the remainder are embolic and small-vessel lacunar infarcts. Hemorrhagic strokes can be due to intracerebral hemorrhage or subarachnoid hemorrhage (SAH). The most common cause of intracerebral hemorrhage is hypertension, and a small number are due to vasculitis and angiopathy. The most common cause of SAH is a cerebral aneurysm and a smaller number are due to arteriovenous malformation.

Patients with hemorrhagic strokes are usually more seriously ill and deteriorate more rapidly than patients with ischemic strokes. They are more likely to have decreased level of consciousness, headache, nausea and vomiting.

Patients with SAH usually present with a headache that they describe as the worst headache they have ever had. The headache usually occurs following an intense physical or emotional activity and reaches maximal severity within seconds. It may radiate to the neck or face, and may be associated with nuchal rigidity that may take several hours to develop. It can be the only manifestation or it can be associated with other focal or generalized signs such as mental obtundation, seizure, nausea, vomiting, photophobia, or cardiac arrhythmias. In about 15-25% of patients, SAH is heralded by a warning leak, 2 to 3 weeks prior to the major episode. The symptoms and signs of a warning leak are similar to those of a SAH but are milder.

Patients with ischemic strokes usually present mainly with focal signs depending on the location of the involved vessel. Also, on the basis of the duration and reversibility of symptoms, ischemic stroke has been traditionally classified into transient ischemic attack (TIA), reversible ischemic neurological disorder (RIND), or Stroke. Symptoms of TIAs are usually resolved within minutes and at most within 24 hours, symptoms of RINDs are usually resolved within 3 days, and symptoms of stroke only improve over a longer period of time, if at all. This temporal classification of stroke is becoming less important; the emphasis is rather being placed on rapid differentiation between ischemic and hemorrhagic stroke and the selection of patients with ischemic strokes who are eligible to receive thrombolytic therapy.

**Location of Stroke**

Focal signs of stroke depend on the location of the involved artery. In general, unilateral paralysis and numbness, language disturbance, monocular blindness, and visual field disturbances are signs of an anterior circulation stroke involving the carotid artery and its branches. Vertigo, ataxia, dysarthria, diplopia, bilateral visual disturbances, paralysis and numbness in one or all of the extremities are signs of a posterior circulation stroke involving the vertebrobasilar artery and its branches. The paralysis of posterior circulation stroke can manifest as a drop attack, which is a sudden onset of paralysis of all four limbs without loss of consciousness.
Severity of Stroke

Assessing the severity of stroke depends on neurological and radiological examination of the patient. Certain neurological scales have been developed as a means of a rapid, standardized assessment of the neurological impairment of the patient.

The National Institute of Health Stroke Scale (NIHSS): The NIHSS evaluates five major aspects of the neurological function of the patient that have been found to correlate with stroke severity and outcome. The aspects that are evaluated are: the level of consciousness, visual function, motor function, sensory function and neglect, and cerebellar function. The total score on this scale ranges from 0 (normal) to 42 (worst condition). The scale has been used to guide decision-making about the use of thrombolytic therapy in patients with ischemic stroke. Patients with an NIHSS score less than 4 have very little deficit and are generally not considered for thrombolytic therapy since the benefit in this situation may be minimal. Exceptions to this are patients with an NIHSS score less than 4 due to isolated severe injury, such as patients with severe aphasia (NIHSS=3) or patients with heminaposia (NIHSS=2 or 3). Patients with NIHSS scores greater than 22 are considered to have severe stroke and may be excluded from receiving thrombolytic therapy since the risk of cerebral hemorrhage in these situation may outweigh the potential benefit. The ultimate decision about the administration of thrombolytic therapy, however, should be considered on an individual basis.

Glasgow Coma Scale (GCS): The GCS evaluates the level of consciousness of the patient by measuring 3 neurological functions: eye-opening, verbal response, and motor response. The total score ranges from 3 (profound coma, no responses) to 15 (normal). A patient with a score of 8 or less is considered to have severe coma and a poor prognosis. Early stupor or coma occurring with a stroke indicates a massive hemispheric stroke, usually hemorrhagic, or a brain stem infarct, usually due to basilar artery occlusion. Metabolic or pharmacologic causes of coma should be considered in the differential diagnosis of these patients. Decreased level of consciousness occurring within hours of the stroke indicates increased intracranial pressure (ICP) due to intracerebral hemorrhage or brain edema and is related to the severity of the infarct. These obtunded patients are at increased risk of aspiration pneumonia and tracheal intubation is generally indicated for patients with a GCS score of 8 or less.

The Hunt and Hess Scale (HHS) for Subarachnoid Hemorrhage: The HHS evaluates a variety of neurological signs, such as headache, nuchal rigidity, drowsiness, hemiparesis, coma and decerebrate rigidity, as a measure of the severity of neurological deficit due to SAH. It has five grades from 1 (asymptomatic) to 5 (comatose, decerebrate patient). The HHS grade correlates with survival and complication rates, and is used to guide the timing of aneurysm clipping.
**Differential Diagnosis of Stroke**

Few conditions, other than ischemic or hemorrhagic stroke, can cause a sudden onset of focal neurological deficit, with or without accompanying symptoms. Hypoglycemia can cause focal neurological deficit with or without a decreased level of consciousness. The postictal phase of seizures can be associated with focal neurological deficit, such as aphasia or paresis (Todd's palsy), that can last for several hours, with or without decreased level of consciousness. Seizures complicate SAH in about 15% of cases, embolic strokes in about 10% of cases, and thrombotic strokes in less than 5% of cases. Abnormal posturing and rigidity at the onset of stroke can be misidentified as seizure. Other conditions that can cause sudden onset of focal neurological deficits with varying degrees of decreased level of consciousness include trauma, meningitis, encephalitis, hypertensive encephalopathy, tumor, subdural or epidural hematoma, migraine, and drug overdose. Diagnostic testing supplements the history and physical examination in the differential diagnosis.

**Computed Tomography (CT):** A noncontrast head CT is the most important diagnostic test in the emergency evaluation of the stroke patient. It is a necessary test to identify patients with intracranial hemorrhage and patients with early CT changes of massive ischemic infarcts, two groups of patients in whom thrombolytic therapy is contraindicated. CT scanning is reliable in detecting almost all intracranial hematomas that exceed 1 cm in diameter and in detecting subarachnoid hemorrhage in more than 95% of cases. Hematoma appears as a hyperdense region, and SAH appears as a hyperdense lining on the surface of the brain and blood vessels. Hyperdensity in the basal cistern is particularly suggestive of ruptured aneurysm. Patients presenting with a clinical presentation highly suggestive of SAH and having a normal CT scan should undergo lumbar puncture. Bloody cerebrospinal fluid with similar red blood cell count in successive tubes indicates SAH. Lumbar puncture excludes subsequent use of thrombolytic therapy and should be performed only in selected patients.

A CT scan done within 3 hours from the onset of ischemic stroke usually reveals no changes; subtle changes may appear between 3-6 hours; within 12 hours approximately half of the patients have some changes; and within 24 hours almost all patients demonstrate changes of ischemic stroke. Early changes appear as blurring of cortical gray-white matter junction and loss of the differentiation between gray and white matter at the basal ganglia. These changes progress later into a hypodense area representing the infarction. Brain edema usually does not develop until after 24 hours of stroke onset. Changes of the scan with brain edema include diffuse swelling of the affected hemisphere, effacement of cerebral sulci, a decrease in the size of subarachnoid cisterns, and a local mass effect with a midline shift and ventricular compression. Large infarcts produce edema more rapidly. Patients who have early changes of infarction or edema on CT scan are excluded from receiving thrombolytic therapy since they are at a high risk of developing...
hemorrhagic transformation of the ischemic stroke.

**Magnetic Resonance Imaging (MRI):**
MRI is not part of the routine evaluation of the stroke patient. MRI is superior to CT scan in visualizing infarcts in the brain-stem and cerebellum and very small lacunar or cortical infarcts but not in detecting intracerebral or subarachnoid hemorrhage. It requires more time to perform and can be the subject of artifacts due to patient motion. Like CT scan, MRI is not reliable in imaging cerebral infarcts within the first 6 hours of the onset of stroke, where therapeutic intervention would have the most safety and efficacy. New MRI modalities are being developed to overcome this limitation.

Diffusion-weighted imaging (DWI) detects ischemic cytotoxic edema and perfusion-weighted imaging (PWI) detects blood flow changes in the microvasculature of the brain. Areas of ischemia detected by PWI can be larger than those detected by DWI in experimental models of ischemia. It is hypothesized that areas detected by PWI and DWI reflect different levels of energy failure (electrical vs. metabolic) in the ischemic area. Combining the two techniques of DWI and PWI can lead to early and accurately localized diagnoses in the hyperacute phase of ischemic stroke.

Magnetic resonance spectroscopy (MRS) detects small signals from protons other than water molecules, such as phosphorus (31p) and non-water hydrogen (1H) which correlate with metabolic changes of ischemia such as increased lactate levels. Unlike DWI and PWI, it does not provide an early diagnosis of stroke, however, it does provide a metabolic diagnosis which is useful in assessing the viability of tissues prior to treatment and assessing the efficacy of therapy afterwards.

Magnetic resonance angiography (MRA) reliably detects occlusions of the major blood vessels of the brain and is, therefore, a useful technique to overcome the limitation of carotid Doppler which is not reliable in differentiating complete occlusion from stenosis in excess of 95%.

Angiography is an invasive technique that reliably demonstrates vascular lesions such as stenosis, occlusion, dissection or rupture, but not the parenchymal changes resulting from such lesions. It is the most definitive technique for diagnosing cerebral aneurysms and arteriovenous malformation and is usually performed in these patients in preparation for surgical repair.

Ultrasonography is a noninvasive technique that is usually performed electively. Carotid Doppler demonstrates carotid artery stenosis in excess of 60% quite accurately, but does not reliably differentiate between complete occlusion and stenosis in excess of 95%. MRA supplements that deficiency. Transthoracic echocardiography is useful in detecting cardiac emboli, and transesophageal echocardiography (TEE) is superior in detecting both cardiac and aortic emboli and the changes due to infective endocarditis.
References

19. European Carotid Surgery Trialists' Collaborative Group: MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. Lancet 1991; 337:1235-43
Stroke implies the ischaemic damage of the brain owing to a blockage in blood flow, or to a haemorrhage of blood vessels in the brain. Without blood, sections of brain tissue quickly deteriorate or die, resulting in paralysis of limbs or organs controlled by the affected brain area. Most strokes are associated with high blood pressure (hypertension), atherosclerosis (development of fatty plaques in artery walls), or both. Some of the signs of major stroke are facial weakness, inability to talk, loss of bladder control, difficulty in breathing and swallowing, and paralysis or weakness, particularly on one side of the body. Stroke is also called cerebral apoplexy and cerebrovascular accident.

I CAUSES

The majority of stroke cases are due to arterial blockage caused by either thrombosis or embolism. Thrombosis involves the clotting of the surface of an atherosclerotic plaque, in a branch of one or more of the four main arteries leading to the brain. As these arteries become narrowed, a potential stroke victim often experiences recurrent warnings, which take the form of transient paralysis (such as in one arm or leg or on one side of the face), or discovers impairments in speech, vision, or other motor functions. At this stage, deposits in the linings of the cerebral arteries can often be treated by surgical removal or bypass of blockages. Anticoagulant drugs, changes in diet, and even daily doses of aspirin are also used. Thrombosis may cause complete occlusion of an artery, leading to permanent brain damage.

Embolism occurs when a cerebral artery suddenly becomes blocked by material—such as clotted blood, air, or fat—coming from another part of the bloodstream. Such masses, known as emboli, often form as clots in a diseased or malfunctioning heart, but can also come from dislodged fragments of atherosclerotic plaque or even an air bubble. Treatment is largely preventive, consisting of monitoring of the diet, and, if possible, use of anticoagulants.

Haemorrhaging of cerebral blood vessels, a less frequent but usually more serious cause of stroke, can occur where aneurysms, or blister-like bulges, develop on the forks of large cerebral arteries on the brain surface. The rupture of aneurysms causes brain damage, owing to the seepage of blood into brain tissue or to the reduced flow of blood to the brain beyond the point of rupture.

II REHABILITATION

Rehabilitation from stroke requires specialized help from neurologists, physiotherapists, and speech therapists—especially during the first six months, when most progress is made. Passive stretching exercises and thermal applications are used to regain motor control of limbs, which
become rigidly flexed after a stroke has occurred. A patient may recover enough to do pulley and bicycle exercises for the arms and legs and, through speech therapy, may regain the language abilities often lost following a stroke; the degree of recovery varies greatly from patient to patient.

The noticeable decline in the incidence of stroke in the developed world since 1950 may be due to the increasing recognition of the leading role of hypertension in stroke, with increased drug treatment and dietary changes such as lower intake of saturated fats and cholesterol. Increased awareness of the dangers of smoking may also be a factor. Nevertheless, stroke remains the third leading cause of death in the Western hemisphere, following coronary artery disease and cancer.
Ensuring Safe Water:
Health and Hygiene education

Imtiyaz Ali

Scope of hygiene education

Studies have shown that the provision of a good drinking-water supply alone is insufficient to ensure health. There are many stages in the collection, storage, and handling of food, the disposal of excreta, and the care of children at which drinking water can become contaminated and the community exposed to pathogens in excreta.

Children, especially those under 5 years of age, are particularly vulnerable to diarrhea. A common belief is that children’s faeces are harmless, whereas in fact they are the main source of infection of other children. Parents may not hygienically dispose of their young children’s faeces, young children may not use latrines, and the yards surrounding homes are often contaminated.

There are many transmission routes for water-related and sanitation-related diseases, and hygiene education can therefore cover a wide range of actions. The most important behaviors from the point of view of health will depend on the community, the disease pattern, and the climate. One of the functions of the initial field inspection and surveillance is to determine which behaviors the hygiene educational program should seek to promote in the community.

<table>
<thead>
<tr>
<th>Table Behaviors to be recommended in hygiene education</th>
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<tbody>
<tr>
<td><strong>Water source:</strong></td>
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<tr>
<td>• All children, women, and men in the community should use safe water sources for drinking purposes and food preparation.</td>
</tr>
<tr>
<td>• Adequate water should be used for hygiene purposes such as bathing, household cleanliness, and clothes washing.</td>
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<tr>
<td>• Water should be efficiently used and not wasted. Wastewater should be properly drained away.</td>
</tr>
<tr>
<td>• Improved water sources should be used hygienically and be well maintained.</td>
</tr>
<tr>
<td>• There should be no risk of contamination of water sources from nearby latrines, wastewater drainage, cattle, or agricultural chemicals.</td>
</tr>
<tr>
<td><strong>Water treatment:</strong></td>
</tr>
<tr>
<td>• Simple purification procedures, e.g. boiling or chlorination, should be carried out on the water source if necessary.</td>
</tr>
<tr>
<td>• If necessary, water should be filtered to remove any solid material, guinea worm, etc.</td>
</tr>
<tr>
<td><strong>Water collection:</strong></td>
</tr>
<tr>
<td>• Drinking water should be collected in clean vessels without coming into contact with hands and other materials.</td>
</tr>
<tr>
<td>• Water should be transported in a covered container.</td>
</tr>
<tr>
<td><strong>Water storage:</strong></td>
</tr>
<tr>
<td>• Water should be stored in vessels that are covered and regularly cleaned.</td>
</tr>
<tr>
<td>• Drinking water should be stored in a separate container from other domestic water wherever possible.</td>
</tr>
</tbody>
</table>
Water drinking:
• Drinking water should be taken from the storage vessel in such a way that hands, cups, or other objects cannot contaminate the water.

Water use:
• Adequate amounts of water should be available and used for personal and domestic hygiene. (It is estimated that a minimum of 30–40 liters per person per day are needed for personal and domestic hygiene.)

Food handling:
• Hands should be washed with soap or ash before food is prepared or eaten.
• Vegetables and fruits should be washed with safe water, and food should be properly covered.
• Utensils used for food preparation and cooking should be washed with safe water as soon as possible after use and left in a clean place.

Excreta disposal:
• All men, women, and children should use latrines at home, at work, and at school.
• The stools of infants and young children should be safely disposed of.
• Household latrines should be sited in such a way that the pit contents cannot enter water sources or the groundwater table.
• Hand-washing facilities and soap or ash should be available, and hands should always be washed after defecation and after helping babies and small children.

Wastewater disposal:
• Household wastewater should be disposed of or reused properly. Measures should be taken to ensure that wastewater is not allowed to create breeding places for mosquitoes and other disease vectors or to contaminate safe water.

Planning hygiene education
Planning hygiene education in a community involves the following steps:

i) Dialogue with the community and local agencies;

ii) Selection of priority hygiene behaviours to be changed based on surveillance data and felt needs within the community;

iii) Analysis of influence on selected behaviors and the implications for hygiene education.

Preparation of an action plan for hygiene education requires answers to the following questions:
• How will community participation be mobilized?
• Who should the education be directed at (target group)?
• What should the content of the education be?
• Who should carry out the hygiene education?
• What educational methods should be used?
• What support should be provided by the surveillance agency?

Community participation and empowerment
Hygiene behaviors are particularly difficult to change because they relate to daily activities, the whole community shares them and they form part of the culture and traditions of the community. The improvement of water supply, sanitation, and hygiene should be seen as part of an overall process of community development. It is important, therefore, to work with the whole community and particularly with schoolchildren, and to involve them in all stages of hygiene education, including selecting priority hygiene behaviors, understanding the influences on such behaviors, selecting educational methods, and implementation.

The educational methods used should be those that strengthen and
empower individuals and communities to work for change. There are no set rules for developing a community participation program, but the stages described in Table I are common to many such programs.

The community may already be highly organized and taking action on health issues. If so, only a few visits by surveillance field staff will be needed to introduce the concepts of surveillance and involve the community in the surveillance program. However, more often there will be no well-developed structure, that sections of the community, such as women, are poorly represented, and that there are disagreements or factional conflicts. In this situation, achieving community participation will take more time and require many visits by field staff to bring people together, resolve differences, agree on common aims, and take action.

Even after the community starts to become involved, further visits, possibly over several years, will be needed to provide support and encouragement, and ensure that the structures created continue to operate.

Table 2. Stages in the community participation process

<table>
<thead>
<tr>
<th>Getting to know the community:</th>
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<tbody>
<tr>
<td><em>Learning about the community, its structure and leadership pattern</em></td>
</tr>
<tr>
<td><em>Initial contacts with families, leaders and community groups</em></td>
</tr>
<tr>
<td><em>Dialogue and discussion on concerns and felt needs</em></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Organization building:</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Strengthening of community organization</em></td>
</tr>
<tr>
<td><em>Establishment of new structures, e.g. water committees, women’s groups</em></td>
</tr>
</tbody>
</table>

| Educational activities within community structures |
| Decision-making on priorities |
| Selection of community members for training as water leaders |

**Initial actions:**
- Action by the community on achievable short-term goals that meet felt needs and bring the community together
- Reflection on initial activities
- Setting of priorities for future activities

**Further actions:**
- Activities in which the community takes a greater share of responsibility for decision-making and management

**Selection of behaviors to be changed**

It is better to concentrate on a small number of behaviors than to attempt to influence all the hygiene behaviors. The behaviors chosen should be selected on the basis of probable public health benefit to the community. Some of the questions that will need to be asked in order to determine priorities include the following:

- What is the evidence that the behavior represents a problem in the community?
- Which behavior changes will have the greatest impact on improving health?
- Which hygiene behaviors will be the easiest to change?
- What are the specific requirements of the water supply and sanitation systems that are being promoted in the community?
- What are the felt needs and priorities of the community?

It is best to concentrate on those hygiene practices shown by the surveillance to be a priority for remedial action in the community concerned; these should be the practices, which are likely to be of the greatest benefit to
health. However, greater efforts will be required to change hygiene practices that the community does not see as important or that conflict with its culture and traditions.

**Factors influencing hygiene behavior and selection of content of education**

Hygiene education programs should be based on an understanding of the factors that influence behavior at the community level. These might include:

i) Enabling factors such as money, materials, and time to carry out the behavior;

ii) Pressure from particular members of the family and community, e.g. elders, traditional healers, opinion leaders;

iii) Beliefs and attitudes among community members with respect to the hygiene behaviour, and especially the perceived benefits and disadvantages of taking action, and the understanding of the relationship between health and hygiene.

An understanding of the factors that influence hygiene behaviours will help in identifying the resources (e.g. soap, storage containers), the key individuals in the home and community, and the important beliefs that should be taken into account. This will help to ensure that the content of the hygiene education is relevant to the community. Good advice should:

- Result in improved health
- Be affordable
- Require a minimum of effort and time to put into practice
- Be realistic
- Be culturally acceptable
- Meet a felt need
- Be easy to understand.

One of the most important characteristics of effective health education is that it builds on concepts, ideas, and practices that people already have. Most communities already have beliefs about cleanliness, diarrhea, and hygiene. In the short term, it may not be necessary to convince people of the correctness of the germ theory of disease in order to get them to use latrines and practice good hygiene. This is a long-term objective that is best-achieved in schools. It is possible to find supporting ideas in many traditional belief systems, and to appeal, for example, to the desire for comfort and privacy.

**Selection of target groups**

Hygiene education is aimed at two kinds of target group:

- **Primary target group** — the members of the household, children, women, men, grandparents, and others who care for children.

- **Secondary target group** — people who need to be involved in the program because of the influence that they have in the community (local leaders, field staff from other agencies, politicians, traditional healers, etc.).

A single hygiene education message and the associated materials are unlikely to be sufficient for all purposes. Ideally, the individual needs of each of the target groups in the community should be addressed, taking into account educational level and any cultural constraints.

**Information needs for hygiene education**

Before a formal hygiene education program is begun, it is important to include in the sanitary
survey an assessment of the socio-cultural factors that characterize the community, in order to determine:

- Local beliefs and attitudes regarding water, sanitation, and health;
- Traditional water use and defecation habits and excreta disposal practices;
- Current levels of knowledge about disease transmission, especially among community leaders and other influential individuals;
- The priority given to improvements in water supply and sanitation in relation to other community needs;
- Existing channels of communication in the community including books, newspapers, and magazines, radio or television, traditional drama, songs, and story-telling;
- The members of the community and field workers from other agencies who might be involved in hygiene education activities.

Educational methods

Some key characteristics of effective communication and health education are summarized in Table 3.

The choice of methods to be used should take account of the nature of what is to be conveyed and of local beliefs, values, and practices; the characteristics of the intended audience, including educational and literacy levels and exposure to, and familiarity with, different educational methods; practical considerations, including the amount of money available and the experience of the staff. Effecting the fundamental changes in lifestyle that are required to improve sanitation and hygiene will usually require an intensive program of face-to-face communication in the community. This might include visiting individual householders or working with groups in community or other local settings: women’s groups, mothers’ groups, children in schools, or trade unions. In hygiene education, it is important to emphasize participatory learning methods; these can include small-group teaching, simulations, case studies, group exercises, and role-play. These methods:

- Avoid formal lecture presentations
- Encourage discussion between participants
- Encourage interaction during the sessions
- Use a variety of games, puzzles, and exercises

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Table 3. Characteristics of effective health education

- Promotes actions that are realistic and feasible within the constraints faced by the community
- Builds on ideas and concepts that people already have and on common practices
- Is repeated and reinforced over time using different methods
- Uses existing channel of communication, e.g. songs, drama, and story telling, and can be appropriately adapted to these media
- Is entertaining and attracts the community’s attention
- Uses clear simple language and local expressions, and emphasize the short-term benefits of action
- Provides opportunities for dialogue and discussion to allow learner participation and feedback
- Uses demonstrations to show the benefits of adopting the practices recommended
Use learning aids that stimulate discussion and comments.

Participatory learning methods have a number of advantages. Their active nature means that participants are more likely to remember what they have learned; participants draw from their own experience and are allowed to discover principles for themselves; opportunities are provided for learning problem solving skills; participants acquire the confidence to tackle problems and improve their conditions. However, many field staff will be unfamiliar with participatory learning methods and will require training.

Traditional media such as drama, songs, and story telling are of great potential value and have been used for education in sanitation and hygiene. They combine entertainment with practical advice and can be used to stimulate discussion and community participation. The actors and musicians should be given the basic information on hygiene and health, and allowed to produce a performance that is both entertaining and understood by the community. It can also be valuable to involve members of the community in the performance.

One of the most powerful forms of communication is through real-life examples, e.g. a demonstration latrine can be constructed in a well-chosen location, and correct practices can be demonstrated. Demonstrations are most effective if they can be seen to produce observable benefits in the short term. However, since the health benefits of sanitation and hygiene can take time to become apparent, it is best to emphasize immediate benefits such as convenience, comfort, and freedom from flies and smells. “Satisfied Acceptors”—people who have improved their sanitation or hygiene practices and are pleased with the results, can also communicate valuable messages. They are the best people to explain the benefits to others, as they will use everyday language and will have greater credibility with the community.

A range of learning materials such as flannel graphs, flip charts, leaflets, posters, slide presentations, videos, and models can be developed to support the work. These should be pre-tested on a sample of the intended audience to ensure that their messages are easily understood, and that the advice is relevant and meets the community’s needs. Local artists can be used and encouraged to work with the community in preparing materials.

In general, health education messages should be reinforced by repetition, ideally through more than one medium.

Face-to-face education can be supported by the mass media such as radio, television, and newspapers if the initial survey shows that these will reach the community. Carefully designed and tested radio programs, for example, can be used to spread simple information rapidly to large numbers of people, and to stimulate increasing awareness of, and interest in, the education program. Broadcasts should use a variety of entertaining and interesting formats such as songs, dramas, quizzes, and interviews with members of the community. The timing of such broadcasts should fit in with community activities. Because the mass media reach
large audiences, it is difficult to make messages specific to local communities; it may be useful to prepare radio programs on cassettes, which can be played to small groups or through loudspeakers in public places. A longer-term approach to improving hygiene in the community is working with children in schools. This enables the concepts of hygiene to become part of a general understanding of health and the influence of the environment. Schoolchildren can then introduce hygiene concepts to their parents and siblings. They learn from what they see around them, so that the school environment itself should meet the requirements of good hygiene, for example by providing latrines, water for hand washing, generally clean surroundings, and hygienic facilities for the preparation and serving of school meals.

**Human resources for hygiene education**

For a hygiene education program to be effectively implemented, management staff must be aware of its importance and committed to it in practice. Such staff includes sanitary engineers and specialists in public health medicine, and hygiene education should form part of their professional training.

The effectiveness of hygiene education within surveillance programs will depend on the extent to which local resources can be mobilized to support educational activities. Most hygiene practices are established early in life and reinforced by parents and elders in the family. In particular, mothers play an important role in encouraging hygiene routines in their children and, in most communities, are involved in the organization of the home, the collection and storage of water, cleanliness, and child care. An essential priority in hygiene education is therefore to involve women, by working either with individual women in their homes or with women’s groups within the community. Women should be represented in any community groups that are formed as part of the surveillance program.

The most important resource for hygiene education is the community itself. A search should be made for any groups or organizations in the community that might be involved in hygiene education including village committees, water committees, health committees, young farmers’ clubs, women’s groups, and religious bodies.

Hygiene education is already part of the activities of many members of the community and field agencies, as well as of the staff of clinics and health centers. Community health workers in primary health care programs are key health educators at the village level. Public health inspectors and rural health assistants are heavily involved in hygiene education as part of their promotion of safe water, environmental sanitation, and hygiene. Health workers in hospitals have a health education role as part of the treatment and rehabilitation process.

Outside the health services, those who may become involved in hygiene education include teachers in schools, adult education, and literacy programs. In order to enable them to fulfill this role, the ministry of education or its equivalent should ensure that subject such as the environment, hygiene and
health are included in educational programs, where appropriate.

Table 4. Potential human resources for hygiene education in the community

<table>
<thead>
<tr>
<th>Health services:</th>
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<tbody>
<tr>
<td><strong>Agricultural and development workers:</strong></td>
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<tr>
<td>Doctors and nurses in primary health care</td>
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<tr>
<td>Agricultural extension workers</td>
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<tr>
<td>Midwives</td>
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<tr>
<td>Community development workers</td>
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<tr>
<td>Health visitors</td>
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<tr>
<td>Nutrition program staff</td>
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<tr>
<td>Public health nurses</td>
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<tr>
<td>Cooperative workers</td>
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<tr>
<td>Medical assistants</td>
</tr>
<tr>
<td>Employment-generating program staff</td>
</tr>
<tr>
<td>Nutrition programs</td>
</tr>
<tr>
<td>Women’s program staff</td>
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<tr>
<td>Immunization programs</td>
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<tr>
<td>Special disease programs</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Education services:</strong></th>
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</thead>
<tbody>
<tr>
<td>Village health workers</td>
</tr>
<tr>
<td>Teachers in primary and secondary schools</td>
</tr>
<tr>
<td>Sanitary technicians</td>
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<tr>
<td>Adult education staff</td>
</tr>
<tr>
<td>Veterinarians</td>
</tr>
<tr>
<td>Literacy program staff</td>
</tr>
<tr>
<td>Preschool program staff</td>
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<tr>
<td>Vocational trainers</td>
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</table>

<table>
<thead>
<tr>
<th><strong>Public health services:</strong></th>
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</thead>
<tbody>
<tr>
<td>Public health inspectors</td>
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<tr>
<td>Water supply staff</td>
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<table>
<thead>
<tr>
<th><strong>Informal resources in the community:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sanitary technicians</td>
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<tr>
<td>Elders</td>
</tr>
<tr>
<td>Hygiene inspectors</td>
</tr>
<tr>
<td>Parents</td>
</tr>
<tr>
<td>Refuse management staff</td>
</tr>
<tr>
<td>Traditional birth attendants</td>
</tr>
<tr>
<td>Sanitary engineers</td>
</tr>
<tr>
<td>Traditional healers</td>
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<tr>
<td>Village leaders</td>
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<tr>
<td>Religious leaders</td>
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</table>

Other workers in the community can also be mobilized for hygiene education. Agricultural extension workers who advise communities on growing crops can also provide education on health and nutrition. Community development officers engaged in promoting community organizations and cooperatives can play a key role in promoting community action on health issues.

In addition to government agencies, many voluntary bodies are actively involved in health education, including nutrition groups, family planning associations, and the Red Cross and Red Crescent and many other societies.

When potential resources for hygiene education are being sought, the following questions should be asked:

- Are any groups involved in hygiene education at present?
- How likely is it that they will support hygiene education?
- What support would they need to become involved in hygiene education, e.g. training, learning resources?

Field staff and volunteers may need training in hygiene education, particularly in the newer participatory learning methods. The aim should be to develop self-sustaining programs of hygiene education as part of the normal workload of local fieldworkers in the community. Although initially such fieldworkers may need training, support, and encouragement to undertake hygiene education, with time they should be capable of doing so with minimal external support.
Child Psychology

Babina Bassaud

Child Psychology is the study of children's behaviour—including physical, cognitive, motor, linguistic, perceptual, social, and emotional characteristics—from birth through to adolescence. Child psychologists attempt to explain the similarities and differences among children and to describe normal as well as abnormal behaviour and development. They also develop methods of treating social, emotional, and learning problems and provide therapy privately and in schools, hospitals, and other institutions.

Two critical problems for child psychologists are (1) to determine how environmental variables (such as parental attitudes) and biological characteristics (such as health) interact and influence behaviour, and (2) to understand how behavioural changes influence one another.

1 HISTORY

Both Plato and Aristotle wrote about children. Plato believed that children are born with special talents and that their training should stress those talents. His views are consistent with modern thinking about individual differences and education. Aristotle proposed methods for observing children's behaviour that were forerunners of modern methods. For many centuries thereafter, little interest was shown in the development of children because they were regarded only as miniature adults.

Plato, one of the most famous philosophers of ancient Greece, was the first to use the term "philosophy", which means "love of knowledge". Born around 428 BC, Plato investigated a wide range of topics.

A student of Plato, Aristotle shared his teacher's reverence for human knowledge but revised many of Plato's ideas by emphasizing methods rooted in observation and experience. Aristotle surveyed and systematized nearly all the extant branches of knowledge and provided the first ordered accounts of biology, psychology, physics, and literary theory. Known to medieval intellectuals simply as "the Philosopher", Aristotle is possibly the greatest thinker in Western history, and, historically, had perhaps the single greatest influence on Western intellectual development.
In the 18th century the French philosopher Jean-Jacques Rousseau seemed to echo Plato when he stated that children should be free to express their energies in order to develop their special talents. His view suggests that normal development occurs best in a nonrestrictive, supportive environment. Similar concepts are popular today.

Jean-Jacques Rousseau contributed to many branches of social philosophy. *The Social Contract* is a classic defence of the democratic form of government.

**A Scientific Study**

In the 19th century, Charles Darwin's theory of evolution provided an impetus for the scientific examination of child development. His emphasis on the survival behaviour of different species stimulated an interest in observing children to identify the various ways that they adapt to things, and in learning about the inheritance of human behaviour. These studies were of limited scientific value because they lacked objectivity and often failed to describe adequately the behaviours being observed, making validation impossible.

Scientific research in child development flourished from the early 1900s. One major stimulus was the introduction (1916) by the American psychologist Lewis Terman of the test known today as the Stanford-Binet Intelligence Test. This test led to a number of studies about children's intellectual development. In the 1920s scientists at more than a dozen leading American universities began large-scale observational studies of children and their families; all used the longitudinal method, in which the same children are observed and tested over a specific time period.

The American psychologist Arnold Gesell established a research institute at Yale University in the 1920s for the sole purpose of studying children. He developed the technique of analysing children's behaviour from film, frame by frame. Gesell also made much use of the cross-sectional method, in which different children are observed at each of several age levels.

The accumulated results of all the major studies reported over a period of 20 years provided information about patterns and rates of child development, as well as age norms for a wide variety of behaviours. These norms are used by both professional workers and parents to assess children's development. One problem with the observational studies was that they emerged from an interest in evolution and genetics. Consequently, environmental influences were largely dismissed as unimportant and were excluded from the work on intelligence.
B Environmental Studies

About the time that the observational work was flourishing, other researchers were writing about the role of the environment in children's development and behaviour. Sigmund Freud, who emphasized the effects of environmental variables on development, particularly stressed the importance of parental behaviour during infancy. To this day, Freud's theory continues to influence child psychologists.

Sigmund Freud was responsible for developing theories central to psychoanalysis, the psychology of human sexuality, and interpretation of dreams. Although his theories, published in the late 1800s, were quite controversial during his day, they were more widely accepted later in his life. Perhaps his most important contributions dealt with the connection between aberrant human behaviour and the unconscious mind.

The American psychologist John B. Watson also stressed the role of the environment in shaping children's development. His views were consistent with those of behaviourism, an approach to psychology that had a great impact in the 1950s on research about children.

Although behaviourists emphasize environment, they almost totally deny the influence of biological variables on development. Their basic assumptions are that the mind of a newborn child is a blank slate, or tabula rasa; all behaviours are determined by environmental events; and differences among children are the result of those environmental variables. Behaviourists encouraged experimental studies and were responsible for moving child psychology into the mainstream of psychology. Although they contributed much to the study of children, their concepts eventually were viewed as being too narrow.

In the early 1960s attention was focused on the work of the Swiss psychologist Jean Piaget, who since the 1920s had been writing about children's cognitive development. Piaget called himself a genetic epistemologist—one who studies the origins of human knowledge—and his theories led to more advanced work in child psychology. This work involves both experimental and observational methods and, in accounting for behaviour, integrates biological and environmental variables. Thus, current studies have their origins in Darwin's theory of evolution but also incorporate Watson's concern for the influence of the environment.

II DEVELOPMENTAL THEORIES

A theory of development should reflect an attempt to relate behavioural change to chronological age; that is, diverse behavioural characteristics should be related to specific stages of
growth. The rules governing the transitions between these growth states also must be identified. The dominant developmental theories are Freud's theory of personality development and Piaget's theory of perception and cognition. Both explain human development in terms of interactions of biological determinants and environmental events.

Freud's theory is based on the concept that a healthy personality requires the satisfaction of instinctual needs. In Freudian theory the personality is composed of the id, ego, and superego. The id is the source of instinctual drives. The role of the ego is to cope with the demands of the id while remaining within the rules of society, which in turn are represented by the superego.

The physical focus of instinctual needs changes with age, and the periods of different focus are called stages. Infants, for example, achieve maximum id satisfaction from sucking; this is called the oral stage. Children progress through four stages, ending with adult sexuality. Freud clearly integrated biological and environmental variables in his theory.

Piaget believed that from birth humans are active learners who do not require external incentives. He proposed that cognitive development occurs in four stages:

Stage I, sensorimotor intelligence (birth-2 years), takes the child from unrelated reflexive movements to behaviour that reflects knowledge of simple concepts. Stage II, preoperational thought (2-7 years), is characterized by an increasing use of abstract symbols as reflected in imaginative play.

Stage III, concrete operational thought (7-11 years), involves relatively sophisticated problem-solving behaviour and attainment of adult thought.

Stage IV, formal operational thought (12 years and older), is characterized by the ability to develop hypotheses and deduce new concepts.

III CHILD DEVELOPMENT

The various aspects of child development encompass physical growth, emotional and psychological changes, and social adjustments. A great many determinants influence patterns of development and change.

A Heredity and Environment

It is generally agreed that patterns of child development are determined by the joint interaction of genetics and the environment, although sharp disagreements occur about the relative importance of an individual's genetic makeup. Research on this problem involves the use of separately reared monozygotic (identical) twins. Their behaviours are compared for similarities and differences, and the results are then compared with behaviours of twins reared together.

If genetics is critical, the twins reared apart will be as similar in most
respects as those reared together. (These studies usually assume that when twins are reared apart, their environments are different in important ways, an assumption that is not always true.) Except in instances of massive environmental deprivation, the patterns and rates of physical and motor development appear to be genetically controlled.

Research also indicated that both genetic and environmental variables contribute to intellectual behaviour. A genetic component also exists in personality characteristics such as introversion and extroversion, activity level, and predisposition to psychoses. Many advances have been made in identifying the genetic causes of mental disorders, but more research is needed to understand better how genetic mechanisms operate among normal children.

B Physical Growth

On the average, a newborn baby in the weighs 3 kg (7 lb) and is 53 cm (21 in) long, with the head disproportionately larger than the lower part of the body. As the child grows, increments in height are greatest from birth to three years; thereafter they are relatively constant until adolescence. The growth spurt at adolescence is far less than during infancy. Weight increments are also large during the first three years but are equally large during adolescence. Research shows that growth rates are influenced by the health of the child. Rates of development decelerate during illness; after an illness is cured, however, growth rates accelerate until children attain their appropriate height and weight.

C Motor Activities

Dramatic changes occur in motor skills from birth through the first two years. At birth infants are capable of extensive uncoordinated movements. One feature of the early motor behaviour of infants is the large number of reflexlike actions. These actions appear for a short time after birth and then disappear. For example, when the palm of the hand is stroked lightly the fingers involuntarily close, forming a fist; this is called the palmar reflex.

From these early movements, distinct sequential patterns of motor development occur. Walking, which occurs on the average between 13 and 15 months, emerges from a sequence of 14 earlier stages. Research shows that the rate of acquisition of motor skills is innately determined and that the acquisition of these skills is not influenced by practice. Severe restrictions on motor activities, however, will alter both the pattern and rate of development.

After basic motor skills are acquired, children learn to integrate their movements with perceptual skills, especially spatial perception. This process is critical for the achievement of eye-hand coordination and for the higher-level skills required for many sports activities.
D Language

The ability to communicate and to understand language is one of the main achievements of human beings. An amazing feature of language development is the speed with which it is acquired: The first word is spoken at about 12 months; by two years of age most children have vocabularies of about 270 words, and this increases to 2,600 words at the age of six. It is almost impossible to determine the number of sentence constructions that can be generated within a single language. Children, however, use syntactically correct sentences by the age of three and highly complex constructions by the age of five.

This extraordinary phenomenon cannot be explained by means of simple learning theory. The American linguist Noam Chomsky postulated that the human brain is especially constructed to detect and reproduce language; the mental system does not require formal learning and will function perfectly when language is available to the child. Although developmental psycholinguists do not agree with all of Chomsky's concepts, they do accept the idea of special mental language systems. Today theorists are concerned with the relationship between cognitive growth and language. It is now assumed that language reflects children's concepts and develops as their concepts expand.

Noam Chomsky
American linguist, writer, teacher, and political activist Noam Chomsky is considered the founder of transformational-generative linguistic analysis, which revolutionized the field of linguistics. This system of linguistics treats grammar as a theory of language—that is, Chomsky believes that in addition to the rules of grammar specific to individual languages there are also universal rules common to all languages, and this indicates that the ability to form and understand language is innate to all human beings. Chomsky is also well known for his political activism. He opposed United States involvement in Vietnam in the 1960s and 1970s and has written and lectured extensively on various political and social issues.

E Personality Formation

Theories of personality are attempts to describe how people behave in satisfying their physical and psychological needs. An inability to satisfy such needs creates a personal conflict. Personality formation is viewed as the process by which children learn how to avoid conflict when possible and how to cope with conflict when it inevitably occurs. Overly restrictive or overly permissive parents limit their children's options in avoiding and coping with conflict.

A normal response to overwhelming conflict is to revert to a defence mechanism such as rationalization—for example, denying that one ever had a
specific objective or goal, it being obvious that they had previously had one. Although everyone uses defence mechanisms at some time, they should not become a person's sole means of coping with conflict. A child with a balanced, integrated personality feels accepted and loved and has been allowed to learn a number of appropriate coping mechanisms.

**F Intelligence and Learning**

Intelligence may be defined as the ability to manipulate abstract verbal concepts effectively. This definition is reflected in the types of questions asked on intelligence tests for children. Two well-known tests—the Stanford-Binet and the Wechsler Intelligence Scale for Children, Revised—are used to index children's mental growth and to predict learning performances. Because school learning seems to depend on the ability to reason verbally, the content of intelligence tests seems appropriate; some relationship does indeed exist between intelligence-test performance and school achievement. Predictions based on tests are imperfect, however, because intelligence tests do not measure motivation and because knowledge about the skills needed for school learning is incomplete. It has also been argued that intelligence tests are sometimes inappropriate when used with children from ethnic minorities, who may not understand or respond appropriately to certain items because of language difficulties or cultural differences. Thus, test scores must be interpreted with great care, as with all forms of psychological testing.

**G Family Relationships**

The attitudes and values of parents and their behaviour towards their children clearly influence patterns of development. Likewise, children's characteristics influence parental attitudes and behaviours; disabled children, for example, require more attention and are likely to cause more parental anxiety than do children without disabilities.

Extensive studies have established that parental behaviour towards children varies widely—ranging from restrictiveness to permissiveness, warmth to hostility, and anxious involvement to calm detachment. These variations in attitudes produce different patterns in family relationships. Parental hostility and permissiveness, for example, are associated with highly aggressive, non-compliant children. Warm, restrictive behaviour by parents is associated with dependent, polite, and obedient children. Punishment techniques also influence behaviour. For example, parents who often use physical punishment tend to have children who rank above average in their use of physical aggression. It appears, then, that one of the ways children acquire patterns of behaviour is by imitating their parents.

**H Social Relationships**

Social relationships among infants involve mutual interest without interaction. This is known as parallel play. Beginning with the years before
school, relationships among children the same age and roughly equal status—the peer group—become increasingly sophisticated social systems influencing their values and behaviour. The transition to the adult social world is aided by the organization of peer groups with a leader, members with varying strengths and weaknesses, and a recognition of the need for cooperative behaviour. Peer-group conformity reaches a peak when children are about 12 years of age. Conformity never disappears, but its manifestations among adults are less obvious.

The members of peer groups change with age. Pre-adolescent groups tend to be homogeneous—that is, members are usually of the same sex and come from the same area. Among older children, social relationships are more likely to be based on shared interests and values.

I Socialization

The process by which children learn acceptable and unacceptable behaviour is called socialization. Children are expected to learn, for example, that extreme physical aggression, stealing, and cheating are unacceptable, and that cooperation, honesty, and sharing are acceptable. Some theories suggest that socialization is achieved only through imitation or through a process of rewards and punishments. Current theories, however, stress the role of cognition, or perceiving, thinking, and knowing; thus, mature socialization requires that a person explicitly or implicitly understand the rules of social behaviour that function in all situations.

Socialization also includes understanding concepts of morality. The American psychologist Lawrence Kohlberg has demonstrated that moral thinking exists on three levels. At the lowest level, a rule is obeyed in order to avoid punishment. This level characterizes the thought of very young children. At the highest level, a person has a rational understanding of universal moral principles necessary for society's survival. Of course, the understanding of such concepts is often inconsistent with behaviour and research has shown that moral behaviour varies with each situation and is not predictable.

IV CURRENT TRENDS

Child psychologists continue to be interested in the interaction of biological traits and environmental events that influence behaviour and development; in the role of cognition in socialization, especially in adapting to sex roles; and in understanding the processes of cognition. Psychologists now generally agree that biological risk factors—such as low birth weight, oxygen deprivation before or during birth, and physical and psychological handicaps—are important in behaviour and development. Extensive longitudinal studies are under way to determine how risk factors affect children's experiences, and how differences in these experiences affect their behaviour. This research will provide methods for helping children with risk factors to develop more successfully.
The role of cognition in children's sex-role learning and stereotypical thinking is also being examined. Although a few general sex differences have been established—for example, girls often excel in verbal ability, and boys often excel in mathematical ability—it is not clear how innate traits and environmental events interact to produce these differences. Sex roles have long been rigidly defined in society, but cultural pressures are slowly breaking down these stereotypes so that members of each sex can more easily change or adapt behaviours to fit specific situations.

Much current work involves identifying the cognitive components (such as memory and attention span) used in problem-solving activities. Researchers in developmental psychology are also trying to identify the processes that occur in the transition from one level of thought to the next. Another area of investigation is the cognitive components in reading and arithmetic. It is hoped that this research will lead to improved methods of teaching academic skills and more effective remedial teaching.
A leaf from history of medicine:

**Edward Jenner**

Jenner, Edward (1749-1823), was a British doctor, who discovered the vaccine that is used against smallpox and who laid the groundwork for the science of immunology.

Born on May 17, 1749, in Berkeley, Gloucestershire, in a rural vicarage, Jenner became a keen observer of nature at an early age. After nine years as a surgeon's apprentice, he went to London to study anatomy and surgery under the prominent surgeon John Hunter, then returned to Berkeley to start a country practice that lasted the rest of his life.

Smallpox, a major cause of death in the 18th century, was treated in Jenner's time by the often-fatal procedure of inoculating healthy people with pustule substances from those who had mild cases of the disease. Jenner observed, among his patients, that those who had been exposed to the much milder disease cowpox were completely resistant to these inoculations. In 1796 he inoculated an eight-year-old boy with cowpox virus; six weeks after the boy's reaction Jenner reinoculated him with smallpox virus, finding the result negative. By 1798, having added similarly successful cases, Jenner wrote *An Inquiry into the Causes and Effects of the Variolae Vaccinae, a Disease Known by the Name of Cow Pox*, a tract in which he also introduced the term *virus*.

Jenner encountered some public resistance and professional chicanery in publicizing his findings, and he experienced difficulties in obtaining and preserving cowpox vaccine. Nevertheless, his procedure was soon accepted, and mortality due to smallpox plunged. The procedure quickly spread through Europe and to North America. Three-quarters of a century later, the French chemist Louis Pasteur, drawing on Jenner's work, set a course for the science of immunology and the discovery of modern preventive vaccines. Jenner died in Berkeley on January 26, 1823.
Edward Jenner’s discovery that cowpox was an effective vaccine against smallpox was initially greeted with skepticism. The cartoon satirizes Jenner who is shown injecting patients with cowpox virus resulting in their transformation into cows.

In 1967 the World Health Organization (WHO) launched a worldwide vaccination campaign against smallpox; at the time, some 10 to 15 million cases of the disease occurred each year, with more than 2 million deaths. By mid-1975, when all India was declared free of smallpox, only a few cases were left in two countries, Bangladesh and Ethiopia. In 1979, after two years without a reported case of smallpox, the WHO marked the disappearance of smallpox from the Earth. It recommended that countries stop vaccinating against the disease and that laboratory stocks of the virus be destroyed. Underlining the importance of this last request was the death of an English woman in 1979 of smallpox contracted from a laboratory working with the virus. Currently, stocks of smallpox virus exist only in the United States, Great Britain, Russia, and China.
Replacement fluids: characteristics

Some important considerations:

(World Health Organization)

Doctors should

1) Check that their own knowledge of crystalloids and colloids is complete and that they are familiar with their characteristics and uses. Sometimes the doctor may not be well-versed with the fluid he is prescribing or administering.

2) Organize a teaching session for relevant staff to refresh their understanding of the use and effects of replacement fluids. The nursing and auxiliary staff may have practical knowledge of administration but don’t recognize the ingredient of the fluid they are administering.

3) Make a list of the replacement fluids that are available in their hospital.

4) Determine if any alternative or additional replacement fluids should be available in your hospital? They can make requisitions through the appropriate authorities.
5) See if the supply of replacement fluids is inappropriate, inadequate or irregular? Most of the times, they will be insufficient for the centre; if sufficient, they may be inappropriate, as providing 5% dextrose instead of normal saline or dextrose saline for rehydration during diarrhoea season.

Practitioners can definitely influence the policy on replacement fluids used in various hospitals. With their senior colleagues they can develop a plan to work towards the wider provision and effective use of those replacement fluids that they consider to be essential.

6) See if there any written guidelines on the use of replacement fluids in your hospital? If yes, are they used appropriately and consistently? If no guidelines currently exist, they should develop some guidelines in conjunction with their senior colleagues. They should organize a teaching session for relevant staff and monitor the implementation of the guidelines.

Crystalloidal solutions

**NORMAL SALINE (Sodium chloride 0.9%)**

Normal saline consists of an isotonic solution of sodium chloride in a near-physiological concentration (ie 154 mmol/L each of Na⁺ and Cl⁻)

- **Unit of issue:** 500 ml or 1000 ml bags
- **Infection risk:** Nil
- **Storage:** In a cool place
- **Dosage:** At least 3 times the blood volume lost
- **Plasma half-life:** Short, approximately 45 minutes: rapidly distributed throughout extracellular compartment.

**Indications:** Replacement of blood volume and other extracellular fluid losses.

**Precautions:**
1) Caution in situations where local oedema may aggravate pathology: e.g. head injury
2) May precipitate fluid overload and heart failure

**Contraindications:**
Do not use in patients with established renal failure

**Side-effects:** Tissue oedema can develop when large volumes are used

**BALANCED SALT SOLUTIONS**

These solutions have a composition that more closely resembles extracellular fluid and can therefore be infused in large volumes without disturbing the electrolyte balance

[**Examples** Ringer’s lactate, Hartmann’s solution]
**Unit of issue:** 500 ml or 1000 ml bags  
**Infection risk:** Nil  
**Storage:** In a cool place  
**Dosage:** At least 3 times the blood volume lost  
**Plasma half-life:** Short, approximately 45 minutes: rapidly distributed throughout extracellular compartment

**Indications:** Replacement of blood volume and other extracellular fluid losses

**Precautions:**
1) Caution in situations where local oedema may aggravate pathology: e.g. head injury  
2) May precipitate fluid overload and heart failure

**Contraindications:** Do not use in patients with established renal failure

**Side-effects:** Tissue oedema can develop when large volumes are used

**DEXTROSE AND ELECTROLYTE SOLUTIONS**

Dextrose solutions containing some electrolytes are dextrose saline solutions. As the concentration of sodium is decreased, an increasing amount of fluid will cross into cells. Examples:
- 4.3% dextrose in sodium chloride 0.18%
- 2.5% dextrose in sodium chloride 0.45%
- 2.5% dextrose in half-strength Darrow’s solution

**Plasma half-life:** Very short: rapidly distributed throughout extracellular and intracellular compartment

**Indications:** Generally used for maintenance fluids, but those containing a higher concentration of sodium can, if necessary, be used as replacement fluids

2.5% dextrose in half-strength Darrow’s solution is commonly used to correct dehydration and electrolyte disturbances in children with gastroenteritis. Several products are manufactured for this use. *Not all are suitable.* Ensure that the preparation you use contains:
- Dextrose 2.5%
- Sodium 60 mmol/L
- Potassium 17 mmol/L
- Chloride 52 mmol/L
- Lactate 25 mmol/L
Plasma-derived (natural) colloid solutions

Plasma-derived colloids are all prepared from donated blood or plasma. They include:

- Plasma
- Fresh frozen plasma
- Liquid plasma
- Freeze-dried plasma
- Albumin

These products should not be used simply as replacement fluids. They can carry a similar risk of transmitting infection, such as HIV and hepatitis, as whole blood. They are also generally more expensive than crystalloid or synthetic colloid fluids.

Synthetic colloid solutions

**GELATINES** (Haemaccel, Gelofusine)

Gelatines consist of molecular chains of gelatine prepared from bovine collagen with an average molecular weight of 30,000

- *Haemaccel*: 3.5% gelatine in sodium chloride 0.9%
- *Gelofusine*: 4% gelatine in sodium chloride 0.9%

**Unit of issue**: 500 ml bags

**Infection risk**: None known at present

**Storage**: At room temperature below 25°C: stable for 5 years

**Dosage**: No known dose limit

**Colloid osmotic pressure**:

- *Haemaccel*: approximately 27 mmHg. Expansion of plasma volume equals the volume infused
- *Gelofusine*: approximately 34 mmHg. Expansion of plasma volume exceeds the volume infused

**Plasma half-life**: Approximately 4 hours: short duration of action, although longer than crystalloids.

**Elimination**: Renal excretion

**Indications**: Replacement of blood volume

**Precautions**: May precipitate heart failure

**Caution**

1) Renal insufficiency
2) Do not mix Haemaccel with citrated blood because of its high calcium concentration

**Contraindications:** Do not use in patients with established renal failure

**Side-effects:**
1) Minor allergic reactions due to histamine release
2) Transient increases in bleeding time may occur
3) Hypersensitivity reactions may occur including, rarely, severe anaphylactic reactions

**DEXTRAN 60 AND DEXTRAN 70**

Dextran consist of macromolecular glucose chains with an average molecular weight of 70,000.

3% dextran 60 in sodium chloride 0.9%
6% dextran 70 in sodium chloride 0.9%
6% dextran 70 in 5% dextrose

**Unit of issue:** 500 ml bottles or bags

**Infection:** risk Nil

**Storage:** At room temperature not exceeding 2°C

**Dosage:**
1) Dextran 60: should not exceed 50 ml/kg body weight in 24 hours
2) Dextran 70: should not exceed 25 ml/kg body weight in 24 hours

**Colloid osmotic:** Approximately 58 mmHg. Expansion of plasma volume exceeds the volume pressure infused.

**Plasma half-life:** Approximately 12 hours

**Elimination:** Predominantly renal excretion

**Indications:**
1) Replacement of blood volume
2) Prophylaxis of post-operative venous thrombosis

**Precautions:**
1) Coagulation defects may occur
2) Platelet aggregation inhibited
3) Some preparations may interfere with compatibility testing of blood.

**Contraindications:** Do not use in patients with pre-existing disorders of haemostasis and coagulation

**Side-effects:**
1) Minor allergic reactions
2) Transient increases in bleeding time may occur
3) Hypersensitivity reactions may occur including, rarely, severe anaphylactic reactions. These can be prevented with injection of 20 ml of Dextran 1 immediately before infusion, where available

DEXTRAN 40 AND DEXTRAN 110 are not recommended as replacement fluids

**HYDROXYETHYL STARCH** (Hetastarch or HES)

Hydroxymethyl starch consists of macromolecules manufactured from natural starch, with a range of molecular weights: e.g. 450 000. The commercially available formulation is 6% hetastarch in sodium chloride 0.9%.

**Unit of issue:** 500 ml bags

**Infection risk:** Nil

**Storage:** In a cool place

**Dosage:** Should not usually exceed 20 ml/kg body weight in 24 hours

**Colloid osmotic:** Approximately 28 mmHg. Expansion of plasma volume slightly exceeds the pressure volume infused.

**Plasma half-life:** 12–24 hours

**Elimination:** Predominantly renal excretion

**Precautions:**
1) Coagulation defects may occur
2) May precipitate fluid overload and heart failure

**Indications:** Replacement of blood volume

**Contraindications:**
1) Do not use in patients with pre-existing disorders of haemostasis and coagulation
2) Do not use in patients with established renal failure.

**Side-effects:**
1) Minor allergic reactions due to histamine release
2) Transient increases in bleeding time may occur
3) Hypersensitivity reactions may occur including, rarely, severe anaphylactic reactions
4) Serum amylase level may rise (not significant)

Hydroxymethyl starch is retained in cells of the reticuloendothelial system; the long-term effects of this are unknown.

**PENTASTARCH**

Pentastarch is similar to hetastarch, but comprises a 10% solution with an average molecular weight of 280 000.
Half-life is shorter: 10 hours
Onotic pressure is approximately 40 mmHg; expansion of plasma volume therefore exceeds volume infused.

SYNTHEtic Blood Products

Two products, stroma-free haemoglobin and perfluorocarbons, are currently under investigation. They are colloids and have the advantage of being able to carry oxygen. Neither is available for clinical use at present.