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CHAPTER 5

Organ Systems and Disease

Clinical syndromes can be determined by the type of infecting organism or the site of the infection:

(a) The clinical picture can be characteristic of the infecting organism. Exotoxin producing organisms are those that present with a clinical picture dictated by the organism. Tetanus is a disease presenting with muscle spasms and convulsions due to the effects of tetanospasmin on the nervous system. Botulism is characterized by paralysis, a direct result of the ingestion of botulinum toxin. The pattern of the disease can be indicative of the causative organism, e.g. diphtheria is a clinical diagnosis only later confirmed by the laboratory.

(b) Infection can be diagnosed on the basis of organ dysfunction, e.g. meningitis, gastro-enteritis, salpingitis, cystitis. Infection of an organ system by any one of a multitude of organisms can give a similar clinical picture. Each organ system has only a limited range of responses, e.g. cystitis manifests as an irritable bladder — the motor activity of the bladder is increased resulting in frequency; the sensory component registers an altered urine — burning on micturition is therefore common in cystitis. Frequency, urgency and burning are diagnostic of cystitis. The organism responsible for the cystitis could be an *E. coli*, a proteus, a klebsiella, a *Strept. faecalis* or many other bacteria. Regardless of the species or even if more than one species of bacterium is responsible, the clinical picture and diagnosis remains the same. The diagnosis of cystitis depends on the bladder's expression of dysfunction, not on the type of infecting organism. Pneumonia is lung dysfunction — this is clinically demonstrated by dyspnoea, chest pain and a productive cough. Different bacteria — pneumococcus, klebsiella, staphylococcus and other bacteria causing pneumonia give a similar picture.

All infections do have one sign in common. Fever is usually present when there is an imbalance in the host-parasite equilibrium. The presence of fever is, however, not pathognomonic of infection. Fever may also be present in non-infective conditions, e.g. the collagen diseases, cancer, Hodgkin's disease.

THE RESPIRATORY SYSTEM

Approximately one quarter of the general practitioner's working life is spent treating respiratory diseases. Infection of the respiratory system accounts for about one third of absenteeism from work. The main cause of this absenteeism is attributable to upper respiratory tract infections.
The respiratory tract starts at the nose and passes through the pharynx down to the alveoli. This whole system is adapted to making air and thus oxygen available to the blood, the erythrocytes, and therefore to the whole body. The inhaled air is moistened, warmed and filtered by the respiratory system. Air passing through the nose develops turbulent flow due to the structure of the turbinate bones. Air turbulence brings particles into contact with the mucous coat. The nose has a normal flora. Included in this flora are corynebacteria, staphylococci and streptococci. These organisms when carried by the medical staff constitute a danger in neonatal, burns and surgical units. Nasopharyngeal carriage of pneumococci (30% of people) and *Neisseria meningitidis* (carrier rate increases from 5% to 90% in over-crowded conditions) can represent a serious hazard to hospitalized patients.

The respiratory tract below the level of the epiglottis is normally sterile. At birth the pharynx, trachea and bronchi are sterile; within 24 hours after birth these passages are colonized by α-haemolytic streptococci and non-pathogenic neisseria.

The respiratory tract is well endowed with physical and chemical components of the superficial defence barrier. The sneeze reflex expels particles from the nose; the cough reflex expels particles from the respiratory tract. In the nose the nasal hairs trap large particles; in the remainder of the respiratory tract the 5 μ thick carpet of mucus traps smaller particles. The particles adhering to the mucus layer are moved to the pharynx by the escalator action of the mucus on the rhythmically beating cilia. Mucus reaching the pharynx is either expectorated or swallowed. The physiological movement of the bronchi also encourages the movement of mucus into the trachea. The mucus layer is not inert but is in itself a chemical barrier, containing both specific and non-specific anti-microbial substances. Glycoproteins can neutralize virus particles preventing adherence to cells; lysozyme and transferrin exert an antibacterial action. Specific IgA is produced locally and secreted into the respiratory tract.

Infectious agents overcome the host’s defences by causing excess mucus secretion thereby causing a defect in the cilial escalator. Patients with a tracheostomy are particularly liable to infection as the inhaled air is less humid than that which has been processed by the respiratory system above the tracheostomy orifice. The mucus dries. Cracks in the mucus blanket lead to defects in the cilial escalator. Patients with tracheostomies require administration of humidified air. Patients recovering from general anaesthesia have depressed cilial activity; they may also be secreting excess respiratory secretions. Patients who have had abdominal surgery are particularly loathe to cough up these excess secretions.

Particles entering the normally sterile bronchioli and alveoli are usually rapidly removed by macrophages. These particles are then transported out of the lung tissue to the draining lymphnodes.
The normal defences of the respiratory system depend upon the following factors —

(a) Mechanical factors — expulsion of matter by the cough and sneeze reflex, the upward movement of the mucus blanket on the ciliary escalator, the introduction of turbulence into the air entering the respiratory system, physiological movement of the bronchi moving secretions upwards.

(b) Chemical factors — mucus containing glycoproteins.

(c) Microbial factors — the upper regions of the respiratory system have a normal bacterial population. Certain potential pathogens may, however, be part of the normal flora in a proportion of the population, e.g. staphylococci.

Humoral defences —

(i) non-specific — lysozyme, transferrin;

(ii) specific — IgA.

Cellular defences — the macrophage plays a vital role in protection of the alveolus.

The respiratory system is a frequent portal of entry for micro-organisms. The anatomical and physiological characteristics of the respiratory system permit not only the entry of organisms into the body by this route, but also provide the organism with a good mode of spread to other hosts. Inhaled air is not sterile. It contains micro-organisms plus other organic and inorganic matter. The larger particles are filtered in the upper respiratory tract and never reach the lung. The smaller particles of under 6 μm enter the lung and tend to settle in the alveoli or smaller bronchioles. Particles of less than 0.5 μm remain suspended and are exhaled. *Mycobacterium tuberculosis* is about 0.4 by 3.0 μm in size; gram positive cocci are approximately 1.0 μm in diameter. These organisms are too large to remain suspended and be exhaled; they are too small to be prevented from entering the lower respiratory tract. Organisms are often inhaled associated with dust particles. Viruses which cause upper respiratory tract infections lead to cell damage. They can destroy cilia and stimulate hypersecretion of mucus. Interference with the muco-ciliary escalator facilitates the spread of bacteria down to the level of the alveoli. Individuals who interfere with the normal defences of their respiratory system, whether by smoking or excess alcohol consumption, all increase their risk of acquiring respiratory tract infections.

During speaking, coughing and sneezing the respiratory system functions as a potential microbial spray gun. Infected secretions are shed in the form of droplets. Large droplets of 100 μm fall rapidly to settle on the floor, on bedding, on surgical equipment, dressings, etc. *Mycobacterium tuberculosis, Corynebacterium diphtheriae*, streptococci, staphylococci are all bacteria which will survive for days and months in dried secretions or dust, provided they are not exposed to ultra-violet light. Dusty, shady areas may therefore harbour pathogenic micro-organisms. These orga-
nisms may be spread to new hosts by direct contact or indirectly by infected dust or fomites. The moisture in droplets of less than 100 μ evap­
rates and the organisms remaining do not settle but may be directly in­
haled, e.g. measles, chicken-pox.

Infectious respiratory diseases are therefore spread by the expulsion of infected secretions from the respiratory system and contracted by the in­
halation of infected dust or secretory droplets. Overcrowding is an impor­
tant factor in the spread of bacterial respiratory infections. The incidence of viral respiratory disease is similar in all sections of the population; the incidence of bacterial respiratory disease is proportional to overcrowding and poverty. The distance between beds in institutions has been shown to be a critical factor in the spread of organisms. The carrier rate of meningococci more than doubles in army camps; this increase in the carrier rate is significantly less if the distance between beds is increased. The incidence of rheumatic fever shows a positive correlation with overcrowding — spread of Streptococcus pyogenes is increased under these conditions. The number of hospital beds permitted per hospital ward is influenced by the respiratory spread of disease.

Medical and nursing staff, when dealing with patients who are immuno­
depressed, when dressing surgical wounds, when delivering babies, or when inserting a urinary catheter, all wear masks. These masks must be worn covering both mouth and nose. The mask is an attempt on the part of the hospital staff to decrease the patient’s risk of acquiring potentially patho­
genic organisms from the upper respiratory tract of the attendant.

Disease of the upper respiratory tract may manifest as coryza, pharyn­
gitis, laryngitis, tracheitis or croup. Viruses are important pathogens in upper respiratory tract infections. Examples include parainfluenza, in­
fluenza, reo, adeno, measles and echo viruses. Important bacterial patho­
gens in this region are haemophilus, bordetella, Streptococcus pyogenes. A major problem associated with viral upper respiratory tract infections and also with bordetella is secondary bacterial infection. Diseases of the lower respiratory tract include lobar and bronchopneumonia. Bacteria are the more important pathogens in this group, e.g. pneumococcus, Staphylo­
coccus aureus, M. tuberculosis, klebsiella, haemophilus and others. Respir­
atory syncitial virus, cytomegalovirus and Pneumocystis carinii, an organism only pathogenic in the immunosuppressed patient, also cause lower respiratory tract infections. Q fever, psittacosis, Mycoplasma pneu­
moniae are all less common respiratory pathogens. Certain systemic diseases may be acquired by inhalation, e.g. smallpox, measles, mumps, whooping-cough, diphtheria, meningococcal meningitis and/or septicae­
mia and streptococcal disease.

Diagnosis of respiratory infections depends on history and examination. Special investigations include X-ray examination and the submission of specimens for microbiological examination. Specimens should reach the laboratory as soon as possible. Swabs are submitted from the upper respiratory tract, sputa from the lower respiratory tract. Faeces are also sub-
mitted. Viruses are usually present during the first few days of the clinical syndrome — specimens submitted at this time only rarely yield viral growth. The isolation of bacteria is more successful. Problems associated with the interpretation of isolates from specimens from the respiratory tract are appreciable because:

(i) The specimen is ‘contaminated’ by normal flora.
(ii) Certain members of the normal flora are potential pathogens.
(iii) Antibiotics or a change in the environmental flora, e.g. hospitalization, can lead to alterations in the patient’s respiratory flora. Hospitalized patients tend to acquire gram negative bacilli in the oropharynx; antibiotics may encourage the growth of oral candida.

SUMMARY
The respiratory system is:
(a) A portal of entry for micro-organisms.
(b) A site from which dissemination of organisms can occur — patient to patient; staff to patient; patient to staff; staff to staff.
(c) Viruses are responsible for the majority of upper respiratory tract infections, bacteria for the majority of the lower respiratory tract infections.
(d) Diagnosis of the clinical condition is made according to the anatomical site of the lesion; identification of the causative organism is made in the laboratory. *Bordetella pertussis* and adenovirus both cause a clinical ‘whoop’. Croup can be caused by *Haemophilus influenzae* and also by the parainfluenza viruses.
(e) Man, by indulging in cigarettes and permitting air pollution, is interfering with the normal defences of his respiratory system.
(f) Sputum is at best a contaminated specimen. The individual collecting sputa is urged to submit a deep cough specimen and not saliva.

Table XXX

| VIRUSES RESPONSIBLE FOR THE COMMON COLD |
|----------------------------------------|
| Coxsackievirus | Rhinovirus | Coronavirus | Reovirus | Adenovirus |
| Influenza      | Parainfluenza | Respiratory syncitial |

**THE URINARY TRACT**

The kidney filters the blood removing certain products of metabolism and correcting the body’s acid base balance. The renal filtrate flows down the renal pelvis and ureters, emptying into a reservoir. Urine is stored in the bladder and when convenient the bladder is emptied via the urethra. Contraction of the bladder is necessary for the expulsion of urine. During pregnancy and in certain cases of abnormal vesico-ureteric anatomy, urine may reflux up to the ureters during bladder contraction. Vesico-ureteric reflux is a potentially dangerous situation.
The urinary tract is a sterile system. Streptococci, corynebacteria, bacilli and yeasts are all found in the distal urethra. Except for the distal portion of the urethra, no bacteria are normally found in the urinary tract. Urine is a suitable culture media for many bacteria. It is deficient in humoral and cellular defences. The hyperosmolarity of urine inhibits phagocytic activity and decreases the bactericidal effect of serum. Fresh urine, with additional nutrients, is continuously added to the bladder; bladder drainage is intermittent. Bacterial metabolites are diluted and removed at regular intervals. Bacterial multiplication is limited by the acidity of urine. A urea concentration of 2-4% has been shown to be bactericidal. The movement of urine out of the urethra acts as a lavage washing bacteria from the distal urethra.

The male has been shown to be less susceptible to urinary tract infection than the female. This may be due to the following —

(a) The presence of bactericidal substances in the prostatic fluid.
(b) The length of the urethra. The male urethra is longer than the female.

Bacteria are thought to reach the kidney by one of the following routes:

(i) Bacteria may ascend from the urethra, bladder and ureters. This is an ascending infection.
(ii) Bacteria may spread to the kidney by the blood stream or in the lymphatics.

The major route of kidney infection is due to ascending infection. The danger inherent in vesico-ureteric reflux is apparent.

Urinary tract infection is expressed as bladder infection — frequency, urgency, burning. Kidney involvement is demonstrated by the presence of loin tenderness.

Cystisis is potentially dangerous as the urine reservoir now contains an infective medium. Reflux of urine is now reflux of an infected medium. Acute and later chronic pyelonephritis can lead to renal failure. The relationship between bacteriuria and pyelonephritis, although strongly suspected, is not completely proven.

Factors which are associated with an increased incidence of urinary tract infection are:

(i) Anatomical abnormalities of the urinary tract.
(ii) The female sex. Females, possibly due to their short urethras and their sex hormones, are more prone to urinary tract infection than males. Trauma to the urethra during coitus aggravates this situation.
(iii) Pregnancy is associated with bacteriuria. The urine excreted by the pregnant female is rich in glucose — glucose provides a good substrate for bacterial growth. The pregnant lady is further a victim of her hormonal balance as vesico-ureteric reflux is a common problem during pregnancy.
(iv) The urine of the diabetic patient is a good culture medium.

Infection of the urinary tract can be exogenous or endogenous. Endogenous infection is usually due to bacteria normally found in the patient’s bowel, e.g. *E. coli, Strept. faecalis*. Over 70% of community acquired urinary tract infections are due to *E. coli*. It is important to identify the bacterium responsible for an infection. Re-infection with a new organism is about twice as common as relapse due to inadequate or incorrect treatment of the original infection. The bacteria most commonly incriminated in urinary tract infections acquired at home are *E. coli* and *Proteus mirabilis*. In hospitals the incidence of klebsiella, proteus (all four species), pseudomonas and antibiotic resistant *E. coli* is markedly increased. Proteus is especially well adapted to cause infection of the urinary tract. This organism produces urease which splits urea and releases ammonia. The ammonia not only neutralises the acid urine producing a more favourable culture medium, it also inactivates any complement which may be present in the urine. Proteus may also, due to its ability to alkalinize urine, precipitate calculus formation.

In hospitals urinary tract infections are characterized by two features:

(a) Infection with antibiotic resistant organisms.
(b) Instrumentation of the urinary tract.

Catheterization of females is routinely performed by the nursing staff. The female patient, already at risk with her short urethra, now has a foreign object introduced into her bladder. Absolute sterility must be observed when a patient is being catherterized. Not only must the catheter be sterile but adequate cleaning of the vulvo-vaginal area is necessary. Catheterization and the presence of an indwelling catheter are a major hazard to the hospitalised patient. Organisms may be introduced into the bladder from both the distal portion of the urethra and from the vulvo-vaginal area. Organisms can penetrate the catheter or be introduced between the urethra and catheter tubing. Nursing staff should not manipulate the connection between catheter and urine bag; if manipulation is necessary it should be done with washed hands. Fluid flows downhill, when catherterized patients are turned the urine in the urine bag should not be emptied into the patient’s bladder. This can be avoided by keeping the urine bag below the level of the bladder or by clamping off, or draining, the urine bag prior to turning sick patients. Similar precautions should be observed when working with urine bags as are observed when working with underwater intercostal drains.

Indwelling catheters which are complicated by bacteriuria may be managed by bladder irrigation. In the absence of systemic disease, pyrexia, or toxaemia, antibiotics are not routinely given to catherterized patients with a bacteriuria. Antibiotic therapy in these cases is seldom successful before the catheter is removed.

Urinary catheters are a potent source of infection because:

(i) They upset the normal physiology of the area.
(ii) They pass from an unsterile environment into a sterile area.

It is of the utmost importance that:

(i) Strict aseptic technique is observed during catheterization of a patient.

(ii) The catheter is removed as soon as the patient’s condition permits.

The collection of urine for bacteriological examination is an important procedure which, if incorrectly performed, results in a valueless laboratory report. A mid-stream specimen of urine is collected. A clean catch is attempted. Urine is collected in a sterile container and submitted to the laboratory as soon as possible. Urine is a good culture medium and specimens reaching the laboratory after a delay are of limited value. When a delay of more than one hour between urine collection and submission to the laboratory is anticipated, the urine should be refrigerated. A temperature of 4°C is not conducive to bacterial multiplication. Bacterial multiplication in the urine specimen renders results of little value as the significance of any bacteria found in a urine specimen is determined by the number of organisms present. Occasionally suprapubic puncture is performed to collect a technically sterile urine specimen. If a patient is already catheterized for medical reasons the collection of uncontaminated urine is not a problem.

*Salmonella typhi*, rubella and cytomegalovirus are excreted in the urine during systemic disease. Urine is therefore a mode of spread for these organisms. An individual with leptospirosis excretes large numbers of organisms in his urine; an individual with bilharzia may excrete viable *Schistosoma haematobium* ova in his urine. This latter urine passed into a snail infested stream converts a safe swimming area into a bilharzial risk. Urine may therefore act as a vector of disease.

Urine is reputed to kill hookworm ova!

**SUMMARY**

(i) Urine is a normally sterile fluid. The presence of organisms in correctly collected urine implies cystitis or urinary tract infection. Occasionally organisms responsible for systemic disease are excreted in the urine.

(ii) Bladder instrumentation is a major factor in the incidence of hospital acquired urinary tract infections. Catheterization assumes rating as a hazardous procedure in view of the risk of introducing bacteria into the urinary tract.

**THE GENITAL TRACT**

The female genital tract is subject to alterations in pH and thus resistance to certain infections varies at various stages of a woman’s life. The hormones initiating and maintaining the menstrual cycle are also responsible for production and storage of glycogen in the vaginal cells. Lactobacilli
are normal vaginal commensals and these organisms metabolise the glycogen, converting it into lactic acid. The vaginal pH is therefore acid as long as the hormones stimulating glycogen are present. The pre-pubertal and post-menopausal female lacks this protective mechanism. Other members of the normal vaginal flora include clostridia, peptostreptococci and \(\beta\)-haemolytic streptococci group B.

The transfer of normal vaginal commensals into other anatomical sites can present a problem. Group B streptococci can be aspirated by the baby during its passage through the birth canal. The premature infant is particularly at risk of fatal meningitis and/or septicaemia as a result of group B infection. Clostridia are a potential hazard to a person requiring an above knee amputation for peripheral vascular disease.

Vaginal infections are often associated with one or more of the following:

(i) the menopause;
(ii) local antibiotic pessaries;
(iii) diabetes.

Vaginal candidiasis is a common complaint. The white plaques characteristic of candida infection are found in the vaginal mucosa. *Trichomonas vaginalis* grows well in a neutral pH and causes a frothy irritating vaginal discharge. This organism is venereally transmitted. Asymptomatic infection of the male prostate can result in repeated reinfection of the female partner. Treatment of both partners is therefore essential.

Certain vaginal or genital infections are more related to sexual exposure than to the vaginal defence mechanisms. The venereal diseases require intimate contact in order for the organism to be spread from one individual to the next. These organisms succumb to drying, heat, cold and other environmental conditions.

| DISEASE                          | ORGANISM                                      |
|----------------------------------|------------------------------------------------|
| Non-gonococcal urethritis        | Chlamydia group or other agents suspected     |
| Gonorrhoea                       | *Neisseria gonorrhoeae*                       |
| Syphilis                         | *Treponema pallidum*                         |
| Chancroid/Soft sore              | *Haemophilus ducreyi*                        |
| Lymphogranuloma venereum         | Chlamydia group B                             |
| Lymphogranuloma inguinale        | *Donovania granulomatis* (organism related to klebsiella) |

Gonorrhoea is clinically characterized by dysuria in the male. A percentage of females are asymptomatic. Infection of the genital mucosa by gonococci requires that the organism adhere to the mucosal cells by pili. The pili confer on the organism the ability to resist phagocytosis and also the ability to cling and remain attached to the mucosa in the presence of
the urine stream. About 10% of males with gonorrhoea are asymptomatic; a very much higher percentage of females are asymptomatic carriers.

Non-gonococcal urethritis, like gonorrhoea, presents as dysuria. Many of the other venereal diseases present with a local lesion on the genitalia, e.g. syphilis, chancroid.

Table XXXII
VENEREAL DISEASES WITH A LOCAL LESION

| DISEASE                        | LOCAL LESION        | DRAINING LYMPHADENOPATHY |
|--------------------------------|---------------------|--------------------------|
| Primary syphilis               | Indurated chancre   | Hard and non-tender      |
| Chancroid                      | Ulcer               | Tender                   |
| Granuloma inguinale            | Granulomatous lesion| May discharge pus        |
| Lymphogranuloma venereum      | Vesicles and papules| Adenitis leads to fibrosis|

The venereal diseases responsible for systemic disease are syphilis and less frequently gonorrhoea.

Other diseases transmitted by sexual intercourse are herpes group II and the virus responsible for serum hepatitis (hepatitis B). Herpes genitalis results in painful vesicles and ulcers on the genitalia — males find intercourse painful and females are particularly prone to relapses at a certain stage of their menstrual cycle. When hepatitis B is transmitted the acute disease may be subclinical.

Table XXXIII
ORGANISMS WHICH MAY BE SPREAD BY SEXUAL CONTACT

| ORGANISM                                      | LESION IN THE MALE | LESION IN THE FEMALE          |
|-----------------------------------------------|---------------------|--------------------------------|
| *Trichomonas vaginalis*                       | often asymptomatic  | vulvo-vaginitis                |
| *Candida albicans*                            | often asymptomatic  | vulvo-vaginitis, cervicitis    |
| Herpes II                                     | painful blisters    | painful blisters               |
| *Neisseria gonorrhoea*                        | urethritis          | often asymptomatic             |
| *Treponema pallidum*                          | painless chancre    | chancroid often not observed   |
| *Haemophilus ducreyi*                         | suppurating ulcer   | ulcer                          |
| Chlamydia (Lymphogranuloma-venereum)         | painless vesicles   | lesion missed in early stages  |
| Chlamydial urethritis                        | urethritis          | asymptomatic                   |
| T-mycoplasma (?)non-gonococcal urethritis    |                     |                                |

Venereal disease is on the increase. Certain sources rate the annual world gonorrhoea incidence at one hundred million cases. In certain geographical areas non-gonococcal urethritis is more prevalent than gonorrhoea. The increase in the incidence of venereal disease is attributed to a number of different factors.
(i) Social causes —
   (a) Industrialization and centralization concentrating the popula-
       tion in large cities.
   (b) An increase in leisure time.
   (c) An alteration in the social norms.
   (d) Loss of the fear of pregnancy due to ‘safer’ contraception.
   (e) Less use of the condom.

(ii) Medical factors —
   (a) The increased resistance of gonococci to penicillin.
   (b) Improved diagnostic techniques.
   (c) Better contact tracing.

SUMMARY

(1) A number of different groups of organisms may be transmitted
    venereally — amongst the viruses — herpes II and hepatitis B; amongst the
    bacteria — $T. pallidum$, $N. gonorrhoea$, $H. ducreyi$; amongst the protozoa —
    $Trichomonas vaginalis$; amongst fungi — $C. albicans$; and chlamydia. T-mycoplasma are suspected genital
    pathogens.

(2) The incidence of venereal disease is proportional to the sexual
    exposure and promiscuity of the individual and the population.

(3) The female genital tract enjoys a better superficial defence barrier
    during the reproductive years of a woman’s life than before this
    period.

(4) Certain venereal diseases may be diagnosed in one partner while
    the other partner is an asymptomatic carrier. In the control of
    venereal diseases it is necessary to treat both partners.

THE SKIN

The skin is one of the well recognised components of man’s superficial
defence barrier. It boasts mechanical, chemical and microbial components.
The mechanical aspect of this barrier is subject to breaches in the hospital
environment due to the surgeon’s knife, the intern’s intravenous drip and
the nurse’s injection needle. The microbial barrier consists of organisms
well adapted to skin conditions. Micro-organisms establishing themselves
on skin are subject to shedding as the superficial squamous epithelial cells
desquamate. The skin is also often too dry for the prolonged survival of
pathogens. Constant contact between the skin and the environment leads
to two different bacterial population groups —

(a) The resident flora: These are organisms which are well adapted to
    skin conditions and are consistently isolated from the skin. Examples of
    resident bacteria include staphylococci, corynebacteria. Non-pathogenic
    mycobacteria are found in areas rich in sebaceous glands; fungi, e.g. yeasts, are found in skin folds where conditions
    are moister.
Transitory flora: These organisms are subject to changes. They may be carried for only a limited period of time.

Neither washing nor scrubbing significantly alters the resident flora of the skin. Hexachlorophene, an antiseptic agent against gram positive cocci, is used by theatre staff in an effort to decrease the gram positive organisms on their hands and forearms. Regular frequent use of hexachlorophene leads to a residual anti-bacterial effect due to penetration of this agent into the glands of the skin. A single application has no significant or prolonged effect on resident flora.

The chemical skin barrier is associated with an acid pH, the presence of toxic fatty acids in sebaceous secretions and the saline and lysosomal content of sweat. Sweat is the secretion of eccrine glands.

Breaches in the skin can lead to local skin infections or to systemic disease. Systemic disease may also be associated with skin manifestations in the form of skin rashes. Numerous members of both the kingdoms of the Protista and Arthropoda are responsible for skin lesions. Only certain important skin diseases will be mentioned.

**Viral Causes of Skin Lesions**

Viruses cause a number of different skin lesions. Four basic types can, however, be differentiated.

(a) The macular-papular skin lesion. Different degrees of these two basic components may be found in rashes caused by, e.g. rubella, rubeola and enteroviruses.

(b) A vesicular rash. Vesicular rashes may in the early phases be macular and may, after the formation of vesicles, progress further to pustules or scabbed areas. The depth of the skin lesion will determine if residual scarring will result. Two viral groups causing vesicular rashes are the poxviruses including smallpox, and the herpes viruses including chicken-pox.

(c) Haemorrhagic rashes. Any of the above rashes may develop a haemorrhagic component. In certain cases the haemorrhage into the skin is a result of platelet insufficiency. Certain members of the arbovirus group are recognised as agents causing haemorrhagic rashes. Haemorrhagic measles is a well recognised entity.

(d) Skin tumours. *Molluscum contagiosum* and the human wart virus are both DNA viruses which cause skin tumours or warts.

**Rickettsia and Skin Lesions**

Rickettsial diseases give rise to a macular-papular skin rash. The nobby papular component of the rash is marked.

**Bacterial Causes of Skin Lesions**

Bacteria can give rise to different skin lesions, e.g. secondary syphilis is manifest by a maculo-papular rash, meningococcaemia is accompanied by a macular skin rash which may become haemorrhagic. A chronic skin ulcer may be due to *Mycobacterium ulcerans* or *Mycobacterium marinum*. 
Tuberculosis, a systemic disease, can occasionally seed organisms to the skin. Leprosy is a disease that has skin and peripheral nerves as its target organ. In hospital there are two organisms that are of especial significance in skin infections. These are *Streptococcus pyogenes* and *Staphylococcus aureus*. Both of these organisms are a particular danger in surgical and neonatal units. Staff with streptococcal or staphylococcal skin lesions should be excluded from these areas. Streptococci, and more especially staphylococci, are carried by a number of healthy individuals in their upper respiratory tract. Nasal staphylococcal carriers present a particular problem. The wearing of masks is an attempt to decrease this problem.

Lesions caused by *Streptococcus pyogenes* include:

(i) Impetigo — a highly infectious skin condition. The superficial layers of the skin are infected and form golden crusts.

(ii) Ecthyma — a deeper lesion with a thicker crust which leaves residual scarring.

(iii) Erysipelas — the organism commonly invades the skin through invisible breaches, but surgical wounds may also become infected. Erythema and swelling are typical.

(iv) Cellulitis — a spreading skin lesion.

(v) Scarlet fever — this is a skin manifestation of a systemic infection by the organism. *Streptococcus pyogenes*, when infected by a particular bacteriophage, develops the ability to secrete erythrogenic toxin. This toxin is responsible for the rash in scarlet fever.

(vi) Sensitization or allergic lesions — in certain diseases caused by *Streptococcus pyogenes*, skin rashes may reflect overtones of the imbalance between host and parasite, e.g. in rheumatic fever the host may develop erythema marginatum or erythema nodosum.

Skin infection by *Streptococcus pyogenes* can be the factor initiating acute glomerulonephritis in certain individuals.

*Staphylococcus aureus* is possibly the single most important organism in certain hospital wards. The clinical manifestations of the disease depend on the site of the organism, e.g. invasion alongside of a hair follicle gives rise to a localized folliculitis; subcutaneous convergence of several adjacent infected hair follicles cause a carbuncle. An abscess starts in many cases as a staphylococcal infection. Staphylococci can cause different forms of impetigo. In infants and young children bullous impetigo can lead to systemic disease and death. This form of impetigo is highly contagious. An important reason for the obligatory gowning, gloving and masking of persons entering neonatal nurseries is staphylococcal bullous impetigo. In adults staphylococcal impetigo is a benign pustular crusted lesion. A particular *Staph. aureus* is capable of causing the scalded skin syndrome (toxic epidermal necrolysis) in the young. The neonate and young infant are particularly susceptible to infection by this organism. A toxin is secreted by the organism which acts on the skin at the level of the stratum granulosum, resulting in skin peeling.
Staphylococcal septicaemia can also give rise to skin lesions. These lesions are generally haemorrhagic with necrotic areas.

The introduction of an epidemic strain of staphylococci into a neonatal ward results in rapid colonization of 90% of the infants. Some of these infants will develop overt disease while others will become asymptomatic carriers. They may transmit the organism to newborn babies joining them in the unit; they may also infect their mothers, giving them mastitis and a breast abscess. The staphylococcus is just as great a problem in the surgical ward. Surgical wounds with their sutures provide an excellent site for staphylococcal infection. The infective dose of staphylococci is decreased by 1 000 to 10 000 times in the presence of foreign material — including suture material.

Certain other bacteria may colonize wounds. Pseudomonas is a particular hazard in burns patients, while Clostridium perfringens is a danger in anaerobic necrotic skin lesions.

SKIN LESIONS DUE TO FUNGI
Ringworm or dermatophytosis is an important superficial fungal infection of the skin. Candida is particularly common in moist skin fold areas. Candida causes paronychia as frequently as staphylococci.

Many other fungi cause skin lesions. These are less commonly encountered and include sporotrichosis, mycetoma and chromomycosis, a warty lesion. Certain systemic fungal diseases may invade the host using the skin as a portal of entry.

SUMMARY
(1) The skin is one of the natural superficial barriers of defence.
(2) The skin may be the target organ in certain local infections, e.g. cellulitis; it may act as the portal of entry for certain systemic diseases, e.g. tetanus, and it may be one of the organs experiencing changes due to systemic infections, e.g. jaundice due to viral hepatitis.
(3) Infective skin lesions may participate in the spread of disease by direct contact or by shedding of bacteria into the environment.
(4) Many different micro-organisms are capable of infecting the skin. The most important of these in the hospital situation is Staphylococcus aureus.
(5) The skin has a limited range of reactions available to it — it may form a macule (an area of redness), a papule (a raised area), a vesicle (a blister) or a pustule (a vesicle with pus). Haemorrhage may occur into any of these lesions. Skin death leads to an ulcer or necrotic area. Deeper lesions of the skin can lead to brawny induration or abscess formation. Occasionally the skin can produce warty masses in response to infection.
THE CARDIO-VASCULAR SYSTEM

The cardio-vascular system represents one of the normally sterile anatomical areas. Invasion of the blood stream by bacteria may occur from breaches in the skin, or mucous membranes. Infection of any organ system may be associated with a bacteraemia, e.g. pneumococcal pneumonia can often be diagnosed by the presence of lung pathology and a positive blood culture. Certain diseases are typically characterized by the blood spread of organisms during particular phases of the illness, e.g. typhoid, measles. During mastication or any dental manipulation, commensal oral bacteria may, for a short period, be found in the blood stream.

In hospitals, intravenous and/or intra arterial catheters are a potential source for the introduction of bacteria or fungi. Removal of these catheters is often followed by the conversion of a hitherto positive blood culture to negative.

The presence of bacteria in the blood stream can result in two different types of complications:

(a) The bacteria can multiply. Bacterial products may be released and severe patient reactions may occur, e.g. endotoxic shock. These organisms may settle in previously normal organs, e.g. a staphylococcal abscess in the kidney. Septicaemia may develop in a previously healthy individual.

(b) The second syndrome of complications associated with release of bacteria into the blood stream is related to a select group of patients. Patients who have previously suffered damage to their heart valves, be it due to rheumatic fever, arthrosclerosis and calcification, or cardiac surgery, are at risk of developing subacute bacterial endocarditis. The organisms settle and multiply in vegetations (clots) which have formed on the damaged heart valves. These organisms are then shed into the blood stream at intervals. The organisms can cause further damage to the heart valve. The patient may develop congestive cardiac failure or may suddenly demise due to rupture of the valve.

Organisms commonly implicated in subacute bacterial endocarditis are *Streptococcus viridans* and *Streptococcus faecalis*. In hospital practice Gram-negative organisms are today more commonly cited as agents causing bacteraemia than Gram-positive cocci.

When specimens are being collected for blood culture, extreme care must be taken in order to avoid contamination of the specimen. Contamination of the specimen by a single skin bacterium can lead to diagnostic and therapeutic headaches.

SUMMARY

(1) Introduction of invasive bacteria into the blood stream may result in septicaemia. Gram-negative bacilli are the group of bacteria most frequently implicated in nosocomial septicaemia.

(2) Patients who have damaged heart valves are at risk of developing infection of these rough surfaces.
(3) The danger of endocarditis is increased in hospitals where invasive procedures may precipitate a bacteraemia. These procedures include prostatic examination, sigmoidoscopy and vascular catheterization.

THE GASTRO-INTESTINAL TRACT

The gastro-intestinal tract (GIT) is that organ system responsible for digestion and absorption of nutrients. The food which enters the GIT enters a tube with an orifice at both ends. During its passage through this tube food is subject to the effects of digestive enzymes acting in the stomach at an acid pH, and in the duodenum at an alkaline pH. The duration of contact between digested food and the absorptive surface of the mucosa is determined by gut motility. Absorption is a function of the stage of digestion which food has reached, the food mucosal contact time and the presence of normal mucosa. Dysfunction of this organ system can be expressed by a change in motility — increased motility results in diarrhoea, decreased motility in constipation. Malabsorption of food may result in weight loss, anaemia, steatorrhoea and general ill health.

With each mouthful of food ingested micro-organisms are introduced into the gastro-intestinal tract. The bacterial count in a fasting stomach is very low — 10-100 organisms per gram of stomach contents. After a meal this count rises to between one million to one hundred million organisms per gram of stomach contents. Half the bulk of faeces is composed of living bacteria or their remains. The superficial barrier of the GIT consists of:

(a) A mechanical component — Peristalsis moves intestinal contents, including bacteria, towards the anal orifice.

(b) A chemical component — the acid barrier in the stomach kills many micro-organisms. Most of the pathogenic micro-organisms are susceptible to an acid pH of 1-2.

(c) A microbial component — faeces consist of bacteria, undigested residual food and various other alimentary secretions and excretions. Faeces may contain as many as $10^{12}$ organisms per gram. The bacterial count decreases from the large intestine, where a count of $10^{10}$ organisms per gram of contents is common, to counts of $10^8$ or $10^9$ organisms per gram of small intestinal contents. It has been shown that the normal intestinal flora has a protective effect.

The normal intestinal flora interferes with superinfection or colonization by pathogenic organisms. The mechanism whereby this protective effect is achieved is not proven but various postulates have been made. The normal bowel flora is well adapted to environmental conditions in the bowel; the pathogens may find the environment created by the digestive processes and the resident bacteria less than ideal. Resident bowel flora are predominantly anaerobic. Bacteroides, the organism present in largest numbers in the bowel, is a strict anaerobe whose growth is stimulated by bile salts. Competition between residents and pathogens for
nutrients and space may play an important role. Certain of the residents may produce products toxic to any would-be intruder.

Various humoral secretory products may also play a role in protection of the intestine against infection. Lysozyme has been detected in intestinal secretions — its importance has, however, not been accurately assessed. Secretory IgA is produced by local lymphoid tissue. This antibody is resistant to digestion by the enzymes present in the lumen of the intestine. IgA is capable of activating complement by the indirect pathway. The presence of IgA in the intestinal lumen is thought to play an important role in the resistance of the gastro-intestinal tract to infection. IgA found in faeces is termed coproantibody.

Breast milk provides both maternal antibody and lactoferrin, a bacteriostatic compound, to the neonatal GIT.

The intestine can provide a portal of entry for agents causing a wide spectrum of disease patterns:

(a) Organisms causing systemic disease, e.g. typhoid, polio, infectious hepatitis, enter the body by means of ingestion.

(b) Ingested ova hatch in the intestine. The larva may leave the intestinal tract to wander about the body before returning to the intestinal lumen as an adult, e.g. ascaris.

(c) Local infection of the intestinal tract may occur.

The presence of intestinal bacteria can lead to the following clinical syndromes:

(a) Malabsorption.

(b) Dysentery or diarrhoea.

(c) Coma associated with liver failure.

(d) Metabolites of intestinal bacteria have been postulated to bear an aetiological relationship to colonic carcinoma.

**Bacterial Infection of the GIT**

(a) **Malabsorption**: This can occur as a result of the blind loop syndrome leading to excessive localized bacterial proliferation. The equilibrium between bacteria and the small intestine is disrupted, leading to interference with digestion. Digestion may be impaired as a result of competition for nutrients, due to decomposition and deconjugation of bile salts or due to the production of irritant metabolites.

(b) **Diarrhoea and dysentery**: Diarrhoea is the frequent passage of unformed stools. Dysentery implies inflammation of the bowel wall and hence the presence of blood, mucus and cells in the stool.

Mechanisms whereby diarrhoea or dysentery may occur:

(i) Due to enterotoxin secretion — Enterotoxin is an exotoxin secreted by certain intestinal pathogens, e.g. *V. cholerae*, enteropathogenic *E. coli*. Enterotoxin attaches to the mucosal cells of the small intestine causing stimulation of adenyl cyclase activity. Adenyl cyclase
is an enzyme which controls fluid and electrolyte secretion. Increased adenyl cyclase activity leads to a nett outpouring of fluid and electrolytes into the small intestine. Absorption is normal. Enterotoxin acts on the small bowel.

(ii) The bacteria may invade the mucosal cells, resulting in destruction of the superficial epithelial cells, e.g. shigella, invasive E. coli.

(iii) The host's immune response to bacteria surviving within phagocytes may be responsible for the diarrhoea in salmonellosis.

Diarrhoea can be a result of intoxication or infection. Intoxication occurs when a preformed toxin is ingested, e.g. as in Staph. aureus, and Clostridium botulinum food poisoning. Infection is due to the ingestion of viable bacteria. The pathogenesis of this diarrhoea is related to the multiplication of the organisms within the bowel. Examples of organisms causing infective diarrhoeas include V. cholerae, Salmonella species. Shigella dysentery is an infection.

The presence of the pathogen in the small intestine often leads to a watery diarrhoea, e.g. V. cholerae, toxigenic E. coli. The pathogens causing dysentery — a bloody diarrhoea — are usually found in the large intestine.

Bacteria may therefore alter the bowel's motility either by production of extracellular toxins or by invasion of mucosal cells.

(c) Hepatic coma: Patients who have suffered severe liver damage as a result of yellow fever, viral hepatitis or toxin ingestion are subject to liver failure and hepatic coma. The liver is no longer capable of adequately playing its role as an organ of detoxification. In cases of liver failure the brain is exposed to toxic substances absorbed from the intestinal tract.

Bowel bacteria produce a variety of metabolites. One of the chief by-products of protein catabolism in the bowel is the conversion of protein and urea to ammonia and various active amines. Ammonia has been particularly incriminated in the pathogenesis of hepatic coma.

Patients with serious liver disease or liver failure therefore have a restricted protein diet and attempts are made to decrease the bowel bacteria by using both antibiotics and purgatives. The presence of blood in the bowel of the patient with liver failure can precipitate hepatic coma.

(d) Bacteria and colonic carcinoma: Colon cancer is one of the most important cancers in modern society. Many of the postulated aetiologies involve dietary habits of modern man. The carcinogen(s) responsible for colonic cancer are thought to be produced in situ. Bacterial action on intestinal secretions or on dietary constituents has been postulated to produce carcinogenic substances. Bacterial metabolism of sterols and various amino acids, e.g. tryptophan, are suspect. Bacteria may also convert primary amines in the presence of nitrite into carcinogenic nitrosamines. Certain foodstuffs have nitrates incorporated to inhibit the growth of Clostridium botulinum. It is feared that these may be converted to nitrosamines by intestinal bacteria. This is the theory behind the uncertainty
expressed by certain people about the consumption of red preserved meats.

**Viral Infection of the GIT**

Certain viruses are regularly found in the gut, e.g. echo and adenoviruses. These may be normal commensals or be associated with disease. Many viruses are suspected of causing diarrhoea — the rotaviruses, a member of the togavirus group, and the parvoviruses, feature prominently on this list.

**Protozoa and Intestinal Disease**

Amoebic dysentery is caused by invasion of the large intestine by *Entamoeba histolytica*. Differentiation between amoebic and bacillary dysentery or shigellosis is rapidly achieved by direct examination of the stool at the bedside.

*Guardia lamblia* and *Balantidium coli* are other protozoan organisms implicated in human disease.

**Infestations of the GIT**

A number of adult worms can inhabit the intestine of man. Certain of these worms are ingested as eggs, e.g. ascaris, others penetrate the skin, e.g. hookworm. The two species mentioned have a systemic migration and return to live as adults in the small intestine. Other worms are ingested, e.g. enterobius, taenia, and mature in the lumen of the intestine. These worms never penetrate the intestinal mucosa and embark on systemic spread. Taenia is found in the small intestine, enterobius and trichuris in the caecum. Man may ingest these parasites as ova or in their larval forms.

The presence of adult worms in the intestine may be detected by the excretion of eggs in the faeces.

**Submission of faecal specimens**

Faecal specimens may be collected as portion of stool or in the form of a rectal swab. Collection of specimens for anaerobic organisms must be made under anaerobic conditions.

**Control of diseases spread by faecal-oral transmission**

Many diseases are spread by the faecal oral route. Organisms may be passed on the faeces of both patients and carriers. The organisms may then directly or indirectly, by soiled hands or fomites, contaminate food and water. In general, diseases are communicable from the end of the incubation period to the onset of convalescence.

Patients suffering from typhoid or cholera should be nursed in semi-isolation. Nursing staff should take particular care in the handling of these patients’ excreta. The excreta should be treated prior to disposal, e.g. the rice water stool of the cholera patient may be collected in a bucket containing disinfectant. Nursing staff entering the patient’s cubicle should glove and gown. Hand washing on leaving is essential. The patient’s eating utensils and bedlinen should be kept separate, and not mixed with that of the general ward.
Nursing of patients excreting pathogenic bacteria is orientated towards breaking the faecal-oral transmission cycle in the patient and preventing the creation of this cycle between the patient and others. Careful handling of excreta and good hygiene are essential.

SUMMARY

(1) Infectious diseases of the GIT are usually acquired by the faecal-oral route. The prevention of these diseases can largely be achieved by the implementation of adequate sanitation and hygiene. Control depends on interrupting the faecal-oral cycle.

(2) A wide spectrum of organisms are capable of being spread by the faecal-oral route and of causing diseases of the GIT, e.g. worms, protozoa, bacteria and viruses.

(3) Some ingested agents cause local disease, others cause systemic disease.

(4) The GIT is endowed with good host defences.

Table XXXIV

| VEHICLE                  | CAUSATIVE ORGANISM                        |
|--------------------------|-------------------------------------------|
| FOOD POISONING           | *Clostridium botulinum*                   |
|                          | *Staphylococcus aureus*                   |
| FOOD-BORNE INFECTION     | *Salmonella species*                      |
|                          | *Brucella species*                        |
|                          | *Mycobacterium tuberculosis* or *M. bovis*|
|                          | *Bacillus cereus*                         |
|                          | *Clostridium perfringens*                 |
|                          | Rarely *Shigella*                         |
|                          | *Vibrio parahaemolyticus*                 |
| MILK-BORNE DISEASE       | *Salmonella species*                      |
|                          | *C. diphtheriae*                          |
|                          | *Strept. pyogenes*                        |
|                          | *Brucella*                                |
|                          | *Mycobacterium bovis*                     |
| WATER-BORNE DISEASE      | *V. cholerae*                             |
|                          | *Salmonella typhi*                        |
|                          | Leptospirosis                             |
|                          | Hepatitis A                               |
|                          | Poliovirus                                |

THE JAUNDICED PATIENT

Jaundice may result from any insult or disease process in the liver which results in loss of functional hepatocytes. Jaundice is also a feature of a number of different infections. Jaundice due to diseases caused by infective agents includes malaria, yellow fever and leptospirosis. From the hospital viewpoint the most commonly encountered infectious cause of jaundice is that associated with viral hepatitis. Infectious hepatitis is due to hepatitis A virus which is transmitted by the faecal-oral route. When the serum bilirubin reaches its peak or the jaundice is most intense, then the
patient is no longer capable of transmitting the virus. The main danger period is before the patient becomes jaundiced and before the diagnosis is made — during this period the patient is excreting the virus and the nursing staff may not be taking adequate precautions. Hepatitis B virus is responsible for serum hepatitis (long incubation hepatitis). This virus is transmitted by injection, especially of blood products, by venereal spread and also occasionally by the oral route. It has also been reported to be secreted in tears. Carriers of hepatitis B may be a hazard to the nursing staff. Hepatitis B presents a particular problem in dialysis units and theatre.

Table XXXV
INFECTIVE CAUSES OF JAUNDICE

| VIRAL                                      | BACTERIAL                                      |
|--------------------------------------------|-----------------------------------------------|
| Hepatitis A virus                          | Leptospira                                    |
| Hepatitis B virus                          | Treponema pallidum — congenital               |
| Infectious mononucleosis                   | Mycobacterium tuberculosis — rare             |
| Yellow fever virus                         | Protozoal                                     |
| Lassa fever virus                          | Amoebiasis                                    |
| Cytomegalovirus — congenital               | Plasmodia                                     |
| Marburg virus                              | Toxoplasma — congenital                       |
| Herpes type I                              | Other                                         |
|                                            | Chlamydia psittaci                            |
|                                            | Coxiella burnetti                             |

THE SKELETAL SYSTEM

Bones are rigid structures important in providing stability in the human body. They have three anatomical layers — the outer membrane or perios­teum, the cortical layer and the inner medulla. Bone infections present a serious problem. Irritation of the perios­teum, e.g. in infection, can lead to laying down of new bone and thickening of the cortex. The cortex interferes with drainage of pus and infective material from the medulla. Although the medulla is a blood “lake”, the volume of blood supplied to the bone is relatively poor, therefore antibiotics do not generally reach high levels in bone. Poor drainage and inadequate concentrations of antibiotics make bone infections difficult to treat.

Acute infection of bone is called osteomyelitis. Osteomyelitis can result from blood spread or be secondary to trauma, e.g. a compound fracture. Staphylococcus aureus is a major culprit. Gram-negative bacilli, e.g. klebsiella, proteus and others are less common but may present problems in orthopaedic units. Children are particularly susceptible to bone infections. The common causative agents of osteomyelitis in this age group are gram positive cocci. Skeletal tuberculosis is usually secondary to tuberculosis elsewhere. Skeletal tuberculosis is characterized by a cold abscess.

Osteomyelitis is an abscess within a rigid cavity and is difficult to cure. Orthopaedic surgery is attended with the risk of bone infection. Of all theatres, the theatre used for orthopaedic surgery is the one where sterility
and aseptic techniques are the most emphasised. All too frequently osteomyelitis requires prolonged antibiotic therapy, surgical drainage and bone curettage.

Other infections which can result in bone changes include:

(a) *Taenia echinococcus* — cysts of the larval stage of this worm are commonly found in the liver but may also be found in bone.

(b) Fungal infections of the bone occur. The fungal disease most frequently incriminated is coccidiomycosis.

(c) Leprosy, syphilis and granuloma inguinale all cause bone changes.

(d) Certain viruses can affect bone, e.g. characteristic changes in the bones of neonates with congenital rubella are found.

The skeletal system consists not only of bones but also of joints. Infection of joints can be due to direct involvement of the joint by an organism. A joint lesion may also be the result of the host's reaction to the presence of an organism. Staphylococci can cause acute suppurative arthritis, *Streptococcus pyogenes* may cause an immune type of flitting joint pain. Other organisms recognised to have an aetiological relationship to arthritis include *N. gonorrhoeae*, brucella, *Mycobacterium tuberculosis* and treponema.

THE CENTRAL NERVOUS SYSTEM

Infection of the brain causes encephalitis, infection of the membrane lining the brain causes meningitis.

THE BRAIN

Infection of the brain is most commonly due to viruses. Viral encephalitis may be the presenting system of certain viral infections or it may present as a complication of a systemic viral disease, e.g. measles, mumps, herpes type I, infectious hepatitis, smallpox, etc. Echoviruses and certain arboviruses have the brain as their target organ.

The brain is separated from the rest of the body anatomically by the meninges and the bony cranium. Physiologically it is distinct from all other organs due to the presence of the blood-brain barrier. This barrier acts as a protective mechanism whereby substances are selectively transported to, or excluded from, the brain. Any organism reaching the brain in the blood stream must cross this barrier. The phagocytic cells of the central nervous system are the microglia.

Encephalitis is clinically manifest as a disturbance of cerebral function. Signs of infection, e.g. pyrexia, may also be present. Pathologically there are three different forms of infective encephalitis. Measles may give rise to any of these forms:

(a) Acute viral encephalitis — This is as a result of viraemia, involving spread to the brain. The signs of encephalitis occur concurrently with other signs of the disease, e.g. skin rash. In mumps about half the cases have a mild form of acute mumps encephalitis.
(b) Post-infectious encephalitis — This is preceded by the clinical signs of the disease, e.g. the morbilliform rash is present 2-3 weeks before the onset of encephalitis. This form of encephalitis is associated with damage to neurones or their myelin sheath, resulting in the release of encephalitogenic protein. This latter substance is antigenic and induces host antibodies. These antibodies react with the host’s neurones. The host is therefore attempting to reject this own brain. The prognosis is poor.

(c) Subacute sclerosing panencephalitis — Approximately one in every million people who have had measles develop subacute sclerosing panencephalitis. The measles virus genome or an altered genome persists and multiplies in neurones. This is a slow virus disease. With loss of neurones the individual becomes retarded. Subacute sclerosing panencephalitis usually presents one or two years after the acute attack of measles.

There is no cure.

Meningoencephalitis is frequently associated with alimentary viruses, e.g. coxsackie, echo and occasionally poliovirus. Meningoencephalitis is clinically characterized by signs of both cerebral involvement and meningeal irritation. There is no loss of consciousness.

Bacterial infection of the brain usually occurs in the form of an abscess. The organism may reach the brain as a result of blood spread or directly due to a fractured base of skull. Brain abscess may complicate a case of meningitis.

THE MENINGS

The meninges are the membranes lining the brain and spinal cord. This is a normally sterile area. The presence of an organism in this area is synonymous with disease. Organisms may reach this area in the blood stream or they may spread directly from the nasal cavity or sinuses through the cribiform plate or through a fractured skull. Meningitis, an infection of the meninges, can be acute or chronic. Bacteria are frequently the pathogens involved. Different organisms assume greater importance in different age groups.

Bacteria causing acute purulent meningitis in the neonate are usually Gram-negative bacilli, e.g. *E. coli*. Babies are particularly susceptible to Gram-negative infections, as IgM, the immunoglobulin which opsonises these organisms, does not cross the placenta. IgG does cross from the mother into the foetal blood stream. At birth the immune system is not fully mature, and these babies are therefore at greater risk than older children. *Streptococcus agalactiae* may cause fatal meningitis and septicaemia in the neonate. The baby is infected during its passage through the birth canal of a maternal carrier. Meningitis in the neonate may have an unusual presentation; a listless, irritable baby who vomits should be suspect. The baby’s temperature may be raised or it may be subnormal. A stiff neck is a late sign of meningitis in the neonate.
Three organisms are commonly implicated as the cause of acute purulent meningitis. These organisms are *N. meningitidis*, *Strept. pneumoniae* and *H. influenzae*.

*Neisseria meningitidis* is responsible for meningitis epidemics. Various groups of this organism are carried in the throat of many individuals. The carrier rate of *N. meningitidis* increases with overcrowding. *N. meningitidis* causes meningitis in all age groups — often following some temporary derangement of host defences, e.g. after a viral illness. Prophylaxis against meningococcal meningitis is in a state of flux. Nursing staff in contact with a patient diagnosed as suffering from meningococcal meningitis were, in the past, all placed on prophylactic sulphamamide therapy. Today certain types of the organism are resistant to sulphamamides. A modern approach to the handling of meningococcal meningitis contacts is to withhold antibiotics and carefully observe the individual. The opportunity to prove this approach is optimal in hospital staff. When prophylactic antibiotics are given minocycline may be used in combination with rifampicin.

The resistance of the meningococcal population to sulphamamides differs in different geographical areas. It is high in Brazil and on the increase in South Africa.

*Streptococcus pneumoniae* is frequently the aetiological agent of a meningitis which follows a fractured base of skull, otitis media or pneumonia. Owing to the associated pathology in pneumococcal meningitis, the death rate may be higher than that of meningococcal meningitis.

*Haemophilus influenzae* causes its peak incidence of meningitis in the age group from 4 to 24 months. Outside of epidemic periods, *H. influenzae* is often quoted as being the major cause of meningitis. *H. influenzae* meningitis is well recognised as being followed by complications, e.g. a subdural effusion.

Clinically meningitis is manifest by a stiff neck and pyrexia. When the cerebral tissue becomes involved, changes in the patient's level of consciousness, mentation and personality occur.

Acute bacterial meningitis is a medical emergency. Nursing staff are obliged to wake up any overworked, slumbering casualty officer if they feel that the new case may be a case of meningitis. Early treatment of meningitis may not only save the individual's life, it may also prevent irreversible deafness and mental retardation.

*Mycobacterium tuberculosis* and *Cryptococcus neoformans* may also cause meningitis. The cerebro-spinal fluid (CSF) picture in these cases differs from the laboratory picture of acute bacterial meningitis. These organisms cause a less acute, often chronic form of meningitis. Aseptic meningitis is a bad term used to describe meningitis where there is a negative bacterial culture of the CSF. Certain viruses, e.g. echo, coxsackie, are the common aetiological agents of aseptic meningitis.
Diagnosis of the type of meningitis depends on the laboratory finding on examination of the CSF. Identification of the agent causing the meningitis determines the treatment. Adequate early treatment of the case determines the prognosis. Cerebrospinal fluid is obtained by lumbar puncture. A needle is pushed through the skin and guided between the lumbar vertebral spines into the meninges. The fluid bathing the brain and spinal cord is then withdrawn. Strict aseptic technique must be observed. Introduction of an organism on the needle or from the overlying skin can lead to infection of this normally sterile area. The cerebrospinal fluid is examined in the laboratory for:

(a) The causative organism: This is done immediately by direct examination. A gram stain is used.

Intracellular Gram-negative diplococci are interpreted as N. meningitidis, pleomorphic Gram-negative coco-bacilli as H. influenzae and lanceolate gram positive diplococci as Strept. pneumoniae. Less commonly other organisms may be seen. In selected cases a Ziehl-Neelsen stain for acid-fast bacilli or an Indian ink preparation for cryptococci may also be performed.

(b) A cell count: In acute bacterial meningitis the polymorphonuclear leucocyte cell count is markedly raised. In fungal, viral or chronic bacterial meningitis, lymphocyte counts are important. The presence of cells in the CSF represents the response of the host to irritation of the meninges.

(c) CSF chemistry: In acute bacterial meningitis the protein level in the CSF is increased. This is related to the increased permeability of vessels and the increased movement of protein across inflamed linings. The presence of necrotic cells may add to the protein content of the CSF. The sugar level is decreased — this may reflect an increase in metabolism due to the efforts of the phagocytic cells to control the infection. In viral meningitis the sugar level of the CSF is unaffected.

Treatment is started once microscopy and a chemical analysis have been performed. Culture, final identification and drug sensitivity of the organism may follow within 2 or 3 days.

SUMMARY

(1) Meningitis can be caused by a variety of agents. The agents most commonly incriminated are bacteria. The three bacteria most frequently involved are N. meningitidis, H. influenzae and Strept. pneumonia.

(2) Acute bacterial meningitis is a medical emergency. Early, adequate treatment significantly alters the mortality and morbidity of this condition.

(3) Strict aseptic technique is essential when performing a lumbar puncture. The lumbar puncture is an invasive technique temporarily connecting the external environment with the sterile fluid surrounding the central nervous system.
| Organ Systems and Disease |
|---------------------------|

**Table XXXVI**

EXAMPLES OF ORGANISMS INFECTING CERTAIN ORGAN SYSTEMS

| WOUND INFECTIONS | Staphylococcus aureus  
|                  | Streptococcus pyogenes  
|                  | Pseudomonas pyocyanea (N.B.: burns) |
| CHRONIC SKIN LESION | Mycobacteria — atypical, *M. tuberculosis*, *M. leprae*  
|                    | Corynebacterium — *C. diphtheriae*, *C. ulcerans*,  
|                    | *C. minutissimum* |
|                    | Ringworm |
| SYSTEMIC DISEASE WITH SKIN INVOLVEMENT | Neisseria — *N. gonorrhoea*, *N. meningitidis* |
| SKIN INVOLVEMENT | Rickettsia  
|                  | Viral exanthemata of childhood — measles, rubella,  
|                  | chicken-pox, roseola infantum  
|                  | *Strept. pyogenes* in scarlet fever |
| MENINGITIS |  
| ACUTE | *N. meningitidis*  
| CHRONIC | *M. tuberculosis* |
| NEONATE | Gram negative bacilli —  
|          | *E. coli* |
| ENCEFALITIS | Coxsackie  
|            | Echovirus  
|            | Arbovirus  
|            | Also mumps, measles, herpes III viruses |
| PNEUMONIA | *Strept. pneumoniae*  
|            | *Klebsiella pneumoniae*  
|            | *Staph. aureus*  
|            | *Mycobacterium tuberculosis* |
| URINARY TRACT INFECTION | *E. coli*  
|            | *Strept. faecalis*  
|            | *Klebsiella*  
|            | *Proteus* |
| SORE THROAT | *Strept. pyogenes*  
|            | Ebstein Barr virus (infectious mononucleosis)  
|            | Adenovirus  
|            | Coxsackievirus  
|            | *C. diphtheriae* |
| OTITIS MEDIA | *Strept. pneumoniae*  
|            | *H. influenzae*  
|            | *Strept. pyogenes*  
|            | A complication of measles |
| SINUSITIS | *Strept. pneumoniae*  
|            | *Strept. pyogenes*  
|            | *H. influenzae* |
| GROUP/LARYNGO-TRACHEO BRONCHITIS | *H. influenzae*  
|            | Parainfluenza virus  
|            | Respiratory syncitial virus  
|            | Adenovirus |
### Table XXXVI (continued)

| SUPPURATIVE LESION | Pathogens |
|---------------------|-----------|
| SUPPURATIVE LESION  | *Staph. aureus*<br>Anaerobic organisms — Peptococci, Peptostreptococci, Bacteroides, Fusobacterium  
| ENDOCARDITIS        | *Strept. viridans*<br>*Strept. faecalis*<br>*Staphylococcus*<br>Rarely rickettsiae, candida |
| SEPTICAEMIA         | Gram negative bacilli — Pseudomonas, *E. coli*, etc.  
| OSTEOMYELITIS       | *Staph. aureus*<br>*N. meningitidis* |
| CONJUNCTIVITIS      | *N. gonorrhoea* — in the neonate  
| VENEREALLY TRANSMITTED DISEASE | *T. pallidum*, *N. gonorrhoea*  
| PERITONITIS          | *Strept. pneumoniae*  

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*Note:* 'Sterile' pus: Mycobacteria

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*Note:* *Strept.* indicates *Streptococcus*. *E. coli* includes *Escherichia coli*. *Salmonella* includes *Salmonella*. *Trichomonas vaginalis* includes *Trichomonas vaginalis*. *Candida albicans* includes *Candida albicans*. Some pathogens are listed multiple times due to their occurrence in different lesions.