Infectious Diseases in Children

A Clinical Guide for Nurses

Tara Walker

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a clinical guide for nurses

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Infectious Diseases in Children

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Melbourne

Australasian Health Education Systems Pty Ltd
(ACN 005 611 626)
trading as
Ausmed Publications
277 Mount Alexander Road
Ascot Vale, Victoria 3032, Australia

First published April 2001
Reprinted March 2002

Further copies of this book and of all other Ausmed publications are available from the Distribution Manager, Ausmed Publications, PO Box 4086, Melbourne University, Victoria 3010, Australia.
Telephone +613/(03) 9375 7311.
Fax +613/(03) 9375 7299.
E-mail ausmed@ausmed.com.au
Home page www.ausmed.com.au

National Library of Australia Cataloguing-in-Publication data:

Walker, Tara.
Infectious diseases in children : a clinical guide for nurses.

Bibliography.
Includes index.
ISBN 0 9577988 7 3.

1. Communicable diseases in children - Nursing. I. Title.

610.73699

Edited by Robyn Whiteley, The WC Company Pty Ltd
Design, typesetting and printing by Hyde Park Press, 5 Deacon Avenue, Richmond, South Australia 5033, telephone (08) 8234 2044, fax (08) 8234 1887, e-mail hpp@olis.net.au

Text set in 11/14 Slimbach

Cover: The drawing on the cover is by Rosanna Moseley, who was 10 at the time. It emphasises the importance of handwashing when caring for children with infectious diseases.

FOREWORD

This text is a valuable addition to the literature available to clinicians concerned with the care of children. By focusing on the essential aspects of the care of children with infectious diseases through a concise, reader-friendly and easy-to-use approach the book will be a valuable clinical companion. Moreover, it will be a great resource for parents in the home who are faced with the management of their children's infectious diseases.

I commend this book as a resource in the care of children both in hospitals and in the home.

Dr Stephen Kermode
Associate Professor
School of Nursing and Health Care Practices
Southern Cross University
Lismore, New South Wales

DEDICATION

This book is dedicated to my paediatric colleagues —
those I know and those whom I have not met yet.

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PREFACE

There is a worldwide rise in infectious diseases in children. This has probably occurred because of low immunisation rates, disease outbreaks in children too young to be immunised, outbreaks of disease in children previously immunised, some vaccinated children not developing immunity to subsequent immunisations and waning immunity. Whatever the reasons, the fact remains that nurses are being confronted more and more by the need to care for children with infectious diseases.

I have written this book because there are no nursing resources available to nurses working in ward areas about the care needed for children with infectious diseases. Texts that are available are largely aimed at medical care, not nursing care; they are very costly and not user friendly for nurses.

I have had an interest in paediatric infectious diseases for a long time. By assembling this book, I believe I have not only increased my own knowledge, I have also fulfilled a professional responsibility to provide the paediatric nursing profession with a user-friendly resource book of clinical information about common infectious paediatric illnesses. I hope I have provided a foundation to assist paediatric nurses in their own quest for knowledge.

When I was researching this book, I discovered that it is vital to understand the basic infectious process so that is identified in the first chapter of this book. The infectious diseases are then presented in alphabetical order.

While I was working as a registered nurse I found that it was not easy to educate families about how to care for a child with an infectious disease while that child was at home so I have included in this book sections called 'Education for Carers'. If nurses are able to communicate to families what is in these sections they will be contributing more than adequately to the seamless care pathway that we strive to provide for paediatric patients from admission to discharge and beyond.

I hope you enjoy reading this book as much as I enjoyed researching it. May you find it a real help in your paediatric nursing.

Tara Walker

1. INTRODUCTION

The nature of infection

An infection is an invasion of the body by pathogens. A pathogen is a microorganism that has the potential to cause disease. If the microorganisms do not cause serious insult to the body's cells, the infection is asymptomatic. Disease results if the pathogens multiply and cause change in normal body tissue functioning. If the infectious disease can be transmitted from one person to another, it is called a communicable or contagious disease (Potter and Perry, 1997).

The chain of infection

A child exposed to a pathogen does not necessarily develop the disease. For the disease to progress, it is dependent on a cycle that contains the following elements:

1. *Infectious agent*
An infectious agent or pathogen must be present for the disease to progress. For example, *Neisseria meningitidis* is the bacteria that can cause bacterial meningitis.

2. *Reservoir*
The pathogen needs a reservoir (a home) to live in so it can survive. If the conditions are sustainable, it will multiply.

3. *Portal of exit*
After the pathogen has grown and multiplied, it must find a way out (portal of exit) to enter a new host and cause disease. The pathogen can enter the new host by a variety of means, most commonly through:

- the skin and mucous membranes
- the respiratory, urinary, gastrointestinal and reproductive tracts
- the blood.

4. *Modes of transmission*

The most common modes of transmission from reservoir to host are summarised in Table 1.1.

Table 1.1 ***Modes of transmission of infection***

Routes and means	Examples of organisms
CONTACT	
1. Direct Person-to-person (faecal–oral) or physical contact between source and potential host (touching the person)	Hepatitis A, *shigella,* Herpes simplex
2. Indirect Personal contact of susceptible host with contaminated object, e.g. needles, dressings	Hepatitis B, Respiratory syncytial virus
3. Droplet Large particles that travel over distance and make contact with the susceptible host, e.g. through coughing, sneezing, talking, spitting etc.	Measles, influenza, rubella
AIR	
Droplet nuclei, residue or evaporated droplets that are suspended in air because of coughing, sneezing etc. or are carried on dust particles	*Mycobacterium tuberculosis,* chickenpox, *Aspergillus*
VEHICLES	
1. Contaminated items Water Blood Drugs, solutions	 *Vibrio cholerae* Hepatitis C *Pseudomonas*
2. Food (not properly stored, cooked, thawed)	Salmonella, *Escherichia coli,* *Clostridium botulinum*
VECTOR	
1. External mechanical transfer	*Vibrio cholerae*
2. Internal transmission (insects) Mosquitoes Lice Fleas	 Malaria *Rickettsia typhi* Plague

5. Portal of entry
Organisms can enter the body by the same routes they use for exiting the body, i.e. through:
- the skin and mucous membranes
- the respiratory, urinary, gastrointestinal and reproductive tracts
- the blood.

6. Susceptible host
Only people susceptible to a pathogen entering their body will develop a disease. The more virulent a microorganism, the more susceptible the person is.

Once the person has been infected by the pathogen, the infectious process will happen in phases (Wong, 1993).

These phases include:

1. *The incubation period* — the time between the pathogen entering the body and the onset of first symptoms

2. *The prodromal stage* — the time when nonspecific signs (malaise, fevers, lethargy) become more specific. The pathogen is growing and multiplying and the infected person is more capable of spreading the disease to others.

3. *The illness stage* — the person develops symptoms more specific to the illness. In children, there is an observational scale (see Table 1.2) for the prediction of serious illness that is bacterial or nonbacterial in origin (Potter and Perry, 1997).

4. *The convalescence stage* — the acute symptoms disappear and the person moves into a recovery stage.

Control of transmission

To control disease transmission effectively, nursing staff must have a sound knowledge of:
- infection control principles
- modes of transmission
- how to prevent the spread of infection.

Table 1.2 ***Observational scale for the prediction of serious illness in children***

Observation item	Normal	Moderate impairment	Severe impairment
Quality of cry	Strong with normal tone or content, not crying	Whimpering or sobbing	Weak, moaning or high-pitched continual cry
Reaction to parent	Cries briefly, then is content or does not cry	Cries on and off	Poor response to stimulation
State variation	If awake, stays awake or wakes up quickly	Wakes only with prolonged stimulation	Falls asleep, not rousable
Colour	Pink	Pale extremities, acrocyanosis	Pale, cyanotic, ashen, mottled
Hydration	Skin and eyes normal, moist mucous membranes	Skin and eyes normal, mouth dry	Skin doughy, tented, dry mucosa and sunken eyes
Social interaction	Smiles and is alert (if older than two months)	Brief smile, less alert	No smile, dull response, minimal alertness

(Adapted from Behrman et al., 1992)

Children should not share baths, cups, spoons or toys.

To minimise transmission of microorganisms through indirect contact, contaminated equipment must not come into contact with a nurse's uniform.

The most important way of preventing the spread of infection is by handwashing. An effective handwash is a thorough cleaning of the hands with a recommended antibacterial soap. The aim is to remove contamination and pathogens from the hands and, over a period of time, to reduce bacterial counts (Potter and Perry, 1997). It is recommended that nurses wash their hands after working in the following situations:

- when the hands are visibly contaminated
- before and after contact with infectious children
- after contact with the source of the pathogen (blood and body fluids, membranes or infected objects)
- before assisting in procedures
- after gloves have been removed following a procedure of any type.

Detection of infection and prevention of transmission also include (Potter and Perry, 1997):

- wearing gowns, masks, gloves and goggles
- disposing of contaminated linen in the correct method
- wearing masks and gowns in public areas, e.g. in lifts or corridors while transporting infectious children to other areas of the health agency (x-ray, theatres).

Some children, especially those who have infections transmitted by small-particle aerosols, need to be nursed in isolation rooms. An isolation room should have a negative pressure so that air moves to the outside as new air enters the room from a duct. The sink and isolation supplies should be accessible within the isolation room.

Infections spread by large-particle aerosols can be contained by bedside isolation. Isolation technique is important to prevent the spread of infection when caring for children with an infectious disease (Behrman et al., 1992). Nurses working with children who have infectious diseases should familiarise themselves with their agency's policies on managing specific paediatric infectious diseases.

2. BACTERIAL MENINGITIS

Prior to discussing bacterial meningitis in children, a review needs to be done on the way a child's nervous system differs from that of an adult.

Paediatric differences in the central nervous system

Wong (1993) points to a series of differences in the nervous systems of children and adults.

- The brain of a term infant is two-thirds the weight of an adult brain. By the time the child is one year old, the brain weighs 80 per cent of an adult brain and by age six, it weighs approximately 90 per cent as much as an adult brain.

- An infant has 50 millilitres of cerebrospinal fluid while an adult has 150 millilitres.

- The peripheral nerves are not completely myelinated at birth. As myelination progresses, so does the child's fine muscle and motor coordination.

- Papilloedema is rare in children because of the open fontanelles and sutures that can expand with raised intercranial pressure.

- The primitive reflexes of the Moro, grasp and rooting (all present at birth) have gone by the time the child is one year old. These reflexes may reappear with neurologic disease.

BACTERIAL MENINGITIS

Meningicoccal disease is an important cause of morbidity and mortality in children (Laboratory Centre for Disease Control, 1994). The term 'meningitis' refers to the inflammatory response caused by an infection of the meninges of the brain and the cerebrospinal fluid (CSF) (Potter and

Perry, 1997). 'Meningismus' refers to the signs associated with meningeal irritation when meningitis is absent. The closeness of the brain to the leptomeninges and the fact that the inflammatory response happens in a very limited space makes meningitis life threatening, with significant mortality and long-term complications. Quick diagnosis and drug intervention save lives. Meningitis is considered a medical emergency as it is one of the only diseases in the Western world that can kill an infant within hours of the first onset of symptoms (Glennie, 1998).

E Etiology of meningitis

The primary bacterial organisms responsible for causing bacterial meningitis can vary according to age. With children aged two months to twelve years, three pathogens appear to be the most prevalent. These pathogens are:

- *Haemophilus influenzae* (Type B)
- *Neisseria meningitidis*
- *Streptococcus pneumoniae*

The types of meningitis that often start as an infection include otitis media, sinusitis, pharyngitis or pneumonia. The infection then migrates into the CSF. The pathogens that commonly cause meningitis in neonates are *Escherichia coli* and group B streptococci (Wong, 1993).

Pathogens may enter the body as a result of an injury when the skin is broken, allowing the pathogen access to the sinuses and the CSF. The pathogen may enter through:

- a lumbar puncture site
- skull fracture
- surgery.

Meningicoccal meningitis caused by *Neisseria* usually occurs in older children and adolescents. As it is transmitted by droplet, the risk increases with the number of social contacts the child has. Sometimes meningitis is caused by protozoal and fungal infections. This is most common in children who have Acquired Immune Deficiency Syndrome (AIDS) (Ashwill and Droske, 1997).

P Pathophysiology of meningitis

The meninges of the brain become inflamed as the body's immune system sets up a toxic response to the pathogen that is invading the brain. As the process continues, intercranial pressure rises; so does subdural empyema. If the infection reaches the ventricles, oedema and tissue scarring around the ventricles result in an obstruction of the CSF, causing hydrocephalus.

The toxic process can happen quickly because the CSF is an excellent medium for bacteria to thrive in since it contains nutrients (protein and glucose) for the bacteria to feed on. Leucocytes are not able to provide a defence for the CSF because it is a liquid environment (leucocytes need a tissue environment to enable them to destroy bacteria) so there is little defence and the invading bacteria can multiply quickly.

As the infection spreads further into the brain, changes happen in the permeability of the capillaries and blood vessels in the dura mata. These changes increase the passage of albumin and water into the subdural space. Protein and fluid collect, causing an increase in intercranial pressure.

The most common neurologic consequences of meningitis, according to Jenkinson and Edwards (1998), are:
- hearing and sight deficits
- mental retardation
- seizures
- behavioural problems.

Other complications can include:
- cranial nerve dysfunctions
- brain abscesses
- syndrome of secretion of inappropriate antidiuretic hormone (SIADH).

The incidence of meningitis

Meningitis most commonly affects children aged between one month and five years, but a secondary peak has been noted in adolescents aged 15–19 years (Jenkinson and Edwards, 1998). Boys are infected more often than girls and risk factors increase when individuals are in close contact

with each other (day care, preschools and communal living). The incidence of *H. influenzae* Type B-related meningitis has declined since the immunisation of infants was commenced (Ashwill and Droske, 1997).

C Clinical presentation of meningitis

Early recognition of symptoms is vital. The symptoms may occur in any order and depend on the age of the child and the duration of any preceding illness (Glennie, 1998). In young children and babies, symptoms are usually vague and nonspecific (Wong, 1993).

Table 2.1 ***Clinical signs of meningitis***

Neonates	Young children	Children and adolescents
Poor feeding and sucking Floppy muscle tone Blotchy or pale skin Vomiting Diarrhoea A shrill, moaning cry Hypo/hyperthermia Apnoea Sepsis Seizures Disseminated intravascular coagulation	Fever Vomiting Irritability Poor feeding Seizures High-pitched cry Bulging fontanelle	Severe headache Photophobia Nuchal rigidity Fever Altered level of consciousness Loss of appetite Diarrhoea Vomiting Muscle/joint pain Purpura Kernig's sign (pain with extension of leg and knee) Brudzinski's sign (flexion of head causes flexion of hips and knees) Seizures (late sign)

(Adapted from Wong, 1993)

The petechial rash associated with meningitis can be identified by pressing a glass against the rash. The rash will not fade and can be seen through the glass. Medical advice is required immediately. In the early

phase of the disease the rash may be maculopapular and fade under pressure but it usually progresses to become a nonblanching petechial or pupuric rash. Good lighting is needed to examine children for rashes emerging on the body. Initially, the rash appears like tiny red pinpricks. It then progresses to purple blotches that resemble bruises or blood blisters. A quickly progressing rash indicates poor prognosis. The rash can be difficult to identify on dark skin so children must be examined carefully. Pay attention to the soles of the feet, palms of the hand, palate and conjunctiva (Glennie, 1998).

Nurses assessing children must be aware that information they collect will provide baseline data for ongoing assessment so it is vital they perform an accurate physical assessment and obtain a complete history of the illness.

Table 2.2 ***Performing a physical assessment and history taking***

History	Observation
Headaches Personality changes and increased irritability Interest levels in fluid and food Nausea and vomiting Hydration status Any recent illnesses, especially • respiratory tract infections • otitis media • surgery • skull fractures • a previous lumbar puncture	Photophobia Hearing loss Seizure activity (this can be as minor as a twitching eyelid or 'staring' episodes) Changes in the level of consciousness Changes in pupil reaction and size Nuchal rigidity Any changes in muscle tone

(Adapted from Potter and Perry, 1997)

The incubation period of meningitis

Haemophilus influenzae

- Incubation period: 2–4 days
- Communicability: until 24–48 hours after starting effective antibiotic treatment
- Spread by droplets and nasal discharge

Neisseria meningitidis

- Incubation period: 2–10 days, usually 3–4 days
- Communicability: until 24 hours after commencing effective antibiotic treatment
- Carried in the nasopharynx and droplet spread from the respiratory tract (Davies, 1996)

D Diagnosis of meningitis

Most people who have bacterial meningitis have leucocytosis with an elevated white cell count. Other laboratory findings will depend on the duration and severity of the illness. Many children become dehydrated from diarrhoea, fevers and vomiting and may develop metabolic acidosis. The urinalysis may show proteinuria from renal damage which may result from shock and slowed renal perfusion. The urine specific gravity may be elevated as the children become dehydrated (Herf et al., 1998).

A lumbar puncture (LP) is necessary to collect a specimen of CSF. CSF is considered an accurate method of detecting infection in the central nervous system. The contraindications for doing a lumbar puncture are:

- evidence of increased intracranial pressure other than a bulging fontanelle
- acute cardiopulmonary compromise where resuscitation has been required
- where the positioning of children for LP would exacerbate cardiopulmonary function
- infection of the skin over the LP site
- thrombocytopenia is a relative contraindication for immediate LP.

LP is indicated in children who shows signs of disseminated intravascular coagulation or petechiae. LP may be delayed in immunosupressed children with chronic thrombocytopenia until platelets have been transfused. If LP is delayed for any of these reasons, it is important to commence empiric therapy against the potential causative microorganism (Buchanan and Witt, 1996).

The CSF may be normal in the early phase of infection or at the other end of the disease, when the meningitis has become so overwhelming that the host cannot respond with an immune response. In many cases, the CSF

contains white blood cells with more than 90 per cent segmented neutrophils, elevated protein levels and decreased glucose levels. CSF may be cloudy on collection (Herf et al., 1998).

A Gram stain identifies the causative organism in most cases. It is necessary to collect blood, CSF and nasopharyngeal cultures in suspected cases. Scrapings from skin lesions can also be cultured. Definitive diagnosis is based on identification and isolation of the pathogen from blood or CSF (Herf et al., 1998).

Some neurologic events related to the meningitis may require computerised tomography (CT) and include:

- focal neurologic deficits
- prolonged fevers after commencement of therapy
- increased head circumference.

Sometimes findings are suggestive of an intercranial mass rather than meningitis. In these instances, a lumbar puncture is not recommended for children until a CT can be done. The suggestive findings may be papilloedema and gradual onset of symptoms rather than an acute onset. Suspicion is higher in adolescents (especially those who have had sinusitis followed by a gradual worsening of intracranial symptoms) than for babies and toddlers (Rudolph and Hoffman, 1987).

M Management of meningitis

As mentioned previously, meningitis is a medical emergency. Isolation of children suspected of having meningitis is initiated and must continue for a minimum of 24 hours after antibiotics have commenced (Bolyard et al., 1998). Prompt initiation and uninterrupted administration of intravenous antibiotics is vital if children are suspected of having bacterial meningitis. Treatment must be started before the pathogen has been identified because cultures can take several days for a pathogen to be identified. To wait this long to commence therapy could be fatal. Antibiotic therapy must be based on the child's age, the most frequently found pathogen for the age group and the initial appearance of the CSF. Children's veins can be difficult to cannulate, so the intramuscular route should be used to give antibiotic cover if cannulation is not possible (Ashwill and Droske, 1997).

Treatment for neonatal bacterial meningitis consists of ampicillin and an aminoglycoside or a third-generation cephalosporin. For older children and adolescents, ampicillin and penicillin G is recommended. Initially, broad-spectrum antibiotics should be used to cover the most likely pathogens. Once the culture and sensitivity reports are available, drug therapy can be modified. Four days of dexamethasone is recommended in infants and children over two months of age (Herf et al., 1998).

Supportive management may be required for septic shock, a complication of meningitis. Repeated medical and neurological assessments of children with meningitis are required to enable health care providers to detect cardiovascular, CNS and metabolic complications. Continuous monitoring is required, including vital signs (pulse rate, respiratory rate and blood pressure), urine output and peripheral perfusion. There may need to be volume resuscitation and the use of pressor drugs (dopamine). Neurologic assessment must cover pupillary reflexes, level of consciousness, motor strength and cranial nerve signs. Nurses should be aware of the signs of raised intracranial pressure and seizures, especially in the initial 72 hours when children are at the highest risk of developing neurologic complications (Herf et al., 1998). Children should be kept nil by mouth, and intravenous fluids should run at a half to a third maintenance during the acute phase of the illness (Wong, 1993).

Complications and prognosis of meningitis

Table 2.3 ***Complications of meningitis***

Common	Uncommon
Cerebral oedema Shock Subdural effusion Seizures and brain infarction and/or necrosis Inappropriate ADH secretion Deafness (hearing loss usually occurs early in the disease and hearing tests should be done on children routinely once they are well)	Obstructive hydrocephalus Subdural empyema Brain abscesses Disseminated intravascular coagulation

(Adapted from Rudolph and Hoffman, 1987)

According to Rudolph (1992), the factors that influence outcome are based on:

- age (mortality is highest in babies less than one year old)
- the infective pathogen
- how long a child has been untreated before therapeutic intervention.

Contact tracing and prophylactic treatment of meningitis

N. meningitidis is situated in the nasopharynx of the carrier and patient. The organism is spread by direct contact, mainly by respiratory droplet. The spread of the disease can be controlled by giving prophylactic antibiotics to people who have been in contact with the children. Deciding who qualifies as a 'close contact' can cause confusion among health care providers. The Laboratory Centre for Disease Control (1994) defines 'close contact' as:

> those residing with the index person or spending 4 or more hours with the index case for 5 of 7 days before the onset of illness.

Those at risk of becoming infected include:

- all household members
- day care and/or preschool contacts.

Health care providers are considered to be at risk if they have close contact with the patients' secretions, e.g. giving mouth-to-mouth resuscitation before the commencement of antibiotic therapy (Herf et al., 1998).

The risk of developing the infection is greatest during the first few days after exposure, so contacts should commence antibiotics as soon as possible, ideally within 24 hours of exposure. Rifampicin is the drug of choice. It is considered a safe and effective way of eliminating *N. meningitidis* from the nasopharynx. Rifampicin comes in capsules and suspension for ease of administration. The most frequently reported side effects are nausea, vomiting, diarrhoea, cramps and epigastric pain. The contacts need to be advised that rifampicin will turn their urine, saliva, perspiration and tears orange/red. Contact lenses may be come discoloured so should not be worn. Rifampicin is not recommended for pregnant women, ceftriaxone can be used instead. Rifampicin can interfere with oral contraceptives so alternative methods of contraception should be used (Herf et al., 1998).

Wide use of antibiotics in those who have not had close contact with the child has not been proven to prevent meningitis so it is not recommended. Health care providers may be pressured by anxious families who consider their children have had close contact with the infected child. Information on the spread of meningitis and the risks and benefits of prophylaxis can assist care providers and families make informed decisions about the management of meningitis (Jenkinson and Edwards, 1998).

If an outbreak occurs, communication with the public, parents and teachers should be kept to a high standard, with one communicator giving up-to-date information about the outbreak and management of the disease (Jenkinson and Edwards, 1998).

3. CONJUNCTIVITIS

The term 'conjunctivitis' refers to a broad group of conditions presenting as inflammation of the conjunctiva. The inflammation can be hyperacute, acute or chronic in presentation and infectious or noninfectious in origin. Conjunctivitis is the most common cause of red eye (Morrow and Abbott, 1998). Red eye is defined by Ruppert (1996) as

> a conjunctival blood vessel infection that results in small blood vessel rupture, causing bleeding into the schlera.

Causes of conjunctivitis

Conjunctivitis is most often caused by a bacterial or viral infection. Sexually transmitted diseases such as gonorrhoea and chlamydia are less common causes but they are becoming more prevalent and are important to recognise because of their significant systemic and ocular implications (Ruppert, 1996).

History and physical examination

A differential diagnosis for the chief complaint of paediatric conjunctivitis can include:

- infection (bacterial, viral or herpetic)
- foreign body/corneal abrasion
- allergic conjunctivitis
- systemic causes.

Evaluation of conjunctivitis includes:

Obtaining a detailed history

The history should contain the following:

- onset of conjunctivitis, including any precipitating events, then progression and duration of the symptoms
- physical data concerning pain, photophobia, itching, tearing, drainage, eye movements, visual disturbances, history of red-eye episodes and treatment and whether the red eye affects one or both eyes

- all allergies
- any associated symptoms such as fever, cough, nasal discharge
- information such as exposure to other children/adults at home, school or day care with similar symptoms.

Physical examination

Physical examination should always include a thorough examination of the external structures of the eye, including the eyelids, lashes, cornea, pupil and bulbar palpebral conjunctivae. Instillation of dye and a penlight may be necessary for examining the cornea (Ruppert, 1996).

Ability to move the eye should be assessed as well as peripheral vision. Visual acuity can be assessed using a paediatric Snellen chart. An ophthalmic examination should be done to exclude damage to the retina of the eye (Ruppert, 1996).

Laboratory tests

Diagnostic and laboratory data will confirm the etiology. Purulent drainage from the eye will require both viral and bacterial culturing and sensitivity determination to be carried out on it (Adams et al., 1996).

When evaluating red eye, the extent of the redness should be noted and documented. Outlining to distinguish the affected area is a good practice so treatment can be evaluated. Infection may be limited to the conjunctivae or may extend to the ciliary area. A purple zone of infection around the cornea indicates a ciliary infection(Ruppert, 1996).

Table 3.1 ***Different causes and presenting symptoms of conjunctivitis***

Etiology	Serous	Mucoid	Mucoprurulent	Purulent
Viral	+	–	–	–
Chlamidial	–	+	+	–
Bacterial	–	–	+	+
Allergic	+	+	–	–
Toxic	+	+	+	–

+ = present – = absent

(Adapted from Morrow and Abbott, 1998)

BACTERIAL INFECTION

The colour and consistency of discharge can differentiate whether the conjunctivitis is viral or bacterial. Bacterial infections usually present with thick green or yellow discharge but the discharge seen with viral conjunctivitis is nonpurulent. Multiple organisms, including *Pneumococcus, Streptococcus pneumoniae, Staphylococcus aureus, Haemophilus* and *Meningicoccus,* can cause bacterial conjunctivitis. History usually reveals bilateral involvement with thick, mucopurulent discharge that causes crusting of the lids. Children may complain when they wake up that their eyes are stuck together. Onset may begin in one eye followed by the other eye in 2–5 days (Potter and Perry, 1997).

D Diagnosis of bacterial conjunctivitis

The nurse can identify the organism and antibiotic sensitivity by swabbing the conjunctiva and then requesting a culture and sensitivity from the laboratory. Both eyes should be swabbed even if there is only one eye affected. The culture of the supposed unaffected eye provides information about the normal flora and can detect autoinoculation of that eye if it has occurred. A conjunctival scraping may also be done if diagnosis is uncertain or if children do not respond to therapy. Leucocytes are found in bacterial infections (Ruppert, 1996).

M Management of bacterial conjunctivitis

Although this condition is usually self-limiting, bacterial conjunctivitis is often treated with topical antibiotic agents. Eye drops can be used in older children but ointment will be easier in younger children. The affected eye should never be patched (Ashwill and Droske, 1997).

Although culture and sensitivity can be used in determining the most effective treatment agent, the costs of these tests may not be warranted in clinical practice. Choice is often based on cost, ease of use, and spectrum of action (Morrow and Abbott, 1998).

- Sodium sulfacetamide is relatively cheap but can burn and sting on administration as well as cause hypersensitivity.

- Erythromycin ointment is cheap and well tolerated but is not effective against some Gram negative enterics.
- Gentamicin and tobramicin are more expensive but have a broad spectrum action including Gram negative enterics. Prolonged treatment of streptococcal conjunctivitis with these drugs can cause corneal ulceration or epithelial toxicity.
- Combination antibiotic cover has been shown to be effective in treating bacterial conjunctivitis with a low incidence of side effects.

Cool compresses may provide some relief. Eyelids should be washed daily with a mild soap such as baby shampoo to remove crusting (Wong, 1993).

There is an increase of *Neisseria gonorrhoeae* as a cause of conjunctivitis in sexually active adolescents and sexually abused children. Diagnosis is based on the presence of a particularly purulent conjunctivitis and purulent genital discharge. While swabbing the conjunctiva for specific diagnosis can be done, treatment should not be delayed. Children should be referred to an ophthalmologist to check for ocular damage, and systemic treatment is needed to prevent ulcerative keratitis (Ashwill and Droske, 1997). If sexual abuse is of concern, the appropriate referrals need to be made for the child's protection.

Infection with *Chlamydia trachomatis* must be considered. Diagnosis can be made by a Gram stain of epithelial scrapings or by antibody testing of the eye discharge. If the cause is chlamydia, topical and oral antibiotics will be needed (Wong, 1993).

VIRAL INFECTION

Up to 20 per cent of acute conjunctivitis presentations are caused by viruses. The main virus that causes conjunctivitis is adenovirus and usually affects children who are younger than ten. Viral infections are highly contagious and may be spread by an upper respiratory infection or by direct contact with an infected individual. This form of conjunctivitis is common in the summer months by cross contamination in swimming pools. Pharyngitis and fever are often associated with red eye (Ruppert, 1996).

Presentation of viral conjunctivitis

Onset of viral conjunctivitis is abrupt and usually affects one eye initially with the other becoming infected in one to two days by autoinoculation. Associated symptoms are cold-like in origin as well as tearing and watering of the eyes and coughing and sneezing. Eye drainage is not offensive and a full history is necessary to make a definite diagnosis.

D Diagnosis of viral conjunctivitis

On examination there will be conjunctival anomalies which may cause a decrease in vision. Children may have tender preauricular nodes and a conjunctival scraping may be done to confirm a specific virus. Mononuclear cells are found if the infection is viral (Morrow and Abbott, 1998).

Other viruses that can cause conjunctivitis in the paediatric population are:
- epidemic keratoconjunctivitis (EKC)
- conjunctivitis/otitis media syndrome
- herpetic infection.

EKC is highly contagious, caused by adenovirus and more common in the autumn. EKC manifests with all the previously discussed symptoms. A large percentage of children develop keratitis and will experience photophobia and foreign body sensation. Corneal opacification and inflammation may develop and an ophthalmologist referral is required if this occurs (Ashwill and Droske, 1997).

Conjunctivitis/otitis media syndrome may be caused by several viruses. Symptoms include irritability, cough, mucopurulent discharge and low-grade fevers. Eyes are swollen and crusty on awakening. Eye and ear symptoms will occur simultaneously. Subsequently, a bacterial otitis media will occur and this will need antibiotic cover (Ruppert, 1996).

Infection with herpes simplex virus should be considered with any presentation of acute conjunctivitis with watery discharge. It is more common in children aged one to five years and they are usually infected by a person who has active lesions on the skin or mucous membranes. Eye involvement is usually unilateral. Vesicular lesions of the eyelid and corneal lesions are distinguishing features of herpetic conjunctivitis.

Definitive diagnosis can be made by viral cultures and by identifying specific herpes antigens. Herpetic infections need ophthalmological review because a severe infection can damage sight (Morrow and Abbott, 1998).

M Management of viral conjunctivitis

Viral conjunctivitis is usually self-limiting. Symptoms may continue for up to two weeks. Children should avoid direct contact with others for a week after the onset of symptoms. The same infection control precautions are required for viral infections as bacterial. It is recommended a child with herpetic conjunctivitis is treated with antiviral medications. The child needs to be reviewed in one week to ensure no epithelialisation has occurred. Steroids should be avoided as they can cause uncontrolled virus proliferation and can place the child at risk of steroid-induced complications such as glaucoma and cataracts (Wong, 1993).

EDUCATION FOR CARERS OF CHILDREN WITH VIRAL CONJUNCTIVITIS

Teaching should include:

- Thorough hand washing is required after touching the eye area to avoid spread of infection to the other eye or other people.
- The child's towel and washer should not be kept with other family members' towels.
- Bottles and eye droppers should not be used for another person. The top of the dropper should not touch the child's eye when drops are being instilled (Ashwill and Droske, 1997).
- Young girls should be told to discard any eye make-up to prevent cross infection or reinfection.
- Young children should be kept out of school or day care until the discharge improves and they have had 24 hours of eye drops. Complete recovery should be within one week (Ruppert, 1996).

Preventing injury

Minimising injury from rubbing the eye is also important (Ashwill and Droske, 1997).

- Mittens may be used for infants.
- Distraction and reminding are recommended for infants and toddlers.
- Contact lenses should not be worn until the infection has cleared. Buying new contacts will reduce the risk of reinfection from the old contaminated lenses and reduce the risk of corneal ulceration.

4. ENCEPHALITIS

Encephalitis affects all age groups. It is an inflammation of the brain as a result of a viral illness or central nervous system infection. It may be a result of measles, chickenpox, rubella or immunisations. Diagnosis can be made with accuracy only by the microscopic examination of brain tissue on the deceased person. In clinical practice, diagnosis of encephalitis relies on:

- clinical symptoms
- neurologic presentation
- epidemiologic information (if there is no histologic material to examine).

When neurologic symptoms suggest encephalitis but the brain is not inflamed (i.e. Reye syndrome), the condition is termed 'encephalopathy' (Aswill and Droske, 1997).

P Pathophysiology of encephalitis

A toxin or infectious pathogen invades the body, generally via the lymphatic system. The body produces an inflammatory response. At this stage, a febrile illness is present. If further viral reproduction occurs, a secondary propagation may take place, resulting in large amounts of the virus.

The central nervous system may become infected as the pathogen travels in the blood and peripheral nerve pathways. The resulting inflammation produces cerebral oedema, cellular damage and neurologic dysfunction.

Neurologic damage is caused by a direct invasion and destruction of the neural tissues by the invading virus or by a reaction of nervous tissue to the antigens of the virus (Wong, 1993).

E Etiology of encephalitis

Encephalitis is usually associated with a preceding illness.

- Viral illnesses such as herpes simplex, rabies, measles, chickenpox, mumps and rubella have all been known to cause encephalitis.

- Equine encephalitis, carried by mosquitoes and known to affect horses, has been implicated but herpes simplex 1 is the most common cause of encephalitis in the neonatal period.
- Other infections, such as meningitis, have been associated with encephalitis. Toxic encephalitis can also result from hyperbilirubinaemia or exposure to toxins, i.e. lead poisoning (Ashwill and Droske, 1997).

C Clinical presentation of encephalitis

The signs and symptoms of encephalitis vary with the causative pathogen and the area of the brain that is affected. Some children are only mildly affected initially then go into a coma and die suddenly. Other children may have high fevers, seizures, convulsions and or hallucinations with brief episodes of rationality and recover with no side effects (Behrman et al., 1992).

- Encephalitis usually starts as an undifferentiated acute systemic illness with children screaming and having abdominal pain, nausea and vomiting.
- As the temperature rises, there may be a mental dullness resulting in a stupor or bizarre movements, seizures and nuchal rigidity, though these will not be as noticeable as in the case of meningitis.
- Focal neurologic signs may fluctuate and there may be a loss of bladder and bowel control in toilet-trained children. Children may suffer unprovoked emotional distress and speech, visual and auditory disturbances (Behrman et al., 1992). These will be traumatic for them and they will become scared, so reassurance is vital.

Specific types of encephalitis include (Wong, 1993):
- Guillain Barre syndrome
- transverse myelitis
- acute hemiplegia
- acute cerebellar ataxia.

D Diagnosis of encephalitis

Careful history taking is essential. The history should include (Ashwill and Droske, 1997:

- contacts with illnesses in the last two or three weeks, including any sick animals
- contact with mosquitoes and ticks
- travel to remote areas as well as local travel (children who have recently travelled in exotic countries may present with bizarre signs and symptoms from encephalitis so any overseas travel should be queried)
- exposure to biologic substances, heavy metals, pesticides and poisonous chemicals.

LP needs to be carried out and the CSF examined to exclude other disorders that may respond to a specific management plan. Cultures and sensitivity testing are required from the CSF. In encephalitis, the CSF is generally clear and the leucocyte count ranges from none to several thousand. Blood, stool and throat swabs may need to be taken to exclude all possibilities. Other conditions that may need to be excluded are fungal or protozoan infection; atypical cells may need cytopathologic examination to exclude neoplasms. Brain biopsies or serum antibody tests may be done if cultures are negative. CT and MRI (magnetic resonance imaging) may be done to exclude tumours and lesions. An EEG (electroencephalogram) may be helpful in diagnosis as generalised slowing of the variable degrees is common in encephalitis (Ashwill and Droske, 1997).

Children who have recently had measles, mumps or similar infections are at increased risk of developing encephalitis, and neurologic involvement may precede the development of other signs of encephalitis. Also at risk are immunosupressed children (Behrman et al., 1992).

M Management of encephalitis

Treatment of encephalitis is supportive and based on symptoms. Acyclovir and a third generation cyclosporin may be administered until the cause of the encephalitis is known. Treatment is aimed at minimising the risk of raised intercranial pressure. Anticonvulsant medications may also be given (Adams et al., 1996).

NURSING CARE FOR CHILDREN WITH ENCEPHALITIS

Hospitalisation is required and children who are admitted will need continuous monitoring for (Adams et al., 1996):

- changes in their level of consciousness, behaviour, headaches, and nuchal rigidity
- alteration in pulse and respirations and pupillary responses
- seizure activity.

Body temperature should be maintained within the normal paediatric range. Fever increases cerebral metabolism and metabolic demands. Antipyretics and cool compresses can be used as well as having children in minimal clothing. If children are hyperthermic, a hypothermic blanket can be used but body temperature must be monitored regularly because the blanket can reduce the temperature quickly. Skin must be assessed because peripheral circulation is decreased with the used of cooling devices and there can be skin breakdown (Wong, 1993).

Fluid and electrolyte balances are important. Input and output must be monitored on a fluid balance chart. Children must be observed for signs of fluid overload such as:

- increased body weight
- electrolyte imbalances
- oedema
- changes in consciousness (Ashwill and Droske, 1997).

Children may have severe headaches and their pain management is important. A pain scale is useful to determine the extent and type of pain and the appropriate analgesia can then be administered. Distraction, rhythmic breathing and cutaneous stimulation can also be used to reduce pain. Reducing light and noise may assist in management of headaches (Potter and Perry, 1997).

Severe illness combined with a long recovery period and the worry of long-term neurologic deficits can be stressful for the family. Nurses must provide the appropriate care for the family during this stressful time. The family should be encouraged to express their concerns. Support from religious and social workers may assist the family in coping with the illness (Potter and Perry, 1997).

Prognosis

Once viruses reach the central nervous system, a number of factors make the brain more vulnerable:

- There is no lymphatic drainage.
- There are no lymph nodes in the central nervous system.
- The blood brain barrier normally slows the entry of humoral and cellular immune components.

The long-term effects of encephalitis are very individual and depend on what virus caused the encephalitis and what area of the brain was affected (Katz et al., 1998).

5. EPSTEIN BARR VIRUS (INFECTIOUS MONONUCLEOSIS)

The earliest descriptions of the illness recognised today as infectious mononucleosis (IM) are based on the observations of Filatov, a Russian paediatrician who wrote about his clinical observations of the condition in 1885. Pfeiffer, a German physician, added to Filatov's observations in 1889. In 1920, Sprunt and Evans were the first to use the term 'infectious mononucleosis'. They observed the associated haematologic changes, including the presence of atypical lymphocytes. Paul and Bunnell continued this work in 1932, examining the heterophil antibody elevation associated with IM. These researchers' observations are what diagnosis is based on even today (Schaller and Counselman, 1995). IM is commonly known as 'glandular fever' (Ashwill and Droske, 1997).

E Etiology of Epstein Barr Virus

It was not until 1960 that Epstein Barr Virus (EBV) was identified as the causative agent of IM (Schaller and Counselman, 1995).

- The incubation period is four to seven weeks.
- The infectious period is not clear because the virus is shed prior to clinical onset of the disease.
- Asymptomatic carriers are common.
- EBV is transmitted by saliva, close contact and blood.
- The primary site of infection is the epithelial cells, parotid gland and B lymphocytes.

As well as being associated with IM, EBV has been related as a co-factor in the development of Burkitt's lymphoma and nasopharyngeal cancer (Mott et al., 1990).

C Clinical presentation of EBV

IM occurs in children who are otherwise healthy. Clinical signs can vary in severity from mild to fatal and include:

- fever
- exudative pharyngitis
- lymphadenopathy
- hepatosplenomegaly
- development of a maculopapular rash
- malaise
- fatigue
- nausea and abdominal pain.

Children will be unwell for about 2–4 weeks, and will recover slowly. The prognosis is good if there are no complications.

When children present at the health agency, all signs and symptoms should be noted. Assessment should include (Adams et al., 1996):

- a pharyngeal examination, with attention to redness and swelling of the throat
- noting of any rashes, with precise documentation as to the distribution and appearance of the rash
- palpation of the liver and spleen to check for enlargement
- a check of temperature and nutrition and hydration status.

Complications of EBV

There is a risk of:

- splenic rupture, most frequently in the second week of the illness — the rupture is related to trauma and can be as mild as palpation during examination
- the respiratory state being compromised if the swelling is severe in the tonsils and pharynx
- neurologic complications, which can include convulsions, ataxia, nuchal rigidity, meningitis, Bells palsy, transverse myelitis, encephalitis and Guillain Barre syndrome
- pneumonia and myocarditis — common but usually resolve
- hepatitis as the disease progresses.

The end result of these complications depends on how severe the virus is and the progress of the complications (Mott et al., 1990).

NURSING CARE OF CHILDREN WITH EBV

The illness is generally self-limiting and treatment is supportive. Complications will require treatment specific to the complaint. Steroids may be used in the management of tonsillar swelling but will require monitoring because of the possibility of immunosupression. Acyclovir has been used but there have been no significant benefits found to substantiate its use. Antibiotics can be used if the throat infection is severe enough (Schaller and Counselman, 1995).

Children may need bed rest in the acute stage of the illness but when they are feeling well enough to return to normal activities, contact sport should be avoided until their spleen has returned to normal size. The family need to be reminded that recovery can be a slow process and fatigue may continue for a long time. The parents may need guidance as to child care if they both work outside the home. If children are not able to attend school for a long period of time and there is concern that they will fall behind in their school work, teachers may need to be asked to organise school work at home.

Nutrition and hydration must be monitored. If children are not hungry, snacks may be more beneficial than regular meals. If children are experiencing sore throats, milkshakes and jelly may be appropriate (Wong, 1993).

6. HELMINTHS

Helminth is a classification for worms that live in humans and exist as a parasite. Helminths infect more than half the people in the world. Helminths are considered a major contributor to the poorer quality of life for people in areas where living situations make it difficult to maintain community health. As travel to these countries and emigration from them to the developed world has become more common, there has been an increase in helminthic infections in the Western world (Mott et al., 1990).

The three groups that impact on children are tapeworm, flukes and roundworms (Anon., 1992).

Children are more likely to be infected than adults because they are more likely to put their hands in their mouths and be subject to faecal contamination. According to Adams et al. (1996) transmission may happen by:

- faecal–oral ingestion
- ingestion of contaminated tissue from another host
- skin penetration or the bite of a blood-sucking insect.

M Management of helminths

Treatment consists of giving infected children medication that is specifically for the helminth that has been isolated. The whole family should be treated. Enteric isolation and education of the family regarding personal hygiene and sanitary practices are also necessary (Adams et al., 1996).

The impact that anthelmintics can have on the health of the world, especially in developing countries, has been emphasised widely in health literature (see Mackenzie, 1993). There are discrepancies between the magnitude of these infections and the drugs used to combat them and chemotherapy is the most important tool for control of these infections. Significant problems encountered in the use of anthelmintics include adverse side effects and poor patient compliance. The emerging problem of helminth resistance to anthelmintic drugs is considered to be a potentially serious problem (Mackenzie, 1993).

Table 6.1 *An overview of common parasitic infections*

Parasite	Transmission	Manifestations	Diagnosis	Treatment
Roundworm *(Ascaris lumbricoides)*	Ingestion of eggs from contaminated soil or food, transfer to mouth from toys, fingers etc.	Abdominal pain, distension, pneumonitis, abdominal obstruction, vomiting with bile staining	Faecal smear	Mebendazole, Pyrantel pamoate
Pinworm *(Enterobius vermicularis)*	Ingestion or inhalation of eggs, transfer from hands to mouth	Nocturnal anal itching, insomnia	Scotch tape test, microscopic examination	Mebendazole, Pyrantel pamoate
Tapeworm *(Taenia saginata)*	Ingestion from handling or eating infected beef or pork	Asymptomatic, segments of worm seen in stool, abdominal pain, nausea, anorexia, weight loss, insomnia	Faecal smear, microscopic examination	Niclosamide
Hookworm *(Necator americanus)*	Skin penetration from direct contact with contaminated soil	Dermatitis, anaemia, blood loss, pneumonitis, malnutrition	Faecal smear, microscopic examination	Pyrantel pamoate

(Adapted from Anon., 1992)

NURSING CARE OF CHILDREN WITH A HELMINTH INFECTION

Nursing care of children with a parasitic infection includes collecting stool specimens for diagnosis by faecal smears. Stool specimens that have not been contaminated with urine are preferable but this can be difficult, especially with small children. Plastic wrap can be placed over the toilet bowl or a potty used. If children wear nappies, the specimen can be collected from that. Faecal specimens do not have to be collected in a sterile technique. The specimen is collected using a clean tongue depressor and putting faecal matter in the appropriate container with the depressor. The container should be marked with the child's name, the date and the time the sample was collected. The specimen should be placed in a pathology bag and taken as soon as possible to the laboratory (Wong, 1993).

Health care providers are at risk of contracting a helminthic infection from children by the faecal–oral route, usually as a result of poor handwashing after caring for children with diarrhoea or faecal incontinence. Transmission of parasites to staff, patients and visitors can also occur as a result of contaminated bathroom and toilet facilities. Nosocomial parasitic infection from food contamination by insects or an infected kitchen worker is possible. Other possible modes of transmission of parasites are by the faecal–subcutaneous route or by contaminated equipment such as scopes or rectal probes. Faecally contaminated instruments and equipment should be cleaned, and if possible, autoclaved, or should undergo ethylene oxide gas sterilisation (Lettau, 1991).

EDUCATION FOR CARERS OF CHILDREN WITH A HELMINTH INFECTION

Nursing staff are able to provide education to the family about management of children's helminthic infection. Carers should be informed of the necessity and rationale for evaluating and treating the whole family for infection. They need to be aware of the different modes of transmission and how to prevent cross infection with other family members (Wong, 1993).

Teaching the family about prevention is an important role for the nurse. Carers of children must understand good hygiene and health practices (Sinclair, 1997):

- Handwashing (including under the nails) with soap should be done before eating and after going to the toilet.
- Children should be dissuaded from putting their hands in their mouth or biting their nails.
- Only toilets that flush should be used for elimination. By using this method of waste disposal, contact with potentially infective body substances is minimised.
- Bathrooms should be cleaned with bleach (but parents should be educated about correct storage of harmful detergents).
- Scratching of the anal area with bare hands should be avoided.
- Pets (dogs and cats) should be kept away from children's play areas and sandboxes. Sandboxes should be covered when not in use.
- Vegetables and fruit should be washed before they are eaten.
- Nappies should be changed regularly and disposed of in a hygienic manner.
- Children should not go swimming in nappies.
- Water should be boiled if away from home (camping, caravanning).

7. HEPATITIS

Hepatitis is an acute or chronic inflammation of the liver. Hepatitis is caused by several different viruses, toxins and diseases. Even though the types of hepatitis are different, there are similarities in assessment evaluations and treatment. The most common causes of viral hepatitis are listed in Table 7.1. Rubella, cytomegalovirus, herpes simplex and Epstein Barr Virus can also cause hepatitis in children.

E Etiology of hepatitis

A large number of hepatitis A (HAV) outbreaks can be traced to childminding centres. The risk of HAV occurring in a day care centre increases with the number of children enrolled who are younger than two years and who wear nappies. Because the virus may be excreted for 2–3 weeks before the appearance of clinical signs and for 2–3 weeks after, outbreaks are common wherever good handwashing is not practised (Mott et al., 1990). In industrialised countries, HAV infection occurs mostly in adolescents and adults.

Hepatitis B (HBV) often affects people in developing countries where sanitation is poor. HBV occurs most often in infant and children younger than five years, but 70–90 per cent of the adult population has been infected, and 8–15 per cent are chronically infected. Infection with HBV is the leading cause of acute and chronic liver disease worldwide (Bolyard et al., 1998).

P Pathophysiology of hepatitis

The hepatitis virus causes the death of the parenchymal cells of the liver. The inflammatory response causes swelling and blockage of the drainage system of the liver. Biliary stasis and further destruction of the hepatic cells occurs. Because bile cannot be excreted by the liver into the intestine, it appears in the blood (hyperbilirubinaemaia), urine (urobiligen) and skin (hepatocellular jaundice).

Table 7.1 ***Common causes of hepatitis***

Type	Transmission	Incubation	Presentation	Prognosis
Hepatitis A (HAV)	Faecal–oral	15–50 days, most contagious 1–2 weeks, onset 25–30 days	Mild, flu-like signs, no jaundice in children In adolescents, fever, malaise, nausea, jaundice	Good prognosis Recovery provides lifetime immunity
Hepatitis B (HBV)	Blood and blood products, secretions pre/perinatally, sexual contact	50–180 days	Same as HAV onset at 4 months Severity ranges from asymptomatic to fatal infection Most children are asymptomatic 90% of neonates will be carriers	Good prognosis General full recovery except in carriers
Hepatitis C (HCV)	Blood and blood products	14–180 days	Same as HAV	50% develop chronic hepatitis, cirrhosis, cancer
Hepatitis Delta Virus (HDV)	Blood and blood products More common in Mediterranean countries, IV users, haemophiliacs Occurs only in people with HBV infection	21–90 days	Occurs with HBV and causes it to be much more severe	More likely to develop fulminating hepatitis than other strains
Hepatitis E Virus (HEV) *Enterically Transmitted Non A Non B Hepatitis*	Faecal–oral route	15–60 days	Epidemic with similarities to HAV Uncommon in developing countries	High incidence of mortality in pregnant women

(Adapted from Ashwill and Droske, 1997)

Hepatitis infection may result in an asymptomatic or mild illness, in which complete regeneration of liver cells occurs within 2–4 months. More severe forms of hepatitis include:

- **fulminating hepatitis**, in which hepatic necrosis and death can occur within 1–3 weeks
- **sub-acute or chronic hepatitis**, which can result in permanent scarring of the liver and impaired liver function — chronically infected persons are carriers of the disease and are at increased risk of developing chronic liver disease (cirrhosis, chronic persistent hepatitis) or liver carcinoma later in life (Wong, 1993).

C Clinical presentation of hepatitis

In infants and preschoolers, HAV is either asymptomatic or causes mild, nonspecific symptoms such as anorexia, malaise, and lethargy. Because most children with HAV are asymptomatic or have mild, nonspecific symptoms the disease may not be diagnosed until an outbreak occurs. Therefore, the spread of HAV infection in a day care centre often occurs before the initial case is identified.

Nursing assessment and history taking may identify a source of infection. In children, the only signs of hepatitis may be:

- flu and fever
- lethargy and fatigue
- anorexia and nausea
- abdominal assessment (it may find right upper quadrant tenderness and hepatomegaly)
- pale and clay-coloured stools
- dark and 'bubbly' urine
- jaundice — best assessed in schlera of the eye, nail beds and mucous membranes (it usually follows a cephalocaudal progression)
- arthralgias in HBV.

Sufferers of fulminating hepatitis are likely to present with acute hepatic failure with associated encephalopathy, bleeding, fluid retention, ascites, and icteric appearance (Ashwill and Droske, 1997).

HBV may cause a wide range of clinical manifestations, ranging from asymptomatic infection to fatal acute fulminating hepatitis. Symptomatic acute hepatitis occurs in two stages:

1. ***Anicteric (absence of jaundice) phase (lasts approximately 7 days)***
Children will experience:
- anorexia, nausea and vomiting
- right upper quadrant or epigastric pain
- fever, malaise, nausea
- fatigue, depression, irritability

2. ***Icteric (jaundice) phase (lasts 4 weeks or less)***
Children will experience:
- jaundice, urticaria
- dark urine and light-coloured stools
- beginning to feel better as jaundice becomes more apparent.

Acute fulminating hepatitis is noted by bleeding problems, ascites, encephalopathy, and acute hepatic failure. Fulminating hepatitis is due primarily to HBV and HCV.

The symptoms of hepatitis and the clinical changes should return to normal within three months from onset; if not, a chronic state should be suspected. Infection with HBV, HCV and HDV can result in chronic hepatitis and cirrhosis. Chronic HBV infection can also cause hepatic carcinoma (Ashwill and Droske, 1997).

D Diagnosis of hepatitis

A hepatitis outbreak in the paediatric community should be considered if:
- presenting children have a history of exposure to jaundiced children
- there is a confirmed outbreak in day care centres
- children have a history that suggests percutaneous exposure to blood or body fluids.

Although there is no liver function test specific for hepatitis, tests of liver function — especially AST, ALT, bilirubin levels and sedimentation rate — can be indications of liver damage caused by hepatitis. Serum bilirubin levels peak 5–10 days after jaundice appears. History and progressional phases of the disease are essential in making the appropriate diagnosis.

Hepatitis is diagnosed by the identification of antigens (HBsAg, HBeAg) responsible for the disease or the antibodies that develop as a result.

IgM antiHAV antibodies are present at the onset of illness and usually disappear within four months, but may persist for six months or longer. IgG antiHAV antibodies develop shortly after IgM antiHAV antibodies. The presence of IgG without IgM antiHAV antibodies indicates previous infection (Bolyard et al., 1998).

HCV serologic assays are used mainly for detection of chronic hepatitis C because they stay negative for at least 1–3 months after onset of the illness. Liver biopsy may be needed to evaluate the chronic active forms of the disease and to determine the extent of damage in advanced or fulminating cases (Adams et al., 1996).

M Management of hepatitis

There is no specific treatment for hepatitis. In uncomplicated hepatitis, treatment is mainly supportive because the disease is self-limiting. Treatment is aimed at maintaining comfort and adequate nutritional balance. A low-fat, balanced diet can be helpful if children are suffering nausea and anorexia. Hospitalisation is rarely needed.

In fulminating hepatitis, intensive care may be needed to provide haemostasis, nutritional and fluid support, neurologic assessment, and management until the liver has recovered (Mott et al., 1990).

Hepatitis A

Control of further spread is essential because HAV can survive on contaminated objects for weeks. Good handwashing and thorough disinfection of nappy changing surfaces is essential. Children and adults who have had direct contact with an infected person should receive immunoglobulin (IG) as soon as possible after exposure. A vaccine has been developed to prevent HAV infection, and immunisation is recommended for child day care centre workers, health care workers and people living in the household of infected children (Adams et al., 1996).

Hepatitis B

Children with acute or chronic HBV should be cared for with meticulous infection precautions. The most effective way of preventing HBV infection is immunisation with HBV vaccine. HBV vaccine is recommended for all newborns as part of the routine child immunisation schedule.

HBV immunoglobulin is effective in preventing HBV infection if given within two weeks of exposure. It is possible to prevent HDV by preventing HBV (Bolyard et al., 1998).

EDUCATION FOR CARERS OF CHILDREN WITH HEPATITIS

Unless fulminating hepatitis develops, children are usually treated at home so parental education is important.

- Children with hepatitis are often anorexic. Several small nutritious low-fat meals and snacks through the day are better tolerated than regular portions at mealtimes.
- Fatigue and malaise can last for several weeks. Adequate rest and sleep are important for recovery. Rest and general supportive care are important.
- As HAV is not infectious within a week after the onset of jaundice, children may return to school at that time if they feel well enough.
- There are some symptoms that could indicate worsening of a child's condition, especially changes in neurologic state, bleeding and fluid retention. Jaundice may become worse before it improves and parents should be prepared for this possibility. Children should not have any over-the-counter medications because impaired liver function may result in inadequate metabolism and excretion of the medication. Adolescents should be cautioned not to drink alcohol during their illness or recovery period.
- Preventing the infection from spreading is an essential intervention for HAV. This should include enteric precautions for at least one week after the onset of jaundice and excellent handwashing. Handwashing is the most important preventive measure. Family members must be taught how to implement appropriate precautions and to clean exposed household surfaces with bleach. Nappies should not be changed on or near surfaces used for preparing food. The nurse should explain to family members the ways HAV (faecal–oral route) and HBV (parenteral route) are spread to others.

- If children have HBV, especially neonatal HBV, parents should be prepared for the possibility of a chronic carrier state and the development of cirrhosis and hepatocellular cancer later in life.
- If children with HBV have a history of illegal intravenous drug use, the nurse has the responsibility of teaching about the dangers of such behaviour, including the risk of transmission of hepatitis and other infections. Children should be assisted to obtain counselling through a drug program.

As Wong (1993) says, nurses can best help parents care for their child at home by emphasising the importance of:

- meticulous handwashing skills
- careful use of gloves
- disinfection of contaminated surfaces and articles
- monitoring for complications
- providing a well-balanced, low-fat diet
- observing other family members for infection.

8. HERPES SIMPLEX TYPE 1 (HSV 1)

HSV 1 is responsible for a common, contagious and often recurrent infection of the mouth and mucous membranes. HSV 1 can be asymptomatic or extremely painful. According to Wong (1993), HSV 1 causes a wide variety of infections including:

- common cold sores
- corneal lesions
- central nervous system infections.

People with primary or recurrent infections are the only natural sources of HSV 1. Those with clinical or subclinical HSV 1 infections can transmit the virus to others by close personal contact, such as kissing, wrestling and sexual intercourse. Spread of the virus by saliva is evident in the documented cases of herpetic paronychia in medical or dental personnel who handle the infected oral cavities of clients. This mode of viral HSV 1 transmission makes HSV 1 a potential nosocomial infection and may account for the occasional outbreaks of HSV 1 infections in families or other closed communities. Airborne transmission by air droplets or skin dander can also be involved in transmission (Rudolph and Hoffman, 1987).

Transmission of the virus from an infected mother is the main mode of contagion for neonates, either when passing through the infected birth canal or when the membranes have been ruptured for more than four hours. Transplacental transmission has been suggested by the documented cases of infants with chronic central nervous system and ocular abnormalities detected soon after birth. HSV 1 can also be transmitted to the neonate by other infected neonates or from a mother or other contact with cutaneous lesions. Infection rates differ, with HSV 1 more common in premature deliveries, but outcomes of the infection are similar in both premature and term babies. Neonatal herpes is more common in primigravida women. Studies have shown that HSV 1 infection is more common in lower-income communities and when sexual activity starts at an earlier age. The incubation period for HSV 1 ranges from two to twenty days with an average of six. The incubation period for herpetic encephalitis is probably longer (Rudolph and Hoffman, 1987).

P Pathophysiology of HSV 1

HSV 1 is the 'oral' type of herpes and generally affects areas above the waist. After initial HSV 1 infection the virus lies dormant in nerve cells that innervate the area of skin first infected. The virus can be reactivated by stress, fever, trauma, sun exposure, menstruation and immunosuppression. When reactivated, the virus migrates to the skin area innervated by the ganglia that harbours it, near the site of initial infection. The recurrent infection can be symptomatic or asymptomatic and just as infectious as the original infection, but less severe than the original infection. The general health of infected children determines the severity of the HSV 1 infection. HSV 1 in neonates and immunocompromised children can be fatal. HSV 1 is the most common cause of viral encephalitis in children, with a mortality rate of 75 per cent (Mott et al., 1992).

In healthy children, the lesions are confined to the skin and mucous membranes. Bloodstream spread of the virus, resulting in systemic spread of the disease, is more common in:

- the neonate
- severely malnourished children
- children with skin conditions, e.g. eczema
- children with defects in cell-mediated immunity.

In these children, the virus spreads from the portal of entry to organs. HSV 1 multiplies within the organs and the resulting secondary viraemia causes extensive cell destruction. The clinical picture may differ depending on the organs involved and the extent of cell damage. Healing starts with a clearing of the viraemia and a decrease in production of the HSV 1 within the cells (Ashwill and Droske, 1997).

C Clinical presentation of HSV 1

A nursing history and assessment must be made on children as they present to the health agency (Adams et al., 1996).

- The carer should be asked about previous HSV 1 infections.
- The skin should be checked for lesions.
- The eyes should be checked for corneal ulceration and oedema and vision should be assessed for photophobia and blurring.

- Hydration of children requires investigation

Specific body systems should be checked with attention to mouth, lips and skin.

The mouth

This is a very common place for HSV 1 infection to manifest, especially in children between one and five years old. The severity and sites of the infections vary. The buccal mucosa, tongue and palate may be affected and the gums may become inflamed and bleed easily. There may be cervical lymphadenopathy and high temperatures are common. Excessive salivation may present because children find it painful to swallow so drinking and fluid intake may become problematic. The oral lesions and symptoms usually resolve within 10 days. Sometimes the virus affects the larynx and children may present with croup. There may be herpetic infection of the face and eyes and children who suck their fingers may develop infections around their nail beds. In compromised children, oral infections may include the oesophagus and spread to the lungs, disseminating to the liver and other vital organs. In immunosuppressed children, oral herpes is often mistaken for the stomatitis associated with neutropenia. Fevers, lethargy, halitosis and drooling may accompany a herpetic infection (Ashwill and Droske, 1997).

The lips

The lips are as common as the mouth for infection but are rarely the site of primary infection. The febrile illnesses trigger a recurrence that extends from the lips to the face and neck. 'Cold sores' as they are commonly known start with a burning sensation several days before the lesions appear. Sometimes no lesions appear. Labial herpes may be confused with herpes zoster, aberrant vaccinia and staphylococcal pustules.

The skin

Primary and recurrent HSV 1 infections can cause skin vesicles and ulcers on any part of the body, including hands, face and feet. Primary skin infections may be associated with deep burning pain, oedema, lymphangitis, lymphadenopathy and fever. The vesicles may occur as individual spots or in clusters. The vesicles will become pustular, crust over and heal within seven days. They do not usually leave a scar. Immunocompromised children will be more prone to large vesicular outbreaks of prolonged duration.

Four new forms of HSV 1 skin involvement have been discovered.

1. *Herpetic paronychia* occurs in the fingers, is painful and can be confused with bacterial infection.

2. *Herpes gladiatorum* develops in skin areas damaged in the course of body contact, e.g. wrestling, after contact with a person infected with HSV 1. Other contact sports have resulted in HSV 1 infection. Care must be taken that burns victims do not become infected because they can develop herpetic pneumonia and disseminated organ involvement.

3. *Erythema multiforme* can be associated with HSV 1 infection.

4. *Eczema herpeticum* affects children who have a chronic skin infection, e.g. eczema. In severe infections large areas of skin are affected resulting in large fluid and protein loss. Eczema herpeticum generally stays localised to the skin but sometimes becomes disseminated and results in encephalitis.

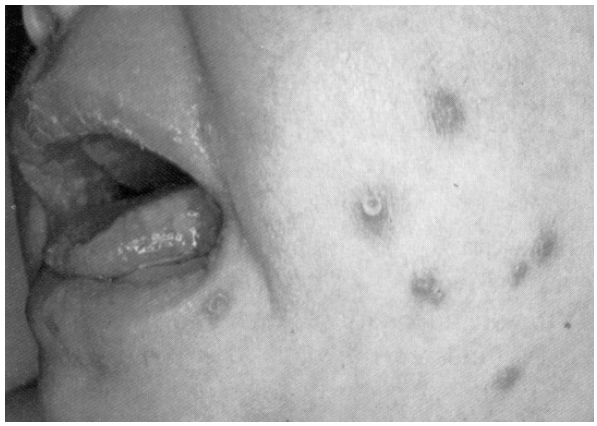


Figure 8.1 Herpes simplex infection in an infant

(From Feigin, R. D., & Cherry, J. D., Eds. [1992]. *Textbook of pediatric infectious disease.* [3rd ed., p.773]. Philadelphia: W. B. Saunders. Used by permission.)

The eyes

Herpetic involvement of the eye is of concern as it can cause loss of vision. Primary infections can be associated with conjunctivitis with or without keratitis. Herpetic vesicles may appear on the eyelids, face or mouth. Tearing and photophobia may also be present. Conjunctivitis is sometimes present with recurrent infections but it is keratitis that is mostly associated with recurrent infections. Corticosteroids must be used with care when children have ocular herpes because they have been known to cause deeper involvement of the eye (Ashwill and Droske, 1997).

Nervous system
Herpes infections have been associated with a variety of neurologic illnesses including encephalitis, meningitis, radiculitis and myelitis.

In healthy children, the lesions are contained to the skin and mucous membranes. Bloodstream spread of herpetic infection, resulting in wide dissemination of the disease, is seen mostly in:
- the newborn
- malnourished children
- children with eczema
- those with lowered immunity

In these children, the virus is transported in the blood from the portal of entry to susceptible organs where it increases, resulting in extensive cell destruction. Most cases of HSV 1 encephalitis (other than those involving the neonate) are caused by neurogenic transmission of the virus to the brain. Healing occurs with a reduction in production of the virus within the cells (Behrman et al., 1992).

D Diagnosis of HSV 1

Diagnosis may be made by clinical manifestations and a history of the child's illness. A Tzanck or Papanicolou smear may be done. A positive smear cannot differentiate between a varicella zoster infection and HSV 1, and a negative smear does not mean children do not have HSV 1. Tissue culture is a more definitive way of making a diagnosis (Katz et al., 1998).

M Management of HSV 1

Management is symptom based. Children with an oral HSV 1 infection should be cared for at home if their fluid intake is adequate. If children become dehydrated, they will require hospitalisation for intravenous hydration. There is no cure for HSV 1 but acyclovir can be used to decrease the severity of the infection. Acyclovir is most often used in neonates, children who are immunocompromised, and children who have encephalitis or ocular implications (Katz et al., 1998).

Antibiotic ointment can be used to treat lesions that become infected. Oral or rectal analgesic with antipyretic agents can be used for comfort.

Anaesthetic mouth washes can be useful to lessen pain and encourage drinking (Ashwill and Droske, 1997).

Primary infections with HSV 1 are self-limiting and usually last one to two weeks. Neonates and malnourished children are usually worst affected. The prognosis for children who develop viral encephalitis secondary to HSV 1 varies in relation to residual effects (Wong, 1993).

NURSING CARE OF CHILDREN WITH HSV 1

Hospitalised children should be placed in contact isolation and precautions implemented with drainage and secretions. Children are considered contagious until the scabs from visible lesions have fallen off. Because these scabs do not form on the mucous membranes, lesions are considered contagious until they have healed. It must be remembered that some HSV 1 infections are asymptomatic. Health care providers should always wear gloves when in contact with children who have HSV 1 infection to prevent herpetic whitlow. Careful handwashing is essential and this applies to caring for children at home. Family members should not share eating and drinking implements or towels with infected children. As HSV 1 can spread to other parts of the body children should be encouraged to keep their fingers out of their mouth. Children with HSV 1 are usually distressed by the illness so lots of cuddles and comfort are needed despite the infectious illness. Particular attention should be paid to handwashing after caring for children with HSV 1 (Potter and Perry, 1997).

Children with oral HSV 1 may experience significant discomfort. Swallowing can be painful and there is a large risk of children becoming dehydrated. Parents need to be educated to the clinical signs of dehydration and the need to take children to a doctor if these signs are exhibited. Children need to be encouraged to drink and this can include iceblocks, noncitrus juices, milk and flat soft drink. Frequent small drinks and snacks of bland food should be offered. Parents need to know that a few days without food will not harm children as long as children drink adequate amounts (Mott et al., 1990).

To prevent secondary infections of the mouth, parents should syringe the mouth with normal saline or antiseptic mouthwash, especially after meals. Pain relief and antipyretic medication should be given on a regular basis. Topical anaesthetic should be used with caution as it can depress the gag reflex in small children (Mott et al., 1990).

9. HIV AIDS

HIV (human immunodeficiency virus) infection is an acquired, cell-mediated immunodeficiency condition causing a broad spectrum of clinical presentations in children. Some children can be asymptomatic and others, depending on the stage of the illness they are in, will have severe symptoms. AIDS (acquired immune deficiency syndrome) is the most severe form of the illness (Adams et al., 1996).

E Etiology of HIV AIDS

HIV in a body fluid can enter an uninfected child in several ways (see Table 9.1). A HIV-infected woman can infect her child via the placenta during pregnancy or at delivery and through breastfeeding (vertical transmission). The chance of such transmission is about 25 per cent and as low as 8 per cent if the mother has taken zidovudine during pregnancy and delivery and the newborn has received zidovudine for six weeks after birth. Some children have been infected by blood transfusions and there have been reports in America of HIV transmission from sexual abuse.

The common way for a child to become infected by HIV is through a body fluid. A list of potentially infectious body fluids prepared by Ashwill and Droske (1997) is shown in Table 9.1.

P Pathophysiology of HIV infection

HIV is a retrovirus consisting of a single positive strand of RNA (ribonucleic acid). It contains an enzyme, reverse transcriptase, which plays a key role in viral replication. HIV enters a cell by direct infusion of the virus to the receptors on the outside of the cell. When the virus has entered the cell, it causes the cell to manufacture HIV DNA (deoxyribonucleic acid). The HIV DNA integrates with the original DNA in the cell. HIV then instructs the cell to manufacture more HIV. The new viruses assemble at the host surface. As they break through the cell membrane, the viruses mature and infect other cells. The most important factor with HIV entry into cells is that it causes

Table 9.1 ***Potentially infectious body fluids***

Human blood
Blood products
Blood components such as clotting factors
Semen
Vaginal secretions
Cerebrospinal fluid
Synovial fluid
Pleural fluid
Pericardial fluid
Peritoneal fluid
Amniotic fluid
Saliva contaminated with blood
Body fluid contaminated with blood
Unfixed tissues and organs
HIV-containing cell and tissue culture

cell death. HIV infection results in the destruction of more and more cells. C-cells control B-cell function so children with HIV infection become deficient in cell- and humoral-mediated immunity. Immunoglobulins become nonfunctional, making children with HIV extremely vulnerable to infections (Potter and Perry, 1997).

C Clinical presentation of HIV

The time between infection and diagnosis of AIDS is shorter in infants and children (average four years) than it is with adults. Only a small number of children experience severe onset of symptoms by four months of age. Most have a longer latency period, with significant illness presenting by the time the child is six or seven years old. The occurrence of *Pneumocystis carinii* pneumonia (PCP), an opportunistic infection, significantly reduces survival. Children who become infected with HIV perinatally often present with PCP between three and six months of age. PCP in infants is often acute in onset and the children have a poor prognosis (Spitzer, 1993).

Table 9.2 *Symptoms of HIV in children*

Mild symptoms of HIV infection include:	The moderate symptoms (if they continue to be recurrent) include:	Other signs include:
Lymphadenopathy Hepatomegaly Splenomegaly Dermatitis Parotitis Recurrent upper respiratory tract infections or sinusitis Otitis media	Anaemia or neutropenia Diarrhoea Fever Herpes simplex Oral candidiasis	Bacterial meningitis Sepsis or pneumonia Cardiomyopathy Complicated chickenpox Hepatitis Nephropathy Herpes zoster Lymphocytic interstitial pneumonia (LIP)

(Adapted from Adams et al., 1996)

According to Adams et al. (1996), the most common AIDS indicators in children aged under thirteen years are:

- LIP
- PCP
- serious bacterial infections
- cytomegalovirus
- encephalopathy
- wasting syndrome.

D Diagnosis of HIV

The diagnosis of HIV is established by measuring HIV antibody in children older than 18 months or HIV antigen in children younger than 18 months. HIV antibody testing in children younger than 18 months indicates only that the mother is infected. It has been reported that 95 per cent of children infected perinatally can be diagnosed by six months of age.

CD4+ counts are used to assess the child's immune status and the need for PCP prophylaxis. The CD4+ counts will be checked at regular

intervals. More regular testing will be required when the child is having PCP prophylaxis and antiviral therapy is indicated (Wong, 1993).

M Management of HIV

The treatment routine for a child with HIV includes:
- a modified immunisation schedule to prevent disease
- prophylaxis against opportunistic infections
- antiretroviral therapy to inhibit viral replication
- aggressive use of medication to treat infections.

There are guidelines to treat PCP prophylactically depending on whether the child has been exposed perinatally or postnatally. There are several antiretroviral drugs (zidovudine and didanosine) commonly used, either alone or combined, to treat children with HIV. According to Ziegler et al., 1996, children on these drugs may experience:
- height and weight gain
- diminished signs and symptoms of HIV
- improved immunologic and neurologic functioning
- an improved short-term survival rate.

NURSING CARE FOR CHILDREN WITH HIV

Most children with HIV infection experience good health initially but as their immune system becomes compromised, they become symptomatic. Caring for these children includes treating the bacterial and opportunistic infections that can cause serious illness. Nursing staff should try and build harmonious relationships with the child and the family when the child first comes into hospital. Any personal feelings and biases the nurse may have should be suppressed, so that they do not interfere in the development of the professional working relationship between the nurse and family.

The initial assessment should include clarifying whether diagnosis of HIV has actually been made as well as assessing what understanding the family has of the disease. The child's height, weight and basic observations should be documented on percentile charts.

Caregivers should be asked whether the child has had any:

- fever
- nausea
- ear pulling
- vomiting
- diarrhoea
- changes in sleep pattern, appetite or general behaviour that may be indicative of secondary infections.

Physical assessment should focus on hydration and respiratory status, mouth and skin lesions, and pain.

Hydration status

Assess the child's skin for turgor.
Check the mucous membranes for moistness, drying or cracking.
Determine whether the child has tears.
Determine whether the child's fontanelle is palpable and soft.
Measure fluid input and urine output as well as urine specific gravity.

Respiratory status
The child needs to be assessed for:
- nasal flaring
- rib retractions
- cough
- respiratory distress
- tachypnoea
- grunting or wheezing
- rhonchi and decreased breath sounds.

Mouth lesions
The child's mouth needs to be observed for white patches on the tongue and/or inside the cheeks and for blistering lips.

Skin lesions
The skin needs to be assessed for blotches; red, flat areas; blistering; and dry patches, especially in the nappy area.

Pain
Assess the child by:
- using an age-appropriate pain scale
- observing the child's speech, facial expression, body movements and responses
- asking the family about the child's pain levels.

HIV is different from other paediatric chronic illnesses because, often, one or both of the parents — and possibly other siblings — will also have HIV. The costs of and the lifestyle implications associated with HIV may mean families have limited financial resources. While the child is in hospital, it is an appropriate time to introduce the family to social workers and other allied health professionals so that basic needs can be met. Other issues such as disclosure, permanency planning and end-of-life decisions about the child need to be discussed at the appropriate time (Boland et al., 1996).

EDUCATION FOR CARERS OF CHILDREN WITH HIV

Diet

The child with HIV needs a high-calorie, high-protein diet. Therefore, formula needs to be mixed correctly with no extra water or cereal. Extra vitamins and supplements should be given by the carer as indicated by the doctor.

Infection control measures

Basic infection control guidelines need to be followed at home.

- Avoid touching the blood of the HIV-infected child.
- Avoid sharing toothbrushes, nail scissors, earrings etc. with other people.
- Use precautions when the child has a nose bleed or a cut.
- Cover open sores and leave scabs alone.
- Clean blood spills with paper towel, wash the area with soap, rinse with bleach and water, and air dry.
- Wrap disposable items soiled with blood in newspaper, put them in a plastic bag and deposit them in a plastic-lined rubbish bin; rinse blood-soiled clothing with hydrogen peroxide and then wash normally.

Immunisation status

Keep the listed immunisations up to date:

- inactivated polio
- pneumococcal vaccine at two years of age
- flu shot annually
- IG after measles exposure
- VZIG if exposed to chickenpox
- TIG if the child has a potential tetanus wound.

Education to recognise symptoms

According to Wong (1993), medical attention is recommended for the following symptoms:

- a temperature higher than 38.5C
- vomiting and diarrhoea
- decreased appetite, difficulty in swallowing, drooling
- rashes, lumps, bumps and sores on the skin
- ear pain, pulling at the ears or discharge from the ears
- wounds that will not heal
- exposure to measles or chickenpox.

10. INFECTIOUS GASTROENTERITIS

Gastroenteritis is one of the most common paediatric infectious diseases. Infections peak in summer and have equal gender distribution. Although gastroenteritis is normally self-limiting, mortality is estimated at 25 deaths per 1000 live births in the first year of life (Wong, 1993).

Infectious gastroenteritis is caused by a group of viruses, bacteria and parasites. According to Mott et al. (1990) these elements are capable of causing:

- serious communicable diarrhoea
- extensive fluid and electrolyte loss
- sepsis
- death.

Transmission of gastroenteritis

Ingestion of contaminated food and water and person-to-person contamination are the most common transmission routes. High-risk groups include children in day care centres, preschools and chronic care facilities, and those infected with HIV. *Giardia* is the most common pathogen in toddlers and rotavirus is the most common in infants (Wong, 1993).

P Pathophysiology of gastroenteritis

As the pathogen bonds to the mucosa of the intestine, it is no longer affected by peristaltic waves and is not removed from the intestine. It invades the epithelial cells causing an inflammatory response and epithelial cell death. This leads to ulcerations, pseudomembranes, bleeding and possible sepsis. Enterotoxins (cholera and shigella) cause fluid and electrolyte shifts that result in decreased secretions into the intestine and a decrease in absorption secondary to oedema. The result is diarrhoea and significant fluid losses. Cytotoxins (salmonella) produce local oedema, malabsorption and dehydration. Some pathogens are also capable of producing neurotoxins that act outside of the gastrointestinal tract (Katz et al., 1998).

Table 10.1 *An overview of common gastrointestinal infections*

Infectious agent	Characteristics	Clinical manifestation	Diagnostic findings	Treatment
Shigella (enteroinvasive with Cytotoxin)	Incubation 1–7 days Most common in summer Faecal–oral spread Communicable for 1–3 weeks	Symptoms last 5–10 days Diarrhoea begins as watery; progresses to small, bloody Severe abdominal pain High fever Neurologic symptoms (headache, nuchal rigidity convulsions) Risk of sepsis, disseminating intravascular coagulation, haemolytic uraemic syndrome, rectal prolapse	Blood, mucous, WBC in stool Positive culture in some cases	Bactrim or ampicillin Enteric precautions Identify source if possible
Salmonella (entero-invasive)	Incubation 6 hours to 3 days Most common in summer and autumn Usually food borne Infectious for duration of illness and variable period afterward	Symptoms last 3–5 days Rapid onset Secretory diarrhoea Abdominal pain, nausea, vomiting common	Positive blood, PMNs in stool	For infants younger than 12 weeks, same as shigella Enteric precaut-ions if possible Identify source
Escherichia coli (entero invasive with Enterotoxin)	Variable incubation Most common in summer Food borne most common	Green, watery, secretory diarrhoea May cause haemorrhagic colitis and fever	Positive blood, PMNs	Same as *shigella* Enteric precautions

Infectious agent	Characteristics	Clinical manifestation	Diagnostic findings	Treatment
Campylobacter	Incubation 1–8 days Most common in infants and adolescents	History of eating contaminated shellfish Severe abdominal pain Offensive, watery diarrhoea	Positive blood, PMNs	Sometimes treated with a week of erythromycin Enteric precautions
Giardia lambli	Most common cause of parasitic diarrhoea Spread in water Common in winter	Afebrile Abdominal distension, flatulence and parasites Variable diarrhoea	Positive blood, PMNs, negative ova Parasite found on duodenal biopsy	Flagyl x 7 days Enteric precautions Treat all unknown water sources with chlorine/ iodine before drinking
Rotavirus	Incubation 1–3 days Accounts for 50% of acute diarrhoea in children	Symptoms last 2–6 days History of preceding or concurrent respiratory illness Fever for 24–48 hours	Negative blood, parasites, ova and PMNs	No drug treatment Enteric precautions Immunisation being developed
Clostridium difficile	Antibiotic associated Most common nosocomial diarrhoea	Diarrhoea develops after commencement of antibiotics	Positive blood, PMNs	Cholestrymine used to enhance mucosal recovery Sometimes treated with flagyl or vancomycin for 10 days

C Clinical presentation of gastroenteritis

Children who have gastroenteritis will present with the following symptoms in varying severity:

- diarrhoea of varying amount and consistency
- tenesmus
- abdominal pain
- fever
- vomiting
- dehydration
- history of recent travel to other countries.

When children present with any of these symptoms, it is important for the nurse to be familiar with the correct assessment required for this paediatric problem (Adams et al., 1996).

- Obtain an adequate history of the event, including the length of symptoms, frequency and consistency of the stool, and the presence of blood or mucous in the stools. Note the amount, colour, consistency and time (ACCT) of each stool as a consistent way to document findings.
- The concurrent appearance of symptoms in other members of the family can be useful in diagnosis.
- Any recent travel to other countries or rural areas should be documented.
- Evaluating food and formula preparation and examining sanitation and hygiene at home and in day care facilities can provide useful information.

Children may appear moderately to severely dehydrated with hyperactive bowel sounds and severe diarrhoea, often bloody in nature. Blood in the stool appears after maximum fluid loss has occurred and can be useful in determining what stage of illness the child is at. The presence of vomiting, tenesmus and fever should be assessed. Complaints of headache, nuchal rigidity, irritability and seizures are important to note as they can indicate the presence of neurotoxins (Ashwill and Droske, 1997).

Assessment of hydration status is critical. Children must be assessed for:

- poor urine output
- high urine specific gravity

- decreased skin turgor
- dry mucous membranes
- no tears when crying
- a sunken/depressed fontanelle (in infants) and skin tenting.

Any of these can happen quickly with large amounts of fluid lost from diarrhoea. Loss of bicarbonate (from severe diarrhoea and dehydration) makes metabolic acid a major concern. The compensatory mechanisms of increased respiratory effort and rate are important to document as children will try and correct an acidosis by increasing respiratory effort (Potter and Perry, 1997).

D Diagnosis of gastroenteritis

Definitive diagnosis of the specific infectious agent can be made from a stool culture that is positive for a pathogen. Only children who appear toxic or have bloody stools, abdominal pain or tenesmus usually require blood tests. Blood cultures may be needed in acutely unwell children. An unprepared sigmoidoscopy can be useful in the diagnosis of the amount of mucosal involvement (Katz et al., 1998).

M Management of gastroenteritis

There are physiological differences in age groups of children when considering the management of their diarrhoea irrespective of the cause.

Neonates and babies (0–9 months old)

Compared with children in older age groups, neonates and babies:

- can lose fluids equal to their extracellular fluid within 2–3 days due to the higher percentage of water in their extracellular fluid
- have a decreased ability to concentrate urine because of their immature renal system
- have a higher rate of peristalsis
- have a harder time compensating for acidosis because of their decreased ability to acidify urine
- have a higher metabolic turnover due to an increased metabolic rate — if losses are not replaced quickly, dehydration occurs
- are unable to verbalise or communicate thirst.

Infants and young children (9–18 months old)

Compared with older children, infants and young children:

- have a proportionately greater body surface area in relation to body mass, resulting in greater potential for fluid loss via the skin and gastrointestinal tract
- have a higher proportionate water content with a larger proportion of fluid in the extracellular space
- have an immature immune system with more tendencies to infectious diseases with the consequent risk of developing dehydration.

The main aim of therapy is to replace water and correct acid base or fluid and electrolyte disturbances with intravenous or oral replacement. The rate of replacement may be as high as 50–100 millilitres per kilogram of body weight over 4–6 hours (1–2.5 times maintenance). Because diarrhoea is high in sodium, potassium and bicarbonate, oral rehydration solutions should be used to match the losses. Oral rehydration therapy is as effective as intravenous fluid replacement in mild to moderately dehydrated children. Children will often be hospitalised to allow for continual assessment and management of symptoms and sepsis.

Assessment of children with acute diarrhoea means keeping a strict fluid balance chart which includes monitoring intake, output and weight loss/gain. Providing safety considerations and neurological assessment for children who have a neurotoxin is important as they can experience seizures and differing levels of consciousness (Barber and Masiello, 1996).

Pain and fever can be relieved by the use of antipyretic medicine and by dressing children in cool clothing. Antimicrobial therapy is indicated with some toxins (see Table 10.1). Antimotility drugs are sometimes used for severe diarrhoea but may delay the clearing of the pathogen and increase the extent of invasion. They should not be used for longer than 48 hours (Barber and Masiello, 1996).

Preventing the spread of the infection is vital. Thorough handwashing is extremely important. Isolation techniques, as directed by the health agency's protocol, must be used for staff and family. Families must maintain the isolation measures for up to two weeks after children go home. Gastroenteritis is a public health concern and sometimes dietary recalls of food products are necessary to establish the cause of the gastroenteritis outbreak and to minimise risk to the public. Some toxins isolated will need to be notified to the appropriate health authorities. (See Appendix.)

EDUCATION FOR CARERS OF CHILDREN WITH GASTROENTERITIS

- Teach home carers adequate handwashing.
- Encourage day carers to use gloves when changing nappies, clothes and linen.
- Allow children to use a separate bathroom if necessary.
- Give children appropriate oral rehydration fluids in small amounts every 30 minutes.
- Keep day carers aware of dietary changes and provide supervision with food and formula preparation as needed.

Gastroenteritis can be communicable for several weeks after symptoms have cleared so precautions are still necessary (Barber and Masiello, 1996).

11. INFECTIOUS SKIN CONDITIONS

Skin conditions constitute a large number of paediatric presentations to health agencies (McDonald and Smith, 1998). To understand the nature of infestations and infections that are often experienced in paediatrics, it is important to know that there are paediatric differences in the skin (Ashwill and Droske, 1997).

- Neonates' skin is thin and at risk of external irritants and infection. Gentle handling is required to prevent blistering.
- Babies and young children have a greater relative skin surface area to body volume ratio than adults so there is greater absorption through the skin. Topical ointments and skin lotions should not be used for a long time or without a doctor's order.
- The skin of young children is more susceptible to infection than that of older children.
- Immunoglobulin A does not reach adult levels until children are aged between two and five years, so children are less resistant to microorganisms.
- Eccrine glands do not reach maturity until children are three years old so infants are less able to regulate body temperature .

PEDICULOSIS

Pediculosis refers to the infestation of lice in the scalp or body. Although pediculosis is not a serious health problem, it is often a source of embarrassment for parents and school personnel.

P Pathophysiology of pediculosis

Pediculosis can involve:
- the scalp (*pediculus humanus capitis*)
- the body (*pediculus humanus corporis*)
- the pubic area and eyelashes (*pediculus humanus pubis*).

Each of these infestations is caused by a different louse but they have a similar life cycle (Ashwill and Droske, 1997). Lice feed by sucking blood (Figueroa et al., 1998). Head and pubic lice spend their life cycles on the skin of the human host while body lice live in clothing and latch onto the skin only to feed. The female louse lays eggs (nits) at the base of the hair shaft. Each egg is covered with a gelatinous material which dries into white masses that are stuck to the hair shaft. The eggs incubate for one week and lice reach sexual maturity at two weeks. Lice can be spread as long as the lice and nits are alive on the host and belongings. Lice can only live 48 hours off the host. Nits, if placed in the right environment, are able to reproduce for ten days (Ashwill and Droske, 1997).

E Etiology of pediculosis

Lice can live only on humans. They are transmitted by direct contact with infested persons and indirect contact with the infested person's belongings (Figueroa et al., 1998). Having clean hair is no deterrent to becoming infested with lice (Ashwill and Droske, 1997). Healthy lice removed from the head with a comb may reinfest if brushed back on within two days (Thompson, 1998).

Incidence

Girls are affected more than boys and there is no higher incidence in any specific socioeconomic groups. Peak incidence is in preschool and young school-age children. Pubic lice are more common in adolescents as they are generally transmitted by sexual contact. Eyelid infestation in young children can indicate sexual abuse (Ashwill and Droske, 1997).

C Clinical presentation of pediculosis

Pediculus humanus capitis

- Itching is often the only symptom. The condition may not manifest until children have been infested for a period of weeks or months (Thompson, 1998).
- Nits are easily seen on the hair shafts near the scalp and are usually found in the nape of the neck and behind the ears. Nits look like

dandruff but are more difficult to remove (Ashwill and Droske, 1997).

- Pruritus of the scalp and neck may be visible as children can develop an allergic reaction to the lice faeces. Children can develop infections from scratching (Thompson, 1998).

Pediculus humanus corporis

- Papular, rose-coloured dermatitis is visible in areas where children have tight clothing, e.g winter leggings.
- The infested areas are very itchy (Ashwill and Droske, 1997). In long-term infestations, skin may be thickened due to scratching (Figueroa et al., 1998).
- Lice are visible in clothing and the bite patterns follow seam lines (Figueroa et al., 1998).

Pediculus humanus pubis

- Lice are found in pubic and facial hair as well as axillary and general body hair.
- Maculae cerulae (blue spots) may be found on the thighs and torso if children are heavily infested. Dark brown marks on underwear and sheets are indicative of louse defecation.
- Infested areas are very itchy.

D Diagnosis of pediculosis

Identification of the nits on the scalp completes diagnosis. The hair should be parted moving from side to side and front to back. The exposed scalp should be examined under a bright light (Adams et al., 1996).

M Management of pediculosis

Treatment includes conventional and alternative methods of treatment.

Wet combing

Wash the hair in the normal way but apply a lot of conditioner. Comb the hair from the roots with a fine tooth-comb. If any lice are found, repeat

the process every three or four days for two weeks. Remove lice in the eye lashes by applying petrolatum to the lashes twice a day for eight days (Adams et al., 1996).

Insecticides

These contain malathion, phenothrin, permathrin or carbaryl. Some products contain lindane which is potentially neurotoxic so it should not be used without a doctor's order. Children with open sores (from scratching) can absorb enough lindane to induce fitting (Thompson, 1998).

Battery-operated comb

Use it like a manual comb but on dry hair. The comb gives off a current which immobilises the lice and they then loosen their grip on the hair (Thompson, 1998).

Natural methods

- Little research has been done into the effectiveness of lavender or tea tree oil and it is not known whether the oils become toxic if used often or if incorrect doses are used.

- Some oils cause allergies or are dangerous in pregnancy so oils should be used carefully and not as a preventive measure (Thompson, 1998).

- A solution of half vinegar and half rubbing alcohol can be used (Ashwill and Droske, 1997).

EDUCATION FOR CARERS OF CHILDREN WITH PEDICULOSIS

Carers who will be managing pediculosis at home must be given clear information about using insecticides.

- Use insecticide treatments only when lice have been found, not as a preventive measure. Never use anti-lice sprays on children.
- Use a plastic comb when detecting lice. Back comb the hair to ensure the maximum number of lice are removed.
- Head lice shampoos are not as effective as insecticides.
- To ensure all the head is covered, use at least 50 ml of lotion for each application. Apply a few drops to small partings on dry hair and spread over the scalp and hair with the fingers. Continue until the whole head is covered.
- If, after rinsing the lotion off, lice are still seen or are seen a couple of days after treatment, the lice may be resistant to the insecticide so switch to wet combing or use a product with a different ingredient. If the lotion contains phenothrin or permethrin, do not use another product containing either of these because they belong to the same insecticide group. Otherwise, a second application is recommended a week later to kill any lice and eggs that were missed.
- Do not use any product containing malathion or carbaryl more than once a week for three weeks at a time (Thompson, 1998).
- Read instructions carefully to avoid dangerous complications of incorrect insecticide use.
- Bedding linen from infested children should be washed in hot water and dried on a hot setting. Items that cannot be washed should be dry cleaned or sealed in plastic bags for three weeks. Combs and brushes should be soaked in boiling water for 15 minutes and the home should be cleaned thoroughly to remove lice.
- Encourage children not to share hats, hair pieces, brushes, combs, clips and hairbands.
- Children should be rechecked in 7–10 days for reinfestation. Contact a doctor if the itching interferes with the children's sleep, if the condition does not clear after one week or if the lesions look infected (Figueroa et al., 1998).

SCABIES ('itch mite')

Scabies or 'itch mite' is a contagious skin condition that has been causing health problems in children for centuries (Bolyard et al., 1998).

P Pathophysiology of scabies

Scabies results from infestation of *Sarcoptes scabei*. The female mite burrows into the epidermis, lays eggs and dies in the burrow after 4–5 weeks. The eggs hatch in 3–5 days and larvae migrate to the skin surface to mature and complete their life cycle. The mites, eggs and excreta cause children intense pruritus. One of the complications of scabies is impetigo which will be discussed next.

E Etiology of scabies

Scabies is transmitted by close personal contact with infected people. People who share a bed or crowded living conditions are likely to transmit scabies to each other. The scabies mite cannot survive for more than three days off human skin so transmitting of scabies by bedding and clothing is rare. Scabies affects all groups, ages and communities but it is most common amongst young children and in preschool day care centres (Bolyard et al., 1998).

C Clinical presentation of scabies

- Intense pruritus is present, especially at night. Children may be bad tempered, may sleep restlessly and may rub their hands and feet together.
- Burrows (fine, grey threadlike lines) may be difficult to see. They can be hard to see because of the excoriation and inflammation that accompanies the pruritus.
- Papules, vesicles and nodules are common. Infestation may also look like eczema and scaly patches. Sites of infestation are most often found in the wrists, finger webs, elbows, umbilicus, axilla, groin and buttocks. In babies, the head, neck, palms and soles of the feet may be involved.

- Lesions are usually secondary infections from scratching.
- Scabies is usually found on more than one family member (Potter and Perry, 1997).

D Diagnosis of scabies

Definitive diagnosis is by examining skin scrapings of the affected areas. Scabies is not easy to diagnose, burrows may not always be seen. If itching is not obvious, ask if anyone else in the family is itchy (Bolyard et al., 1998).

M Management of scabies

Treatment consists of topical lotions. All household members and close contacts should be treated at the same time. This includes day care groups and preschool classes. Care must be taken if a contact may be pregnant. The lotions used are usually based on malathion, lindane, crotamiton or permethrin. Because of the risk of neurotoxicity, lindane should not be used on children younger than two years old. Malathion should be used with medical supervision on children younger than six months. Permethrin should not be used for children under two months of age and should be used under medical supervision if the child is younger than two years old (Figueroa et al., 1998).

A hot bath is not needed before application of lotions and may cause harm by increasing absorption of the scabicide into the bloodstream and removing it from the skin. The lotion should be applied to all skin surfaces from the neck down, with attention to the webs of fingers and toes, behind the ears and under nails. Lotion instructions need to be read carefully to see whether a chosen lotion can be applied to the face. Lotions should be reapplied to hands washed during the treatment phase and babies should wear mittens to stop them sucking the scabicide. Children are usually clear 24 hours after treatment although the papules may remain and cause itching for several weeks after treatment. Treatment failure is usually related to incorrect use of the prescribed lotions (Potter and Perry, 1997).

EDUCATION FOR CARERS OF CHILDREN WITH SCABIES

- Children's bedding should be washed in hot water.
- Lotions must be kept on for the recommended period of time.
- Carers should be told that continued itching after treatment can occur and it is not an indication for a repeat of lotion application, which may cause neurotoxicity.
- In severe cases of scabies, a repeat application may be required but this should be assessed by a health care provider prior to reapplication (Ashwill and Droske, 1997).

IMPETIGO

Impetigo is one of the most common skin infections in children.

P Pathophysiology of impetigo

Impetigo begins with an area of broken skin, such as an insect bite, scabies or dermatitis. The incubation period is 7–10 days. Impetigo is very contagious and can spread to other parts of children's bodies and to others who have contact with infected children or use the same equipment as the children. Spread of the infection is related to poor hygiene practices, crowded living areas and a hot, humid environment. Impetigo lesions resolve in 12–14 days, with treatment (Potter and Perry, 1997).

E Etiology of impetigo

Impetigo may be caused by *Staphylococcus aureus* or group A beta-haemolytic streptococci or a combination of both. *S. Aureus* is the main cause of most cases of impetigo. Impetigo is most common in the summer months when there is humidity. Toddlers and preschoolers are most affected.

C Clinical presentation of impetigo

- Primary lesions are small red macules that quickly become vesicles or large bullae that rupture easily and release serous fluid.
- Secondary lesions are thick, honey-coloured crusts that are lightly attached to the lesions. A surface lesion that bleeds easily is noticeable if the crust is removed.
- Lesions are pruritic. Scarring is rare but will occur if children pick at the lesions.
- Lesions usually present around the mouth and nose but may be present on the extremities.
- Children should be assessed for size, spread and distribution of the impetigo lesions. If children are receiving antibiotic therapy for the lesions, they should be observed for signs of allergy. If the impetigo is caused by haemolytic streptococci, children should be monitored for periorbital oedema or blood in the urine as they may develop acute glomerular nephritis secondary to the streptococcal infection (Thompson, 1998).

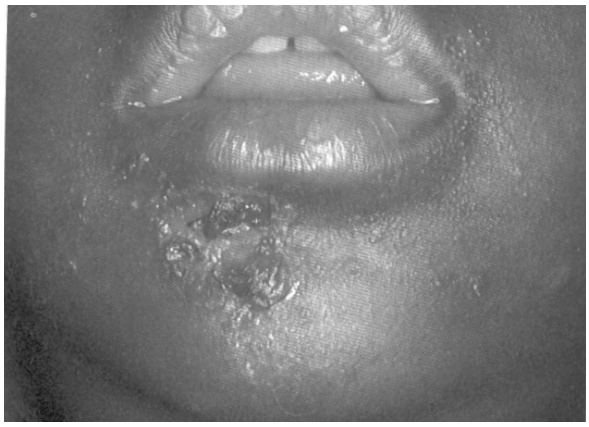


Figure 11.1 Impetigo lesions are usually located around the mouth and nose but may be located on the extremities.

(From Hurwitz, S [1993]. *Clinical pediatric dermatology: A textbook of skin disorders of childhood and adolescence* [2nd ed., p. 280]. Philadelphia: W. B. Saunders. Used by permission.)

D Diagnosis of impetigo

Diagnosis is usually made from the visible characteristics of the lesions. Skin scrapings and cultures are not usually done unless children do not respond to treatment. If a culture has to be done, it should be obtained

from under the skin lesions or from the fluid from inside the lesions (Adams et al., 1996).

M Management of impetigo

Impetigo is treated with topical and oral antibiotics. The lesions should be washed frequently with warm water and attempts should be made to remove the crusts. A topical ointment can then be applied. Severe cases of impetigo are treated with oral antibiotics, usually erythromycin or dicloxacillin. Impetigo that is extensive or involves internal organs is treated with intravenous antibiotics. Antibiotic treatment of streptococcal impetigo does not prevent glomerular nephritis but it can assist in the healing of lesions (McDonald and Smith, 1998).

Strict handwashing and careful hygiene are necessary to prevent the spread of infection (Potter and Perry, 1997).

- Children can spread impetigo simply by touching another part of the skin after scratching the infected areas.
- Fingernails should be kept short and hands washed often with antibacterial soap.
- Family members should not share the same living equipment with infected children.
- Children should not attend school for one to two days after the commencement of systemic antibiotics or two days after topical antibiotics. The school should be told of the diagnosis.
- Parents must be encouraged to make sure their child completes the course of antibiotics as prescribed.

12. MEASLES (RUBEOLA) AND ROSEOLA

Measles is an acute communicable disease that has three stages (Katz et al., 1998).

1. The **incubation** stage is approximately 10–12 days with mild if any symptoms.
2. The **prodromal** stage is characterised by Koplik spots on the buccal and pharyngeal mucosa, slight to moderate fever, mild conjunctivitis, coryza and an increasingly severe cough.
3. The last stage is a macropapular **rash** which erupts over the face, neck, body, arms and legs. A high fever is usually associated with this stage of the disease.

E Etiology of measles

Measles is an RNA virus of the family Paramyxoviridae, genus *Morbillivirus.* Only one antigenic type is known. During the prodromal stage and when the rash appears, it can be found in the nasopharyngeal secretions, blood and urine. The virus can live at room temperature for more than 24 hours (Rudolph and Hoffman, 1987).

P Pathophysiology of measles

Children become infected either directly or by inhalation into the upper respiratory tract or indirectly via the conjunctival sac or by virus-laden droplets from a person infected with measles. At the portal of entry a short period of local virus multiplication and limited spread ensues. This is followed by a viraemia that transports the virus to distant sites where the virus replicates actively in lymphoid tissues. A more prolonged secondary viraemia occurs associated with the widespread dissemination of the disease. From that time (about 9–10 days after initial exposure) until the beginning of the rash, the virus can be detected in the body and can be found in urine and blood. Children are most infectious at this time.

With the onset of rash (about 14 days after the initial infection) the viral replication slows and by 16 days it is difficult to detect the virus apart from in the urine (Behrman et al., 1992).

C Clinical presentation of measles

Respiratory symptoms will appear about 10 days after the measles virus has entered the body. Children will have a runny nose, cough and fever, and may have conjunctivitis and photophobia. Koplik spots may occur two days before the rash becomes visible. Koplik spots are blue–white spots with a red base found on the buccal mucosa. These spots will last about three days and then slough off. The measles rash often begins behind the ears, at the hairline, on the forehead and on the upper part of the neck and will spread towards the feet. The rash:

- is red
- lasts about 6–7 days
- blanches easily with pressure
- will turn a brown colour as the disease progresses.

Children younger than nine months who still have maternal antibodies or children who have been given immune gamma globulin may develop modified measles — the prodromal period is shorter and the symptoms are minimal with few or no Koplik spots.

Atypical measles can occur in children who have been given killed measles vaccine. These children may present with a sudden onset of headache and fever, a nonproductive cough and chest pain. Often they do not have Koplik spots. A rash more yellow than in typical measles begins on the distal extremities and spreads upward, stopping at the nipple line. The extremities can be oedematous and hepatosplenomegaly can be present (Katz et al., 1998).

D Diagnosis of measles

Diagnosis is usually made from the clinical picture and laboratory diagnosis is often not required. During the prodromal stage, the virus can be found in nasal mucosal smears. The virus can be isolated in tissue culture and there can be an elevation in antibody titres. The white blood

cell counts tend to be low with a relative lymphocytosis. The measles rash must be differentiated from other rashes such as Kawasaki disease, adenovirus, meningococcemia and drug rashes (Elgart, 1993).

Complications of measles

Viral complications
These include:
- laryngotracheobronchitis
- bronchiolitis, pneumonitis and intestitial pneumonia
- keratoconjunctivitis
- myocarditis
- mesenteric adenitis or appendicitis
- encephalomyelitis
- other central nervous system complications including subacute sclerosing panencephalitis.

Bacterial complications
These include:
- otitis media
- sinusitis and mastoiditis
- pneumonia
- noma
- furunculosis (Behrman et al., 1992).

Uncertain etiology
These complications can include thrombocytopenic purpura, exacerbation of tuberculosis, cystic fibrosis and nephrotic syndrome (Mott et al., 1990).

M Management of measles

Treatment of measles is supportive. There is no specific treatment for uncomplicated measles. Bed rest, avoidance of bright light, encouragement of fluids and antipyretic measures are recommended. Respiratory compromise may occur and children will need high humidity to assist in opening their airways. Sometimes oxygen may be required. Specific antimicrobials should be used to treat secondary bacterial infections. Vitamin A is recommended as it has been found to reduce

morbidity and mortality. It is believed that Vitamin A plays a role in cell integrity and promoting the immune system (Wong, 1993).

Respiratory isolation for children with measles requires masks for those in close contact with the infected children. Handwashing is also advised after contact with children (Adams et al., 1996).

When a measles epidemic is suspected, prophylactic treatment for the community is recommended. The initial measles immunisation is usually given when children are 18 months old but seroconversion is not 100 per cent and immunity can decrease over time. This is why children are reimmunised at school age.

The use of live vaccine is not recommended for pregnant women and children with untreated tuberculosis. Live vaccine is contraindicated in children who have leukaemia and those who are having immunosuppressive treatment because it puts them at risk of chronic and persistent infections such as pneumonia. If susceptible children are exposed to measles, measles immunoglobulin should be given as soon as possible after contact (Katz et al., 1998).

ROSEOLA INFANTUM (exanthem subitem, sixth disease, three-day fever)

Roseola infantum is caused by human herpes virus six and was discovered in 1986 by Yamanishi. There are now other viruses emerging that are thought to be linked to the disease (Cunha and Johnson, 1995).

C Clinical presentation of roseola

Most clinical cases of roseola occur in the 6–18-month age group. Children do not usually present with a toxic picture but they may present with:

- high fevers, malaise and irritability, though they will usually be active and alert (Stevenson and Brooke, 1994)
- an intermittent or constant fever which may continue for three to five days
- a cough
- abdominal pain
- headache

- vomiting
- diarrhoea.

After three to five days the fever will subside and within hours or days, a rash will appear. The rash is rose pink and maculopapular (macular that blanch under pressure). The rash will be predominantly on the neck and trunk and may be surrounded by a whitish ring. The rash will last from 24 to 48 hours before it fades (Mott et al., 1990).

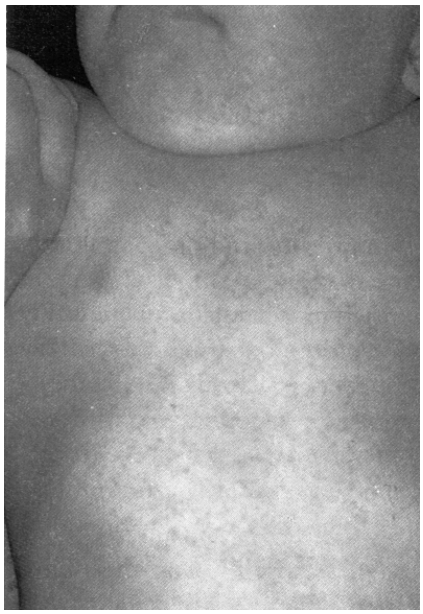


Figure 12.1 Roseola infantum lesions and rash distribution

(From Hurwitz, S [1993]. *Clinical pediatric dermatology: A textbook of skin disorders of childhood and adolescence* [2nd ed.]. Philadelphia: W. B. Saunders, p. 358, Fig. 12–16. Used by permission.)

M Management of roseola

Treatment is supportive. Antipyretic measures are recommended for children prone to convulsions to assist in settling them if they are restless (Wong, 1993).

Complications of roseola

Complications do occur but their frequency and predisposing factors require further research. Convulsions occur and there have been rare cases of hemiplegia, encephalitis, paresis and mental retardation (Breese-Hall, 1996).

13. MUMPS

Mumps produces a generalised infection that is usually characterised by the development of parotitis. Because of widespread vaccination programs against mumps, the disease is not as common as it was earlier this century (Katz et al., 1998).

E Etiology of mumps

The causative organism is the *paramyxovirus* which is transmitted by airborne droplet, salivary secretions and, possibly, urine. The incubation period is usually 16–18 days but can last as long as 25 days. People infected with mumps are considered contagious from 48 hours before the parotid swelling starts until nine days afterwards. If children are infectious when they are admitted to hospital, widespread nosocomial infection may result. The large number of subclinical infections and the fact that children are contagious before the parotitis begins makes it very hard to prevent the spread of infection simply by isolating children with mumps (Mott et al., 1990).

C Clinical presentation of mumps

It is uncommon for children with mumps to present with severe systemic manifestation. They may be febrile for 3–4 days. Parotid swelling is often the first sign of illness. The swelling may last 7–10 days and can be unilateral or bilateral. The submandibular glands may swell along with or in the absence of parotid swelling. Parotid swelling produces discomfort and headaches may be present. Some children complain of abdominal pain. This is because the pancreas or female ovaries may be involved (Wong, 1993). A large percentage of mumps infections will not require medical attention. These children do not develop parotid swelling and may have only mild symptoms (Katz et al., 1998).

D Diagnosis of mumps

All mumps infections are not associated with parotid swelling and not all children with parotid swelling have mumps. The virus can be identified by throat swab and can be isolated in urine and spinal fluid (Wong, 1993).

M Management of mumps

Uncomplicated mumps may need only symptomatic care. Respiratory isolation is indicated until nine days after the onset of parotid swelling. Orchitis requires bed rest, intermittent application of ice packs and emotional support. Meningomyeloencephalitis, which reflects central nervous system involvement, presents with:

- fever
- headache
- nausea and vomiting
- nuchal rigidity
- changes in sensorium.

Affected children are treated symptomatically and generally have an uneventful recovery.

Prophylactic management can be considered if there is a mumps epidemic. There are two vaccines for prophylactic management of mumps:

Passive: Hyperimmune mumps gamma globulin is not effective in preventing mumps or decreasing complications.

Active: Vaccinated children do not usually develop a fever or other detectable clinical signs. They do not excrete the virus and are not contagious to susceptible children (Ashwill and Droske, 1997).

Complications of mumps

The viraemia that occurs early in the infective process is what most likely accounts for the widespread complications.

According to Katz et al. (1998), complications can include:

- meningoencephalomyelitis
- orchitis and epididymitis
- oophoritis
- pancreatitis
- nephritis
- hyroiditis
- myocarditis
- mastitis
- deafness and ocular complications
- arthritis
- thrombocytopenic purpura
- mumps embryopathy

NURSING CARE OF CHILDREN WITH MUMPS

- It is important to obtain a history of the onset of symptoms and to examine children's ears and throat. The characteristics of the lymph nodes in the neck should be documented, as should temperature and general level of wellness. A neurologic evaluation should also be performed. In boys, an examination of their testes should be included in the assessment.
- Usually, children with mumps are not hospitalised unless there are complications. When children are hospitalised, isolation is required according to hospital policy. Handwashing is of utmost importance. Contaminated articles should be disposed of according to hospital policy. Patients, family and visitors should be instructed on good handwashing because the disease is airborne. The family should be instructed to cover the patient's nose and mouth during sneezing and coughing episodes.
- Generally, children with mumps are not seriously ill. They will be uncomfortable. Antipyretics and analgesia can be given to control malaise. Warm or cold compresses applied to the neck may be of benefit. As intake of food may be painful, soft, bland foods may be preferred. Foods that are acidic or spicy should be avoided.
- Infected children must be isolated while infectious. Children in isolation will need diversional activities according to their interests. Bed rest may be of benefit during the illness.
- Boys with orchitis may need ice packs, bed rest, and scrotal support. Emotional support may be of importance to the parents. Concern over sterility may be a source of stress.
- Parental education regarding the course of the disease or any potential complications may need to be reinforced. Parents often need additional instruction on preventing the spread of infection and maintaining their children's immunisations (Adams et al., 1996).

14. PERTUSSIS (WHOOPING COUGH)

Pertussis is a communicable infection of the respiratory tract. It is characterised by severe paroxysms of cough that peak in a gasping, stridorous inspiratory effort. The ensuing 'whooping' is how the name 'whooping cough' originated. It is the result of air being drawn forcibly through the narrow glottis. The word 'pertussis' means intensive cough. Pertussis is thought to be a more appropriate term as not all children with pertussis whoop. Because babies do not receive maternal immunity, they are very susceptible to pertussis and there is a high infant mortality associated with pertussis (Wong, 1993).

E Etiology of pertussis

The causative organism is *Bordetella pertussis.* It was first isolated by Bordet and Gengou in 1900 but it was not until 1906 that *B. pertussis* was cultured in a laboratory setting, where its characteristics were able to be identified. Pertussis is spread through the air from droplets or droplet nuclei from aerosols generated by the intense cough of infected persons (Mott et al., 1990).

C Clinical presentation of pertussis

After an incubation period of 6–20 days, the onset of clinical illness progresses in four stages.

1. *The catarrhal stage*
This phase consists of several days of children having a clear mucous runny nose, nasal congestion and sneezing followed by the occasional cough. In this phase, the illness is no different from a common cold and unless there is a history of other family members having the illness with the symptomatic whoop, there is little reason to believe children have anything other than a cold. Later the cough becomes more persistent and severe, worsening at night. The mucous expectorated is thick, stringy and profuse. There is a heavy growth of *B. pertussis* in these secretions.

2. *The paroxysmal stage*
This phase does not often begin without the preceding catarrhal stage. Violent, protracted bouts of coughing happen in distinct paroxysms that can last up to several minutes. The severity of the illness is indicated by how frequent the coughing episodes are. In children older than six months, the characteristic 'whoop' will be heard as children gasp for breath between paroxysms of coughing. Towards the end of the paroxysm, children will often vomit the previously swallowed mucoid secretions. Severe coughing will make children go florid and there will be venous enlargement of the head and neck. There may also be facial petechiae, periorbital oedema and subconjunctival and scleral haemorrhages. Younger and weaker infants may not whoop but more often they will become exhausted, vomit, aspirate and lose consciousness. The end result may be anoxic brain damage or death. The paroxysmal stage of the infection may last from a few days to a month, sometimes longer. Coughing lessens gradually until paroxysms are no longer present.

3. *The convalescent stage*
This begins when a chronic cough replaces the paroxysms. It can last up to a month, with the cough improving slowly.

A complete immunisation history and any known exposures should be obtained about any children suspected of having pertussis. Nurses also need to ask about:
- events prior to admission
- coughing activity
- secretions
- cyanotic episodes
- the child's activity level.

An assessment should be done of the respiratory, fluid, nutrition, output and neurologic states (Ashwill and Droske, 1997).

Complications of pertussis

Suppurative complications
Secondary infections of the respiratory tract should be considered if children develop a temperature because pertussis is not usually associated with a fever. Acute otitis media is common as is sinusitis.

Non-suppurative complications

Increased intrathoracic pressure from the severe coughing episodes, hypoxia and persistent vomiting cause venous engorgement and hypoxaemia with petechial haemorrhages and epistaxis. Convulsions and encephalopathy can sometimes occur. Increased thoracic pressures have been recognised as contributing to ruptured diaphragms, umbilical and inguinal hernias and rectal prolapses. Pneumonia is a common complication and there are varying degrees of respiratory complications including atelectasis, emphysema and pneumothorax. Most deaths from pertussis are attributed to the respiratory complications associated with the infection. The vomiting associated with coughing can result in metabolic alkalosis with tetany (Behrman et al., 1992).

D Diagnosis of pertussis

The diagnosis is confirmed by identifying *B. pertussis* organisms in respiratory tract secretions through nasopharyngeal swabs cultured in the appropriate medium. Maximal growth occurs after five days but plates should not be read as negative or discarded until after seven days. Blood tests for pertussis serology can also be done (Wong, 1993).

Pertussis should be considered for any children who report a troublesome cough especially if there are paroxysmal episodes associated with the cough or it becomes worse at night. Other pointers are where there has been recent contact with a person known to have pertussis, where infection is known to be prevalent in the local community and where a child is not fully immunised (Rudolph and Hoffman, 1987).

The differential diagnosis includes respiratory infections caused by viruses. Chlamydia pneumonia in infants younger than four months of age can be confused with pertussis. An obstruction due to a foreign body or undiagnosed cystic fibrosis can also be mistaken for pertussis.

M Management of pertussis

Hospitalisation and supportive care is usually required for infants; older children may be better managed at home. Infants need to be put on respiratory isolations and they will require close monitoring of their respiratory status.

Antibiotics are recommended during the catarrhal stage but if the infection has progressed, antibiotics have little benefit. The antibiotic of choice is erythromycin. Corticosteroids and albuterol can be used to assist with the coughing episodes (Mott et al., 1990).

NURSING CARE OF CHILDREN WITH PERTUSSIS

Nursing care is generally supportive.

- Children in the acute phases will need cardiorespiratory monitoring on a pulse oximeter. The limits on the oximeter should be checked each shift. Monitors should be explained to parents to assist in relieving their anxiety when the alarms beep. Suction and oxygen should be set up at the bedside for immediate use. If children need oxygen therapy, this should be explained to parents. If children have paroxysmal spells, their oxygen levels should be closely monitored and parents must be given reassurance as these spells can be frightening to observe.
- Children may experience exacerbated coughing as a result of loud noise or fright so attempts should be made to keep the environment quiet. Rest is important for children with pertussis and activities should be offered that are diversional but minimise tiring.
- Children's nutritional needs must be observed. Small frequent feeds should be offered, especially for young children, because they will become exhausted with large feeds. If nutritional intake is poor, gavage or parenteral nutritional routes may be considered. If children have been vomiting, they may need extra oral care.
- Parental support and education are important. Parents may need support to deal with grief emotions especially if they have chosen not to immunise their children (Adams et al., 1996).

EDUCATION FOR CARERS OF CHILDREN WITH PERTUSSIS

- Parents need to encourage quiet activities to preserve their children's energy.
- Fluid intake should be encouraged by offering ice blocks, lemonade etc.
- If no humidifier is available, humidified air can be provided by putting children in a bathroom filled with mist from a shower.
- Parents need to offer small and frequent meals rather than large meals.
- Good handwashing is important to prevent the infection spreading to others.
- Parents need to be aware of the signs and symptoms that indicate the need for medical attention (Wong, 1993).

15. RESPIRATORY SYNCYTIAL VIRUS BRONCHIOLITIS (RSV)

Bronchiolitis is an inflammation of the bronchioles. It causes 50 per cent of hospitalisation in children aged younger than one year (Ashwill and Droske, 1997). RSV was first discovered in 1956, when it was isolated in a chimpanzee with coryza (Katz et al., 1998).

E Etiology of RSV

RSV has a worldwide distribution but it is difficult to find seronegative individuals beyond the toddler age group in any population. It is the most important viral respiratory pathogen of infancy and childhood. The peak incidence of the disease occurs in the first year of life with the most serious illnesses happening in the first six months. Disease in the neonatal period is uncommon but there have been documented epidemics in neonatal units.

RSV produces epidemics in late winter and early spring but in the tropics, the epidemic pattern is less clear (Behrman et al., 1992). Bronchiolitis and pneumonia due to RSV are more common in boys than girls by a ratio of 1.5:1. Racial factors do not affect RSV infection rates. Lower respiratory tract disease occurs early in life and usually in low socioeconomic groups and in crowded living conditions (Behrman et al., 1992).

Transmission of RSV

RSV is spread by close or direct contact with secretions, including airborne droplets (Schwartz, 1995). RSV can live on paper or skin for up to one hour and on cribs and other nonporous surfaces for up to six hours. Although it is non-airborne, it is highly communicable and infection is generally spread by hands (Ashwill and Droske, 1997). Infection usually occurs through conjunctival or nasal epithelia as the end result of random collisions between viruses and target cells. Following attachment and absorption into the cell, the virus spreads along the epithelium, mainly by cell-to-cell transfer. This causes a fusion of infected

cells which form large masses called syncytia. It is this process that gives the virus its name (Purrsell and Gould, 1997). The incubation period from exposure to first symptoms averages about four days with a range of 3–7 days. The virus remains viable in secretions from three days to four weeks depending on the severity of the illness and the immunological status of the child (Schwartz, 1995).

Understanding how the virus is spread enables nurses to take the necessary precautions to prevent nosocomial infection, while avoiding unnecessary restrictions. RSV is spread mainly through contact with infected secretions and large-particle aerosols. It does not spread over long distances. Self-inoculation by touching the eyes or nose after having contact with infected secretions promotes dissemination. RSV does not spread easily via the oral route so masks are not helpful in controlling the spread (Purrsell and Gould, 1997).

Combinations of cohort nursing, wearing gowns and gloves and education have been shown to reduce the nosocomial spread of the infection. The routine use of gloves is debatable because handwashing with soap will destroy the virus. Although cohort nursing is recommended, it can be difficult to organise and relies on rapid isolation of the virus by laboratory testing. If children are suspected of having RSV, isolation precautions should be implemented to minimise the spread of infection (Purrsell and Gould, 1997).

P Pathophysiology of RSV

In bronchiolitis caused by RSV the oedema and collection of mucous cause an obstruction of the bronchioles. Infants' bronchioles are very narrow and quickly become obstructed. Airway resistance is increased during the inspiratory and expiratory phases of respiration because of the small airways. Hyperinflation of the lungs results from air trapping because bronchioles constrict during expiration. Atelectasis can happen if the airway obstruction occludes the airways and trapped air is reabsorbed. Normal exchange of gases is impaired and the infant becomes hypoxic. Some infants will develop respiratory alkalosis but more often the infant will develop metabolic acidosis. An infant with bronchiolitis is most acutely ill in the first 48–72 hours after the onset of the disease and improvement is a gradual process (Wong, 1993).

Abnormalities in pulmonary function can persist for years after an acute RSV infection and are measurable by arterial blood gases. Current research is examining the relationship of bronchiolitis and the subsequent occurrence of chronic asthma. About 40 per cent of children who have had proven bronchiolitis have been reported to develop asthma in later years. The most likely agent is thought to be a potent stimulator of wheezing in the asthma-prone individual. It is also possible that the virus may be a factor in chronic lung disease in adults (Schwartz, 1995).

Predisposing conditions for RSV

Certain underlying conditions make infants susceptible to RSV infection. These include:

- premature birth
- neonatal oxygen requirements
- congenital heart disease (especially cyanotic heart disease with increased pulmonary flow)
- chronic pulmonary disease, the most frequent being bronchopulmonary dysplasia followed by cystic fibrosis
- immunocompromised status, including organ transplant recipients, those on chemotherapy or those with congenital neutropenia.

Males up to the age of five years are more likely than females to have RSV infections and will require hospitalisation more than females for management of the condition (Purrsell and Gould, 1997). There may be anatomical reasons why males have more lower respiratory tract involvement with RSV infections. Males are more likely than females to have smaller airways and decreased conductance which is related to lung- and chest-wall elasticity (Wong, 1993).

An environmental factor that affects the incidence rates of RSV infections is crowding. Two or more people sharing a room with an infant is a risk factor. Crowded child care centres also increase infants' risk of contracting RSV, especially those in the 7–9-month age group (Schwartz, 1995).

C Clinical presentation of RSV

The first sign of RSV infection in children are rhinorrhea and pharyngitis. A cough may appear simultaneously but more often occurs one to three

days after the onset of rhinorrhea. At this time there may be sneezing and a low-grade fever. Soon after the cough has developed, children will wheeze audibly. If the disease is mild, these symptoms may not progress beyond this stage. Auscultation often shows diffuse rhonchi, fine rales and wheezes. Rhinorrhea and fevers usually persist throughout the illness (Behrman et al., 1992).

If the illness progresses, some of these clinical symptoms may manifest:

- Cough, wheezing and air hunger increase.
- There is evidence of hyperexpansion of the chest.
- Intercostal and sub costal retraction occur.
- Nasal flaring may be present and is a sign of acute respiratory compromise.
- The respiratory rate increases and cyanosis occurs.

Signs of severe, life-threatening illness are:
- central cyanosis
- tachypnoea of more than 70 breaths per minute
- lethargy
- apnoeic phases.

At this stage the chest may be very hyper expanded and silent to auscultation because of poor air and gas exchange. Children will experience feeding difficulties because of the increased respirations which interfere with sucking and swallowing (Ashwill and Droske, 1997).

Chest x-rays of children hospitalised with RSV bronchiolitis are normal in about 10 per cent of cases; air trapping or hyperexpansion of the chest is present in 50 per cent of cases. Peribronchial thickening or interstitial pneumonia is seen in 50–80 per cent of cases and segmental consolidation occurs in 10–20 per cent of cases. Pleural effusions are rare. In some children, the progression of the illness may be more like that of pneumonia. In these cases, the prodromal rhinorrhea and cough that are followed by dyspnoea, poor feeding and lethargy may result in a diagnosis of RSV bronchiolitis (Behrman et al., 1992).

Temperatures are an inconsistent symptom of RSV bronchiolitis and can vary from hyperthermic to as high as 41 degrees (Ashwill and Droske, 1997). Rash and conjunctivitis occur in a few cases. In young children, especially those who are premature, periodic breathing and apnoeic spells have been common signs even in a mild case of bronchiolitis. It is likely that a small number of deaths included in the sudden infant death syndrome are due to RSV infection (Behrman et al., 1992).

D Diagnosis of RSV

The only definitive RSV diagnostic test is laboratory isolation of the virus or antigens in the respiratory secretions. Before proceeding to viral cultures or antigen testing, the issues of practicality, cost effectiveness and clinical relevance of the information obtained must be considered. Viral cultures or antigen detection methods may be considered if:

- RSV is suspected during a non-epidemic season
- recurrence of illness is within a relatively short period of time
- ribavarin therapy is being considered.

Because of sensitivity, nasopharyngeal washings (known as nasopharyngeal aspirates) are preferred over nasopharyngeal swabs for obtaining specimens. Confirmation of RSV infection through viral cultures or antigen testing is usually done only on children who need hospitalisation for their respiratory illness.

Routine laboratory tests offer minimal information in most cases of RSV. At first, the white cell count is slightly elevated or normal and the differential count may be normal or shifted from either right or left. Once the disease is established, a lower leucocyte count with lymphocytosis may be noted. Bacterial cultures are usually of little assistance (Wong, 1993).

Non-invasive oxygen saturations may be the best method of determining the severity of the RSV infection and can be a more effective assessment tool than monitoring the respiratory compromise in most children. A reading of less than 95 per cent in room air at rest or feeding can indicate a more severe RSV infection. In mild cases of RSV infection, chest x-rays are usually normal. In about half the children with severe RSV infection, chest x-rays show trapping of air or hyperexpansion of the chest, peribronchial thickening or pneumonia.

In a child younger than one year old presenting with tachypnoea and wheezing, the diagnoses of asthma, pneumonia and congestive cardiac failure must be considered (Schwartz, 1995).

M Management of RSV

Uncomplicated RSV infection involving only the upper respiratory tract may be able to be treated at home. Treatment consists of supportive measures such as increased intake of fluids, humidification of air and the use of antipyretic medications to soothe children and decrease body temperature. If children are to be treated at home, it is important to reassess them within a 24-hour period. Oxygen saturation by pulse oximetry should be part of the initial assessment and revaluation (Schwarz, 1995).

Essential to outpatient treatment is an evaluation of resources available to the carers of children. Social situations need to be considered with regard to the reliability of the carers and their willingness to return to the hospital for ongoing evaluation or if children deteriorate. The carers must be able to generally assess the work of breathing and describe the signs and symptoms of respiratory distress as well as be able to evaluate children's hydration level.

If children deteriorate, hospitalisation should be considered. Hospitalisation is recommended if any of the following is present:

- severe respiratory distress evidenced by rib retractions
- nasal flaring and grunting respirations
- a respiratory rate of more than 70 breaths per minute
- apnoea or cyanosis
- dehydration or poor oral intake
- less severe symptoms than those described above if a child has a previous medical condition.

NURSING CARE OF CHILDREN WITH RSV

- Continually assess oxygenation and administer humidified oxygen as indicated by the vital signs. Aim to keep oxygen levels between 93 and 95 per cent.
- Respiratory evaluation must include assessment of respiratory distress (tachypnoea, dyspnoea, rib retraction, cyanosis and nasal flaring) and auscultation of breath sounds. Respiratory assessment should be ongoing, with documentation every hour during the acute phase. Apnoea monitoring is required in acute phases of the disease. Nurses must check monitors to be sure that the alarms are set so that any periods of apnoea are documented.
- Keep children close to the nurses station for close observation.
- Perform laboratory tests for the presence of RSV or antigens.
- Take a chest x-ray to exempt pneumothorax, pneumomediastinum, atelectasis or bacterial pneumonia.
- Assess children for signs of dehydration. This includes checking for dry mucous membranes, decreased urine output, sunken fontanelle and weight loss. Depending on the assessment findings, intravenous fluids or nasogastric feeding may be required to achieve euvolemic status. Monitor body temperature closely as it can cause insensible water loss. The temperature in oxygen cribs should be monitored as well as the moisture in the crib, in the tubing and on the bedding and/or infant.
- Consider ribavarin therapy. Ribavarin is an antiviral drug and is usually indicated only for children who have congenital heart disease, bronchopulmonary dysplasia or cystic fibrosis; children with deteriorating respiratory function with hypoxia and hypercapnia; premature babies; and immunosupressed children. Ribavarin has few side effects although it is tetragenic in nonprimates so it should not be used by pregnant women. Use antibiotics only if bacterial infection is suspected (Purrsell, 1996; Ashwill and Droske, 1997).

Prognosis

The mortality of hospitalised children with RSV infection is about 2 per cent. The prognosis is worse in premature, young children or those with a comorbidity of the neuromuscular, pulmonary, cardiovascular or immunologic systems (Behrman et al., 1992).

EDUCATION FOR CARERS OF CHILDREN WITH RSV

Nurses can teach carers how to care for children with RSV in their own homes. The following tips are adapted from Wong (1993).

- The signs and symptoms of respiratory distress include tachypnoea (fast breathing), chest retraction (drawing in of the chest when breathing) and cyanosis (bluish discolouration of the skin and mucous membranes).
- Air can be humidified through the use of a humidifier in children's bedrooms.
- Steam vaporisers should be avoided because of the danger of burns. Fill the humidifier with warm water and children will breathe in the warm mist.
- Mucous can be suctioned by using a bulb syringe. Secretions can be loosened by using three drops of warm saline in each nostril and waiting a minute before using the bulb syringe.
- Fluids should be encouraged. Small frequent formula or breast feedings should be offered if children tire when feeding. If coughing makes children vomit, they should be refed. Increase fluid intake through the use of play, for example, tea parties with small cups and teapots, may encourage children to drink.
- Smoking should be avoided in the presence of children with a respiratory illness.

16. TUBERCULOSIS (TB)

One in every three people on earth is believed to be infected with *Mycobacterium tuberculosis*. Seven to eight million cases of active TB are treated annually, resulting in three million deaths each year (Ginsberg, 1998). The incidence of TB had declined in the middle of the twentieth century but it began to rise again and in 1985 the World Health Organisation declared TB a global crisis.

E Etiology of TB

When a person who has active TB coughs or sneezes, tiny droplets are dispersed into the air. Inhalation of the bacilli-laden droplet nuclei and their consequent infection of the lung tissue in the bronchioles and alveoli is required for successful transmission of the disease (Adams et al., 1996).

According to Wong (1993), children are at risk of TB infection if they have close contact with:

- people who have HIV
- intravenous drug users
- poor or medically ignorant city residents
- residents of nursing homes.

The risk of infection in children is increased by the following factors:

- having contact with another person who has active TB
- having a chronic illness, being immunosupressed or having HIV
- suffering malnutrition
- being an infant or an adolescent
- belonging to nonwhite ethnic and racial groups
- living in substandard living conditions
- being an incarcerated adolescent.

P Pathophysiology of TB

TB is most often transmitted by the inhalation of contaminated droplets coughed up by or expelled from an adult with infectious pulmonary TB.

The most common site of organism implantation is the respiratory tract. The bacillus multiplies in lung tissue, alveoli and regional lymph nodes. After an incubation period of 2–10 weeks, hypersensitivity develops. At that time, children who have been infected will test positive on a skin test. Most infected children are asymptomatic at the time of the first skin test (Katz et al., 1998).

The active disease state of TB is different from chronic TB infection by the presence of clinical symptoms. The risk of developing TB disease is between 5 and 10 per cent over a lifetime and the risk time is two years after infection. Months or even years may pass between the infection and development of the disease. Usually, untreated infections lie dormant and never progress to the disease phase in a healthy person. Children's immunologic response is usually enough to keep the bacteria from multiplying and spreading. If the host response is adequate, the organism is walled off and the tubercle becomes a healed calcified mass. TB bacilli can stay dormant and cause active disease at a later time if the host becomes immunologically suppressed. If the lesion does not heal and is not walled off, it may enlarge and spread to other tissues or it may enter the blood and spread to the middle ear, brain, kidney, bones, joints and skin (Behrman et al., 1992).

TB destroys host tissue. When tubercle bacilli multiply they may damage tissue so badly that the centre of the infected area turns to liquid pus. When this liquid escapes into an airway, it is coughed up as sputum, leaving a tiny hole (cavitation) in the lung. This sputum is very infectious. Children rarely develop active pulmonary TB with cavitation and only in such instances is the child infectious to others. Children with primary pulmonary TB are not usually contagious because their lesions are small and their cough is minimal or absent (Behrman et al., 1992).

C Clinical presentation of TB

Children aged 3–15 years:
- are usually asymptomatic
- have normal chest x-rays
- are identified only through a positive skin test.

Some children develop:
- malaise
- fever
- slight cough
- weight loss and anorexia
- lymphadenopathy or more specific symptoms depending on the site of infection.

When doing an assessment on a child who may have TB, these symptoms must be assessed as well as the contacts the child has had recently (King and Tomasic, 1999).

D Diagnosis of TB

Skin testing is the initial method of screening and testing in children suspected of having TB. In most children, skin testing will produce a positive reaction 3–6 weeks after infection and sometimes as long as three months after infection Positive tuberculin reactivity usually continues for the person's lifetime, even with treatment.

There are two types of antigen preparations used for testing; old tuberculin (OT) and purified protein derivative (PPD). Both must be kept cold and in the dark to preserve their potency. Skin testing can be done by an intradermal Mantoux test or a multiple puncture test. The test should be done and read by a health care professional. The reading is based on induration (hardness) not erythema (redness). Results should be recorded in millimetres (mm) and not as 'positive' or 'negative'. Repeated Mantoux tests will not sensitise an uninfected person to TB and will not cause future Mantoux to be positive (King and Tomasic, 1999).

Interpretation of Mantoux test results

Current guidelines suggest that if a child has an area of induration of greater than 15 mm it is a positive sign. This includes children who have no known risk factors. Induration more than 10 mm is considered positive in children younger than four years of age, children with chronic illness or those who have a high environmental risk to TB. A reaction of 5 mm or greater is considered positive for the high-risk groups. Children with a

negative reaction to a Mantoux test may still have TB, especially young children (Katz et al., 1998).

The tests for children who return a positive test include regular chest x-rays, sputum collections and smears. Gastric washouts are sometimes done as children swallow sputum rather than spit it out. A thorough history should be done on children who are suspected of having TB, including all social contacts (Katz et al., 1998).

M Management of TB

TB management involves:
- destroying the bacilli with medication
- providing nutrition to boost immunity
- preventing exposure to infection which will further compromise children's immune state.

Children may need hospitalisation depending on:
- how ill they are
- their age
- the need for ongoing testing
- their social situation.

Isoniazid is given to prevent TB infection from progressing to active disease. A chest x-ray must be done prior to commencing therapy. Isoniazid is usually given to babies and children for nine months. If children have HIV, they need isoniazid for 12 months.

In children found to have a resistant strain of TB, rifampicin for nine months is recommended. If there is concern that children and/or carers will not comply for nine months with the daily medication regime required, support by the appropriate health care providers in the community must be enlisted, perhaps community nurses or liaison/ethnic health workers (Adams et al., 1996).

Two issues are of particular concern: the rising number of illegal immigrants moving into Australia and the global increase in multi-drug-resistant TB which develops in people who do not comply with their medication regime (Ginsberg, 1998). In fact the biggest problem in treating active TB is poor

compliance in taking medications. Because taking multiple medications can be confusing, pills that contain different combinations of the TB drugs have been developed in the last few years (King and Tomasic, 1999). Treatment is also complicated by the tendency of the bacilli to mutate into resistant strains. Drug resistance is an increasing problem and poor client compliance can result in the development of drug-resistant TB. If multiple drug resistance develops and treatment is ineffective, surgical resection can be an option (Mott et al., 1990).

Bacillus Calmette-Guerin vaccine is the only vaccine available for TB prevention but it varies in the immunity it provides (Mott et al., 1990).

Prevention of TB

Contact tracing is an important factor in preventing transmission of TB. Most children are infected by a family member so the best way of preventing transmission is to identify who is infected and to provide TB therapy.

Prevention includes:
- isolation of infected people
- administration of medication to household contacts of people with TB
- the administration of BCG vaccine to at-risk groups.

Early detection is accomplished by regular screening of children. Children in high-risk groups should be screened annually. A test should be done when a child has had contact with TB. If the test is negative, it should be repeated ten weeks later. Children remaining in contact with an infected person will require testing every three months (Katz et al., 1998).

NURSING CARE OF CHILDREN WITH TB

- Children's respiratory status should be assessed every 2–4 hours depending on how unwell they are.
- If children have a cough, the cough and sputum characteristics should be documented. Children will swallow mucous rather than expectorating, but mucous acts as an emetic so vomiting may be a management problem. Physiotherapy is recommended to assist expectorate sputum.
- Skin colour, activity level and breath sounds should be noted.
- Because children with TB are often not contagious, they are seldom isolated, but in some areas of increased incidence and when the children are very ill, they will require isolating to prevent nosocomial TB. Children and adolescents with infectious pulmonary TB, whose sputum cultures identify acid-fast bacilli, should be isolated until medication therapy has commenced and their sputum cultures show a decreased number of bacilli. Children with TB infection can attend school as long as they are receiving medication.
- A nutritious diet is recommended. Children can become anorexic and have significant weight loss so a dietitian may be needed to educate the carers and nurses on the most suitable foods.
- Children will need regular rest periods and quiet time. Quiet activities such as videos and books should be offered.
- An important nursing role is to educate the carers about the importance of compliance with drug therapy. Families must be aware that even though children may have no symptoms, the organism can lie dormant. Families need to understand the importance of completing treatment (Wong, 1993).

EDUCATION FOR CARERS OF CHILDREN WITH TB

Wong (1993) points out that family members need to be made aware of the importance of rest and a good diet, the correct administration of medication and the importance of completing the course of medications. To prevent the transmission of TB to others all family members must know to:

- cover their mouth and nose with a tissue when coughing
- burn contaminated tissues
- practise good hygiene and handwashing when handling sputum-infected material
- use disposable articles when possible (e.g. tissues not handkerchiefs)
- disinfect contaminated articles by boiling for five minutes or putting the items in the sun for twelve hours.

17. VARICELLA ZOSTER INFECTIONS (CHICKENPOX/SHINGLES)

Chickenpox is a disease of early childhood. It is the primary infection caused by the varicella zoster virus which is a type of herpes virus. Shingles, commonly seen in adults, is the secondary infection caused by the reactivation of the latent zoster virus (Braun, 1996).

E Etiology of varicella zoster

About 90 per cent of children contract chickenpox during the first ten years of life. Almost half the children entering school have had chickenpox. It is a highly contagious disease. In Australia it must be notified to the appropriate state or territory health department when a definite diagnosis has been made. If an infected individual is introduced into a household, almost 90 per cent of susceptibles develop the disease within the incubation period. The incubation period for chickenpox is usually about 14 days and 90 per cent of infections happen 11–20 days after household exposure. Children are believed to be contagious one day after the rash develops (Bolyard et al., 1998). The source of contagion cannot be documented because the virus is difficult to identify in respiratory secretions. In healthy children, the period of contagion is approximately five days after the onset of the skin lesions and until the lesions have crusted over. In children who are immunocompromised and develop progressive varicella, the virus can be recovered from vesicular lesions for a longer period of time and these children must be considered contagious for 7–10 days. Chickenpox is most common in the late winter and early spring. Transmission appears to happen more frequently in temperate than in tropical climates (Rudolph and Hoffman, 1987).

Transmission of varicella zoster

Although spread probably occurs by close person-to-person contact with lesions, transplacentally and airborne droplet spread has been documented. It is recommended children with varicella not attend school, day care or camp until six days after the onset of the rash or until all the lesions are crusted (Braun, 1996).

Hospitalised patients with varicella are placed in strict isolation for at least five days after the onset of the rash and for the duration of the vesicular eruption (Braun, 1996). Isolation requires carers to wear a mask, gown and gloves at all times. All contaminated materials must be bagged and labelled before reprocessing, or treated in accordance with the health agency's infection control practice protocol. Hands must be washed thoroughly after contact with the infected patient and before contact with another patient. Children exposed to varicella must be kept in strict isolation for 8–21 days after the onset of rash in the infected individual. At birth, all neonates with mothers who have active varicella infections should be placed in strict isolation. Isolation measures also pertain to secretion and drainage of the lesions and wounds the infected child may have (Ashwill and Droske, 1997).

Children who are in hospital should be isolated in a room where air pressure is negative in relation to the outside. The room should have an air exhaust unit that prevents recirculation of air into the hospital and the door to the isolation rooms should be kept closed. It is recommended that health personnel who may be pregnant should not come in contact with a child suspected of having varicella (Behrman et al., 1992).

For hospitalised children who receive varicella zoster immune globulin (VZIG), the isolation period is increased to 28 days after exposure. VZIG is available only in limited supplies and is recommended for the following groups of children as it can provide passive immunity against varicella zoster virus and can be used to minimise the risk of the child developing varicella:

- immunocompromised children, including children currently having immunosuppressive treatment (chemotherapy, radiation and corticosteroids)
- newborns of mothers exposed within five days before or 48 hours after the delivery (the mother may be given VZIG on exposure)
- premature infants of longer than 28 weeks' gestation, exposed postnatally
- premature infants of less than 28 weeks' gestation, regardless of maternal history of exposure.

If VZIG is indicated it must be given as soon as possible after exposure and no later than 96 hours after exposure. It is administered as an intramuscular injection (Braun, 1996)

P Pathophysiology of varicella zoster

The following sequence of events is thought to occur when children become infected.

1. The virus enters through the respiratory mucosa and multiplies in the regional lymphatic tissue.
2. About 4–6 days after infection, a low-level primary viraemia occurs, allowing the virus to infect and multiply in the liver, spleen and other organs.
3. Some 10–12 days after infection, a secondary viraemia happens, and the rash reaches the skin. The rash results, on average, 14 days after infection.

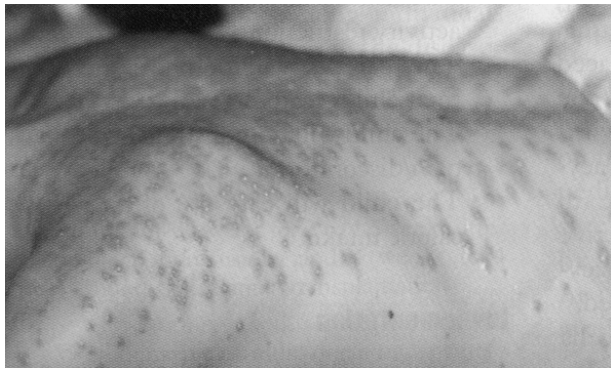
Viraemia which is evident in the clinical symptoms of varicella is harder to find in immunocompromised children. The skin lesions of varicella begin as macules, progress to papules, vesicles, pustules and crusts over a few days. Most lesions are only on the epidermal layer and they are formed by an accumulation of fluid derived from dermal capillaries which fill the space created by degenerating epidermal cells (Katz et al., 1998).

One attack of chickenpox usually results in permanent immunity. After recovery from varicella, the viral infection persists in the absence of clinical symptoms in a latent form. This virus can be reactivated and result in zoster. Little is known about the maintenance of latency or the factors that cause reactivation of the virus (Rudolph and Hoffman, 1987).

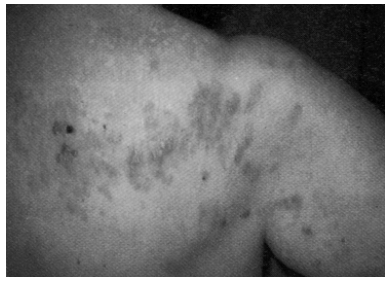
C Clinical presentation of varicella zoster

Healthy children who develop varicella often have no prodromal symptoms. The first sign of illness may be pruritus or the appearance of vesicles. Vesicles usually appear first on the trunk, scalp and face. The rash then spreads to the extremities. Lesions can be seen in various phases of maturation, with macules, papules and vesicles present in an area at the same time. The vesicular lesions, which are a few millimetres in size, are superficial and can be ruptured easily. Later, as the disease progresses, an erythematous base can be observed surrounding the vesicles. Excoriation is normal because of the pruritic nature of the

lesions. New lesions will erupt for about 4–5 days and the number of spots will vary. The vesicles will then become crusty. Late in the disease only crusts will remain, with the occasional deep-seated vesicular lesion. Mucous membrane involvement is not common but ulcerative lesions can be present in the mouth and, less commonly, on the conjunctiva and pharynx. Fever usually lasts 3–4 days with an average temperature of 38.5 degrees Celsius. Higher or persistent temperatures should make health professionals focus on other sources of infection. The white blood cell count is not usually affected by the disease but elevated transaminase levels have been reported in some children (Rudolph and Hoffman, 1987).



Chickenpox



Shingles

Figure 15.1 Chickenpox and shingles lesions, and rash distribution

(From Moschella S. L., & Hurley, H. J. [1992]. *Dermatology* [3rd ed.]. Philadelphia: W.B. Saunders, p. 219, Fig. 8–25D. Used by permission.)

In contrast to the mild illness that occurs in healthy children, progressive varicella can occur in children with malignancies or those who are immunocompromised. These children may have constantly high temperatures of up to 41 degrees Celsius even a week after the eruption starts. Late in the disease, the lesions may become more prominent on the extremities than on the trunk. They may be umbilicated and quite deep. Hepatitis, encephalitis and pneumonia can develop. About half the children who have leukaemia, who have no prophylaxis and are untreated, develop progressive varicella, with a mortality rate of 20 per

cent. Usually death is a result of pulmonary involvement (Rudolph and Hoffman, 1987).

If varicella develops in a pregnant woman the foetus can be affected. First-trimester infection may result in many different congenital malformations characterised by atrophy of a limb, usually with cicatrised skin. Infected children usually suffer intrauterine growth retardation. Cortical atrophy and microcephaly are common. Autonomic nervous system involvement can result in difficulty with sphincter control, intestinal obstruction or Horner syndrome. Eye anomalies include cataracts, microthalmia and chorioretinitis. Infected children usually die within the first year of life. Children who survive are generally significantly neurologically impaired, with extensive motor and intellectual deficits. The risk of a foetus being damaged after maternal infection is small. Children whose mothers have varicella at any stage of the pregnancy or children who get varicella during the first few months after birth have a higher risk of getting zoster during the first few years of life than other children (Krugman et al., 1998).

D Diagnosis of varicella zoster

Varicella usually poses no problems to diagnose. Children with a characteristic rash who have few symptomatic signs of illness during late winter or early spring but a history of exposure are easily identified as having chickenpox (Potter and Perry, 1997).

Differential diagnoses of varicella zoster virus include:
- disseminated herpes simplex
- impetigo
- other viral exanthems
- insect bites
- scabies.

Smallpox used to be the most important disease to differentiate from chickenpox but today it is no longer a diagnostic consideration. Disseminated herpes simplex can resemble chickenpox rash, but the history and progression of the illness usually differentiate between the two viruses. Impetigo can produce bullae that resemble varicella but they do not appear in clusters, differ in appearance and distribution and do not involve mucous membranes of the mouth. Viral exanthems such as

Coxsackievirus and Echovirus can produce vesicular exanthems. However, they usually follow a different course of development and do not crust. Insect bites and scabies can cause confusion at times if they are vesicular but they will not be present on the mucous membranes (Braun, 1996).

Generalised zoster can be differentiated from varicella by a history of vesicular lesions localised to 1–3 dermatomes for several days prior to the generalised eruption. Usually a concentration of lesions in a dermatomal distribution is evident at time of dissemination. Generalised herpes simplex can be confused with varicella. Generalisation usually occurs in immunocompromised individuals. Varicella encephalitis can be difficult to distinguish from Reye syndrome, which is associated with abnormal liver function tests and elevated blood ammonia levels (Rudolph and Hoffman, 1987).

Laboratory tests can be used for confirmation but are not often used which may make laboratory staff unfamiliar with the possible testing. Tests include direct electron microscopy, immunoflourescence staining of the vesicular fluid and a Tzanck smear prepared from scraping the base of the lesion. But it is thought that virus isolation is not practical for diagnosing varicella. The virus can be found in lesions for up to four days after the first lesions occur. Cytopathic effects do not appear in cultures until five days after inoculation. Generally, varicella is very difficult to isolate and relies on observation to make the diagnosis (Braun, 1996).

When laboratory isolation can be difficult, it means the questions asked must be specific to get an accurate history, and health professionals must have a sound rationale for the questions they are asking. Ashwill and Droske (1997) recommend the following questions be asked when children are suspected of having been in contact with varicella:

- Is there a history of exposure to chickenpox (school, day care, among family members)?
- Has there been a recent outbreak of chickenpox in the community?
- Have the children had chickenpox before?
- Did the children have a history of low-grade temperatures, malaise, anorexia, upper respiratory tract infections and/or headaches before they developed the lesions?

- What do the lesions look like?
  - Are they raised red pimples or bumps?
  - Are they filled with fluid?
  - Is there crusting and or scabbing?
  - Did the lesions start on the child's trunk and spread to the face and scalp?
  - Are there lesions on the lips, tongue and throat?
  - Are the lesions more abundant on clothed areas or in areas of local inflammation such as the nappy area?
  - Are the lesions itchy?

Prognosis

The prognosis is usually good. Fatalities are usually the result of complications (Behrman et al., 1992).

M Management of varicella zoster

Children will require a negative-pressure room and contact isolation. Gloves should be worn on entering the room and a gown is necessary if close contact is expected with infected children. Immune health workers do not need a mask but those not immune should wear a mask. Children should remain in isolation until all lesions are dry and crusted (Weber et al., 1996). Staff caring for children with varicella should familiarise themselves with the infection control policy of their hospital. Symptomatic treatment should be directed at alleviating the itching by using local and systemic antipruritic agents and sedation as needed. The effects of scratching will be minimised if children wear mittens and their fingernails are kept short. Daily changes of clothes and linen, and antiseptic baths reduce the incidence of secondary bacterial infection (Behrman et al., 1992). Lukewarm soaks, oatmeal or cornstarch can also be used to provide relief from itchiness (Braun, 1996). Topical products such as calamine may exacerbate skin infections because using it on weeping skin may cause caking and make children pick at it more (Potter and Perry, 1997).

Antihistamines may be used for severe pruritus. However, knowledge of dosage and the potentially toxic effects of nonprescription medicine is important. Topical application of some antihistamines requires care as

applying lotion all over the body can result in potential drug toxicity because of the combined use of topical and oral antihistamine drugs (Braun, 1996).

If secondary infection occurs, intravenous antibiotics will be required. Because the use of aspirin in children who have varicella increases the risk of developing Reye syndrome, other antipyretics should be used when symptomatic relief is necessary (Behrman et al., 1992). A substantial number of deaths that occur in otherwise healthy children are related to Reye syndrome or varicella pneumonia (Braun, 1996).

Acyclovir is effective therapy for varicella pneumonia or immuno-compromised children who develop varicella. Acyclovir interferes with virus replication and leads to a decrease in symptoms of varicella. Acyclovir therapy does not shorten isolation time so its economic advantage in otherwise healthy children is negligible. An accurate dosage prevents the development of pneumonia or the involvement of other viscera. The best results are obtained when therapy is started before the third day of the illness (Behrman et al., 1992).

Complications of varicella zoster

The most common complications of varicella are a staphylococcal or streptococcal cellulitis and impetigo due to secondary bacterial infections caused by scratching. Pneumonia is rare in healthy children. Neurologic complications following varicella include:

- transverse myelitis
- optic or peripheral neuritis
- Guillain Barre syndrome
- aseptic meningitis
- encephalitis.

Encephalitis occurring a few days after the rash is often accompanied by acute cerebral oedema with a fulminating course. Encephalitis can include signs of ataxia, tremor and nystagmus. The prognosis if encephalitis develops is usually good unless there is severe central nervous system involvement presented by convulsions and coma. Children with these complications may have long-term difficulties including seizures, mental retardation, and or behavioural disorders.

Acute cerebellitis usually occurs late during the first week of the rash (or later) and generally has a good prognosis.

A significant number of cases of Reye syndrome are said to be preceded by chickenpox.

Corneal involvement may occur if lesions affect the eye.

According to Ashwill and Droske (1997), respiratory complications include:

- viral and bacterial pneumonia
- upper respiratory tract infections
- otitis media.

Various haematologic manifestations have been associated with chickenpox including thrombocytopenic purpura.

Rare complications have included orchitis, arthritis and nephritis (Rudolph and Hoffman, 1987).

EDUCATION FOR CARERS OF CHILDREN WITH VARICELLA ZOSTER

- Most children with varicella are treated at home. They should be isolated from all people who may be susceptible to being infected with the virus (those who are immunocompromised, elderly or pregnant). Parents should notify the school and or day care facility.
- Fever should not be controlled with aspirin.
- Pruritus can be managed with antihistamines and special baths. Nails should be kept short and clean to help prevent secondary infection. Nurses may recommend using, for comfort, mittens and clothing that is light, soft and in one piece. Daily baths and clean linen may help prevent infections of the lesions.
- Children who have lesions in their mucosa must have their hydration monitored. Offering fluids that are soothing such as iceblocks and jelly may be beneficial. Acidic and scratchy foods may irritate the mouth so the diet should be soft and bland.
- An ophthalmologist should be consulted if children develop lesions of the cornea.
- Diversional activities during the infectious period can help children cope with the isolation from their normal activities and friends. The activities chosen should reflect the children's level of wellness and hold their interest. It is recommended that activities should not cause overheating or irritation of the skin.
- Parents should be educated to recognise the signs and symptoms of complications. They should also have a good understanding of the importance of preventing the spread of varicella, especially among individuals who are highly at risk for complications from the disease (Ashwill and Droske, 1997).

APPENDIX EXAMPLE OF NOTIFIABLE DISEASES

Health authorities usually publish a list of diseases which are notifiable, that is, anyone finding someone with one of the listed diseases must notify the relevant health authority.

Below is a list of the notifiable diseases in the state of New South Wales in Australia. Notification is required within 24 hours of diagnosis. All notifications are strictly confidential.

AIDS
Acute viral hepatitis
Adverse event following immunisation
Botulism
Cholera
Cancer (to NSW Cancer Registry)
Diphtheria
Foodborne illness (where there are two or more linked cases)
Gastroenteritis (any age, in an institution)
Haemolytic uraemic syndrome
Haemophilus influenzae Type B
Invasive infections
Legionnaires' disease
Leprosy
Measles
Meningococcal disease
Paratyphoid
Pertussis (whooping cough)
Plague
Poliomyelitis
Rabies
Syphilis
Tetanus
Tuberculosis
Typhoid
Typhus (endemic)
Viral haemorrhagic fever
Yellow fever

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INDEX

Dealing with children who have or may be exposed to infectious diseases takes extra special care. Immature bodily systems cannot handle exposure to disease in the same way as adult systems. Action must be taken swiftly but carefully and all those involved with the sick child must know what they are doing.

The particular audience for this book is not nurses who work all the time with children (though they will find it a quick and easy reference). This book is for nurses who work only occasionally with children, nurses who need easy access to information about infectious diseases as they affect children, e.g. incubation times, isolation methods and notification requirements.

An important difference about this book is that it views nurses and the child's family as members of the same team. Each chapter of the book includes a section on Education for Carers, being those at home who are looking after a child with an infectious disease. The information for nurses to pass on to families is direct and helpful; in fact the book itself can be recommended to families with young children as a handy reference.

In the author's own words, 'this book is up to date and applicable in busy clinical areas'. It is highly recommended for all nurses working in clinical situations.

Dr Tara Walker's experience in paediatrics has been a combination of study and practice. Her studies have resulted in a Diploma and Bachelor of Health Science in Nursing from the University of New England — Northern Rivers and a Bachelor of Health Science in Nursing with honours and a doctorate from Southern Cross University in Lismore, New South Wales. Her practical experience includes eight years of work in paediatrics in a rural setting, the last two of those as a nurse unit manager.

It was from this clinical work in particular that Tara realised the need for a good textbook on infectious diseases in children. Her unit at the Lismore Hospital is the referral unit for all ill children in the Northern Rivers Area Health Service of New South Wales so she had plenty of cases to draw on to decide what would be most useful in the book and how it ought to be presented.

Fired by the experience of having written this book, Tara hopes to continue her combined career of academic investigation of and clinical management in paediatrics and she wants to write another book.

ISBN 0 9577988 7 3