Overview of Viral infections

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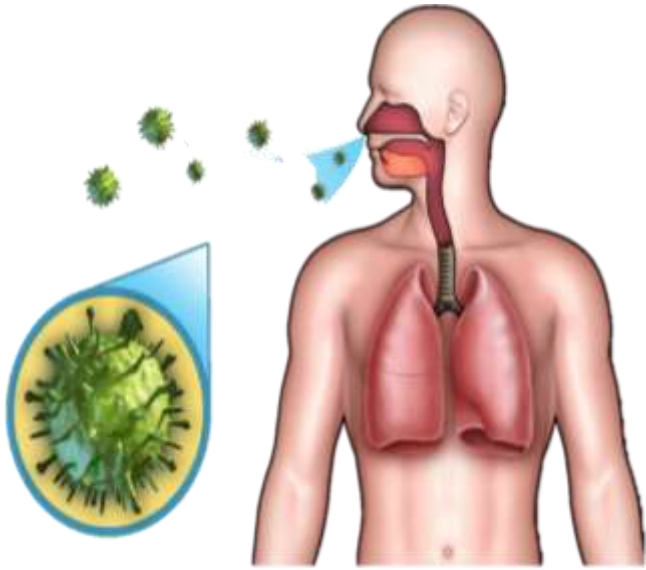
Overview of Acute Viral Respiratory Infections

viruses gain access to the human body primarily in the form of aerosolized droplets or saliva, despite normal host protective mechanisms, including

- **the mucous covering most surfaces,**
- **ciliary action,**
- **collections of lymphoid cells,**
- **alveolar macrophages,**
- **secretory IgA.**

Many infections remain localized in the respiratory tract, although some viruses produce systemic spread (eg, **chickenpox, measles, rubella**).



The symptoms depend on whether the infection is concentrated in the upper or lower respiratory tract. **Definitive diagnosis requires**

- **isolation of the virus,**
- **identification of viral gene sequences,**
- **demonstration of a rise in antibody titer,**

the specific viral disease can frequently be deduced by considering the major symptoms, the patient's age, the time of year, and any pattern of illness in the population.

The severity can range from **inapparent** to **overwhelming**. The most severe illness is usually seen in infants **(paramyxoviruses)** and in elderly or chronically ill adults (**influenza virus**).

Table 30–4. Viral Infections of the Respiratory Tract.

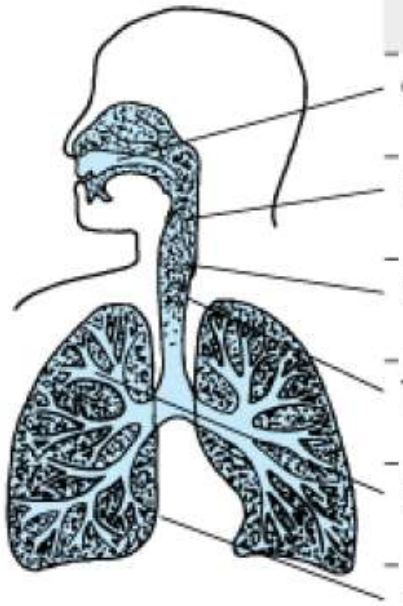
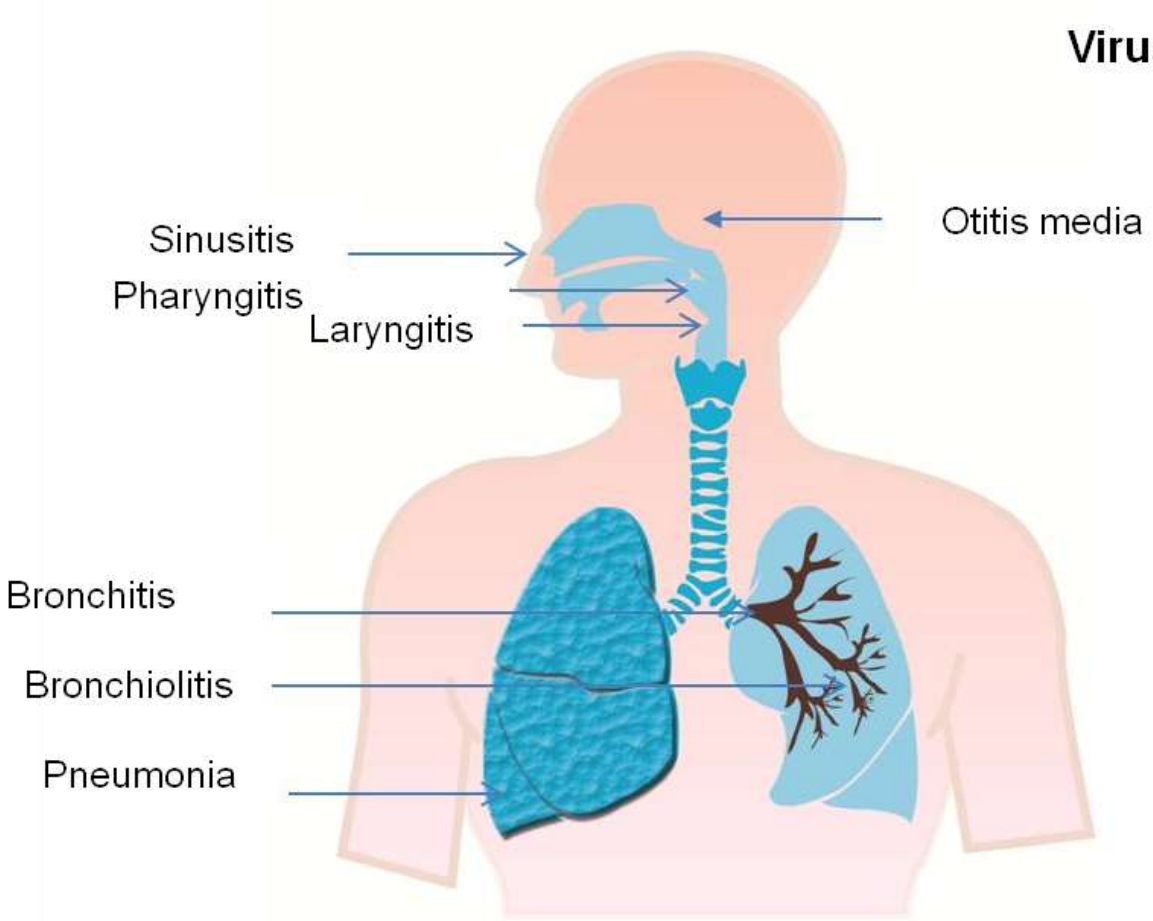


Table 30–4. Viral Infections of the Respiratory Tract.

Syndromes	Main Symptoms	Most Common Viral Causes		
		Infants	Children	Adults
Common cold	Nasal obstruction, nasal discharge	Rhino Adeno	Rhino Adeno	Rhino Corona
Pharyngitis	Sore throat	Adeno Herpes simplex	Adeno Coxsackie	Adeno Coxsackie
Laryngitis/croup	Hoarseness, "barking" cough	Parainfluenza Influenza	Parainfluenza Influenza	Parainfluenza Influenza
Tracheobronchitis	Cough	Parainfluenza Influenza	Parainfluenza Influenza	Influenza Adeno
Bronchiolitis	Cough, dyspnea	Respiratory syncytial Parainfluenza	Rare	Rare
Pneumonia	Cough, chest pain	Respiratory syncytial Influenza	Influenza Parainfluenza	Influenza Adeno



Viruses that infect the upper respiratory tract

Rhinovirus
Coronavirus
Influenza virus
Parainfluenza virus
Respiratory Syncytial virus
Herpesvirus
Adenovirus
Bocavirus
Coxsackivirus

Viruses that infect the lower respiratory tract

Influenza virus
Parainfluenza virus
Respiratory Syncytial virus
Adenovirus
Bocavirus
Metapneumovirus

Overview of Viral Infections of the GIT

A few agents, such as herpes simplex virus and Epstein-Barr virus, probably infect cells in the mouth. in the intestinal tract it exposed to harsh elements (acid, bile salts, and proteolytic enzymes). Consequently, viruses are all acid- and bile salts-resistant.

There may also be virus-specific secretory IgA and nonspecific inhibitors of viral replication to overcome.

Acute gastroenteritis is with symptoms ranging from mild, watery diarrhea to severe febrile illness characterized by vomiting, diarrhea, and prostration. Rotaviruses, Norwalk viruses, and caliciviruses are major causes of gastroenteritis. Infants and children are affected most often.

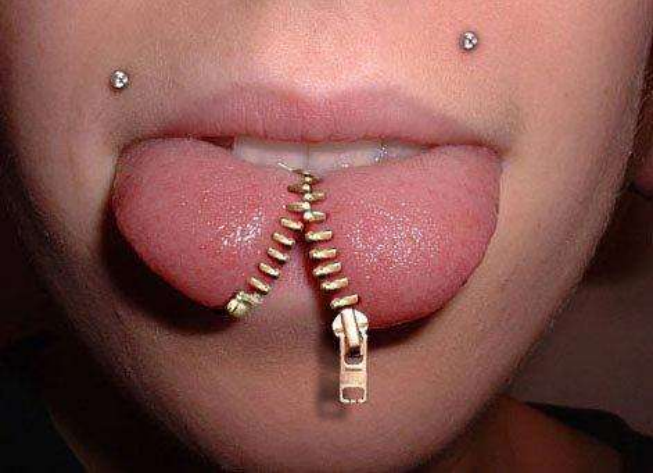
Some enteric viruses utilize host proteases to facilitate infection (proteolytic digestion of viral capsid that then facilitates events such as virus attachment or membrane fusion).

Enteroviruses, coronaviruses, and adenoviruses gastrointestinal infections are often **asymptomatic**. Some enteroviruses (polioviruses, and hepatitis A virus) are important causes of systemic disease but do not produce intestinal symptoms.

Overview of Viral Skin Infections

The skin is a tough and impermeable barrier to the entry of viruses. However, a few viruses are able to breach this barrier and initiate infection of the host through:

- small abrasions of the skin (poxviruses, papilloma-viruses, herpes simplex viruses),
- the bite of arthropod vectors (arboviruses)
- infected vertebrate hosts (rabies virus, herpes B virus),
- injected during blood transfusions or via contaminated needles, such as acupuncture and tattooing (hepatitis B virus, HIV).



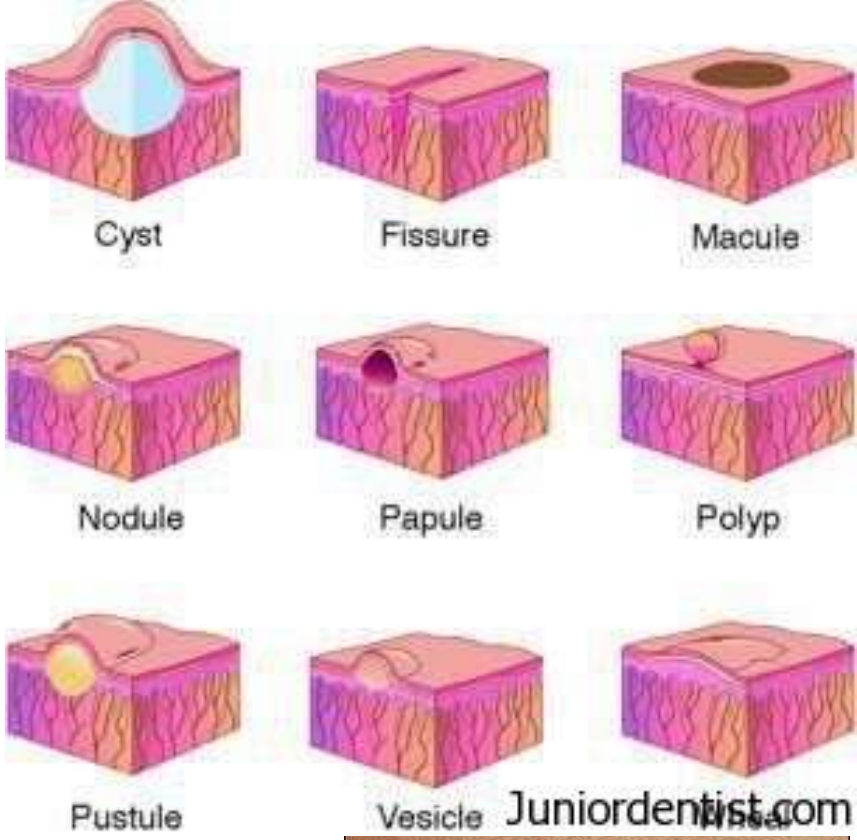
Some agents produce localized lesions (papillomaviruses and molluscum contagiosum); most spread to other sites. The epidermal layer is devoid of blood vessels and nerve fibers, so viruses that infect epidermal cells tend to stay localized.

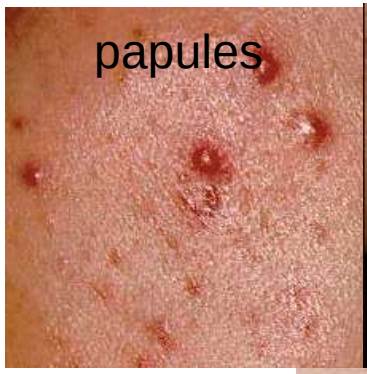
Viruses introduced deeper into the dermis reach blood vessels, lymphatics, dendritic cells, and macrophages and usually spread and cause systemic infections.

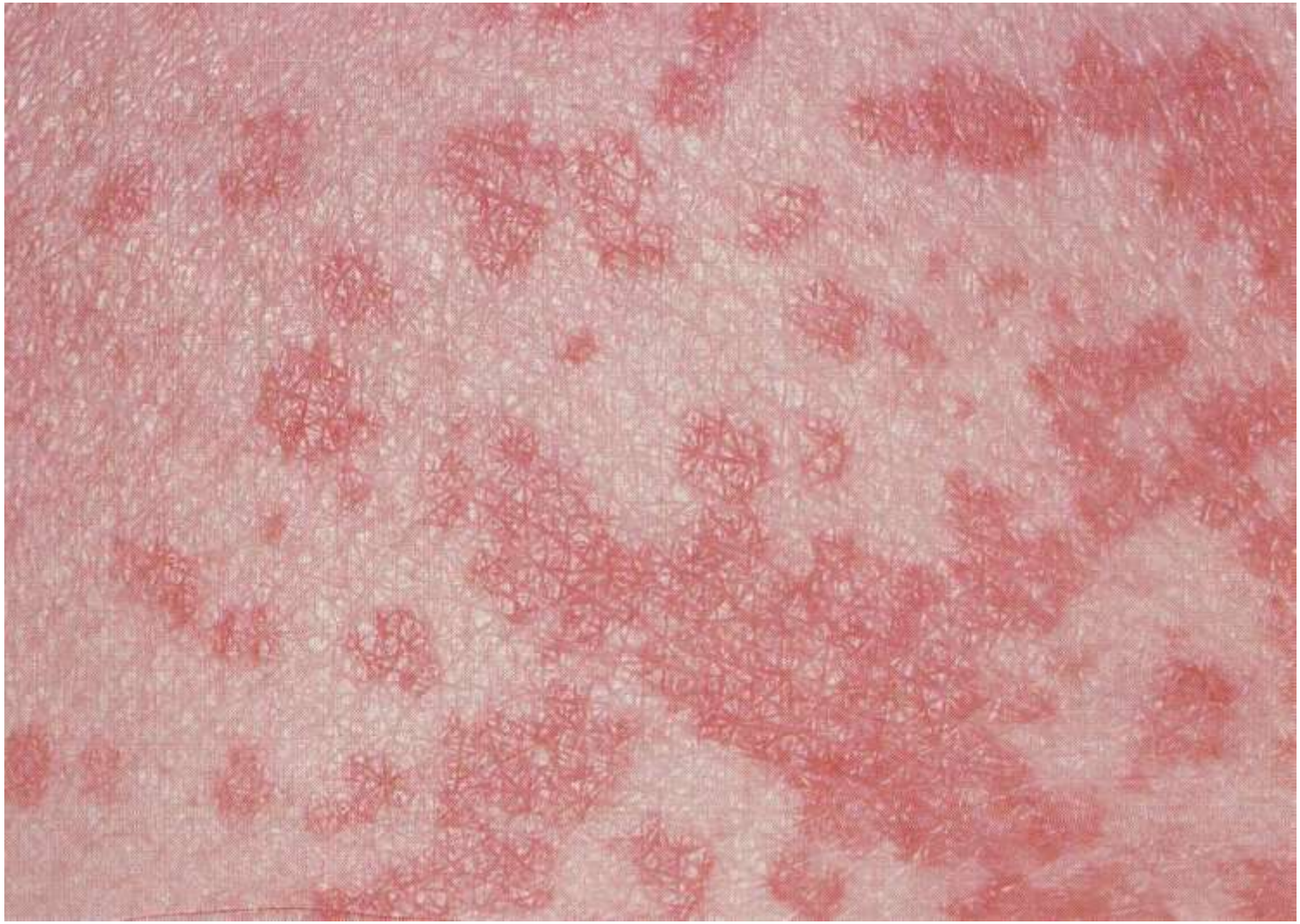
Such infections originate by another route (eg, measles virus infections occur via the respiratory tract), and the skin becomes infected from below.

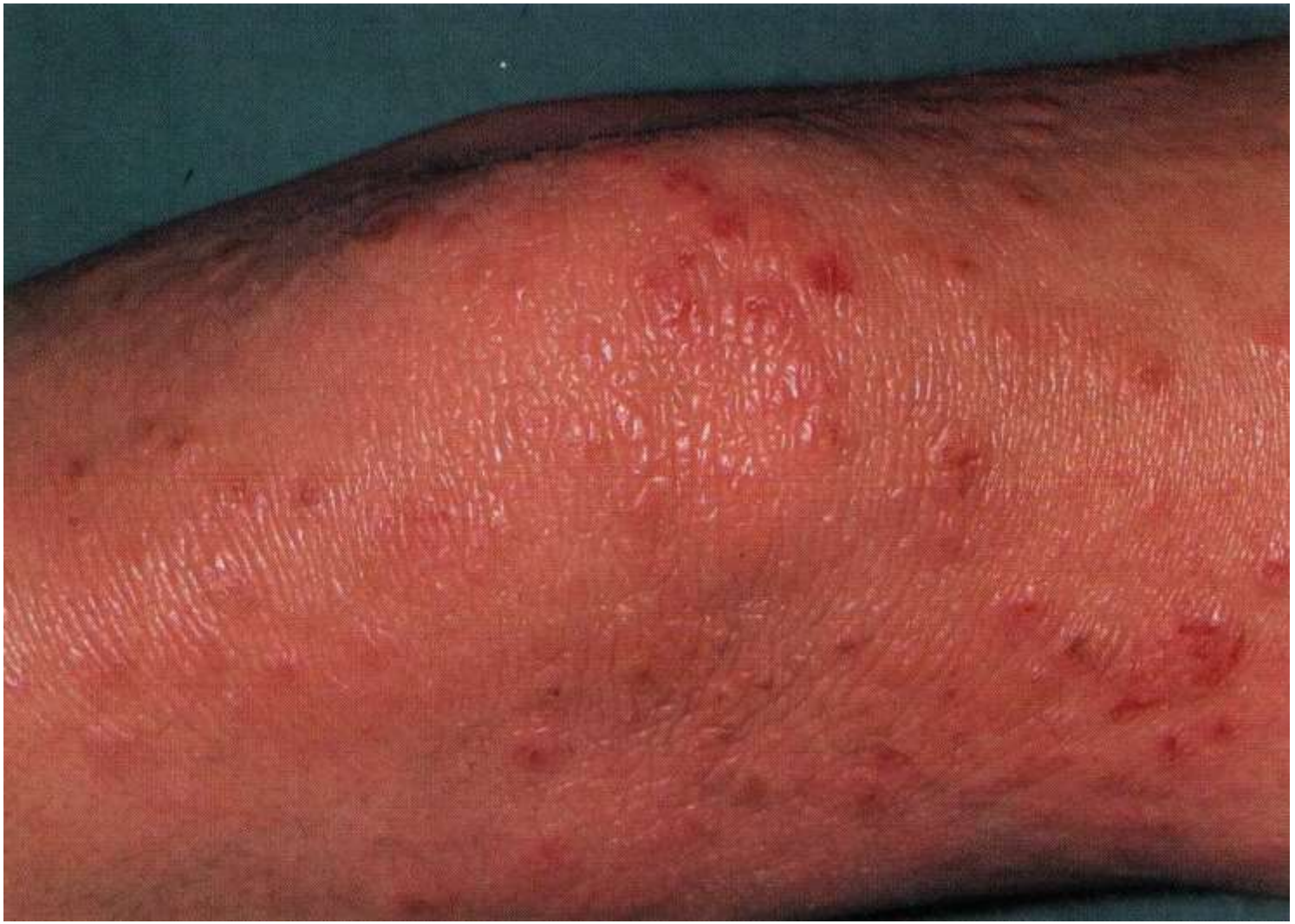
Lesions in skin rashes are designated as macules, papules, vesicles, or pustules.

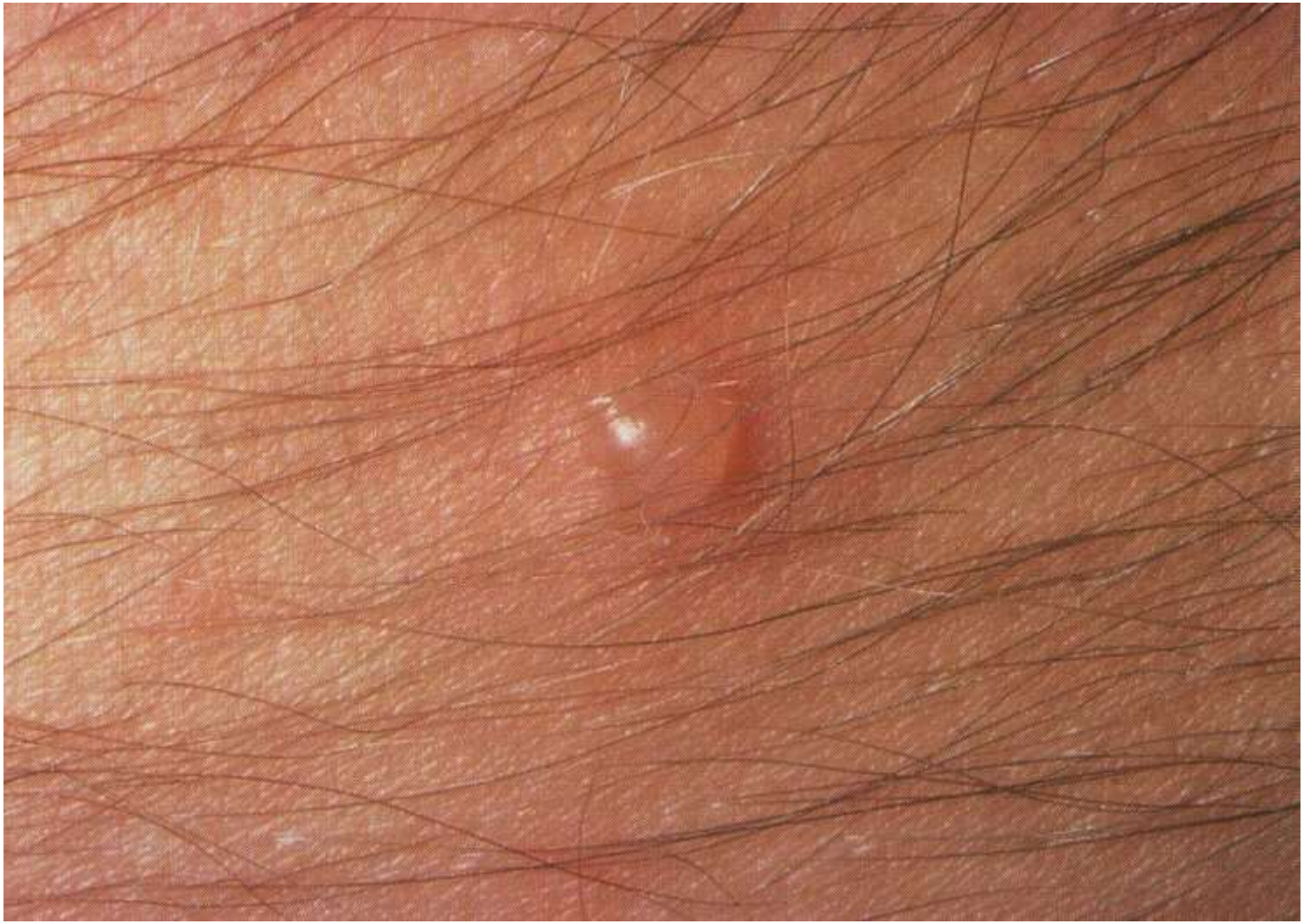
Macules, which are caused by local dilation of dermal blood vessels, papules if edema and cellular infiltration are present in the area. Vesicles occur if the epidermis is involved, and they become pustules if an inflammatory reaction delivers polymorpho-nuclear leukocytes to the lesion. Ulceration and scabbing follow. Hemorrhagic and petechial rashes occur when there is more severe involvement of the dermal vessels.











Infectious virus is not shed from the maculopapular rash of measles or from rashes associated with arbovirus infections.

In contrast, skin lesions are important in the spread of poxviruses and herpes simplex viruses (high titers in the fluid of these vesiculopustular rashes, and they are able to initiate infection by direct contact with other hosts).

Overview of Viral Infections of the CNS

viral infection of CNS is always a serious matter. Viruses can gain access to the brain by two routes:

- by the bloodstream (hematogenous spread)
- by peripheral nerve fibers (neuronal spread).

blood spreading may occur by:

- through endothelium of small cerebral vessels,
- passive transport across the vascular endothelium,
- through the choroid plexus to the cerebrospinal fluid,
- transport within infected monocytes, leukocytes, or lymphocytes.

Once the blood-brain barrier is breached, more extensive spread throughout the brain and spinal cord is possible.

via peripheral nerves.

Virions can be taken up at sensory nerve or motor endings and be moved within axons, through endoneural spaces, or by Schwann cell infections.

Herpes viruses travel in axons to be delivered to dorsal root ganglia neurons.

The virus may utilize more than one routes of spread. Many viruses, including herpes-, toga-, flavi-, entero-, rhabdo-, paramyxo-, and bunyaviruses, can infect the central nervous system and cause meningitis, encephalitis, or both.

Encephalitis caused by herpes simplex virus is the most common cause of sporadic encephalitis in humans.

Pathologic reactions to cytocidal viral infections of the central nervous system include necrosis, inflammation, and phagocytosis by glial cells.

The postinfectious encephalitis (after measles infections after rubella infections) is characterized by demyelination without neuronal degeneration and is probably an autoimmune disease.

The neurodegenerative disorders, called slow virus infections, are uniformly fatal. Features of these infections include a long incubation period (months to years) followed by the onset of clinical illness and progressive deterioration, resulting in death in weeks to months; usually only the central nervous system is involved. Some slow virus infections, such as progressive multifocal leuko-encephalopathy (JC (John Cunningham) polyomavirus) and subacute sclerosing panencephalitis (measles virus), are caused by typical viruses.

Overview of Congenital Viral Infections

Viral diseases produce in the human fetus. Most maternal viral infections do not result in viremia and fetal involvement. if so, serious damage may be done to the fetus.

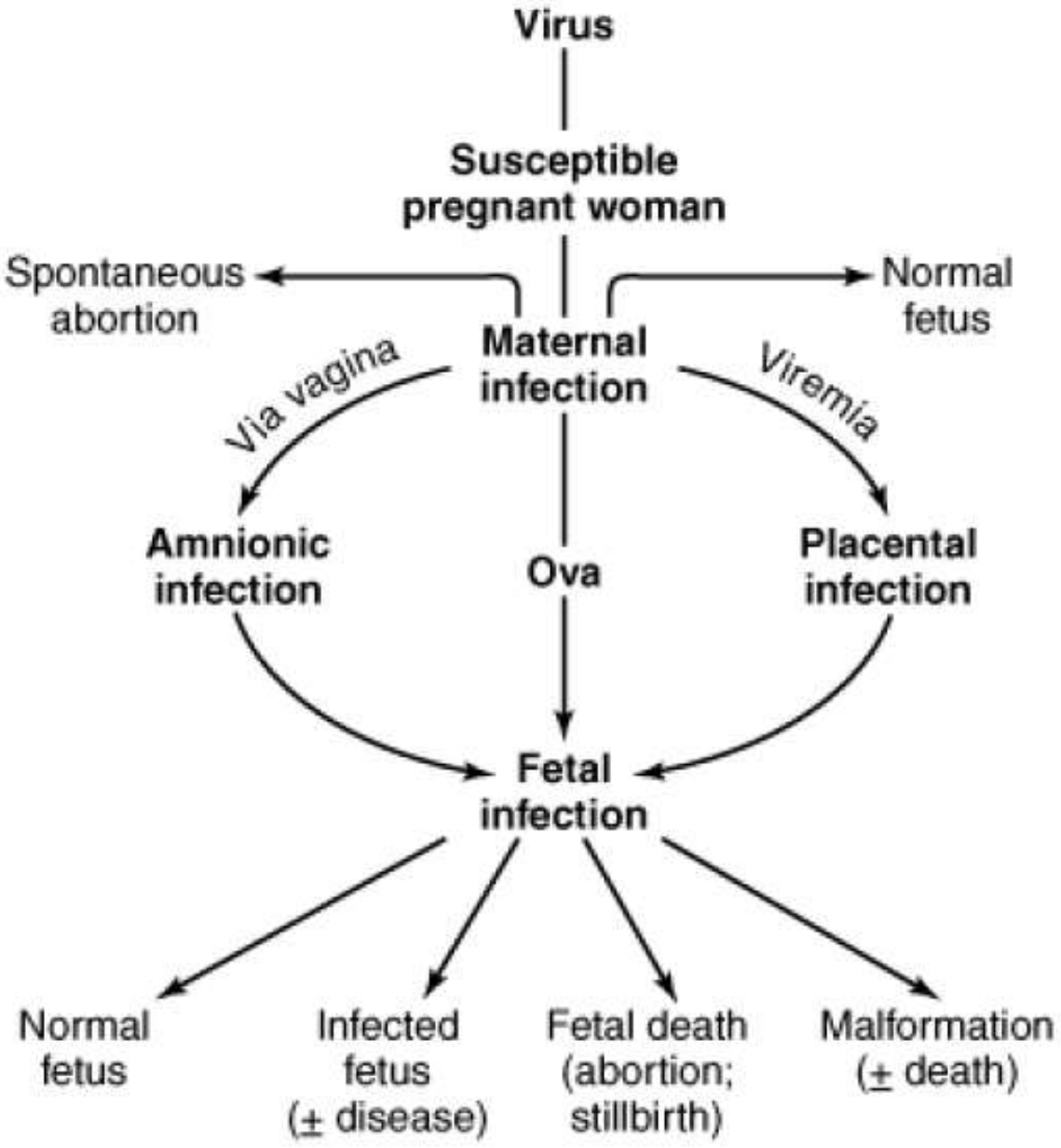
Three principals involved in the production of congenital defects are:

(1) the ability of the virus to infect the pregnant woman and be transmitted to the fetus;

(2) the stage of gestation at which infection occurs;

(3) the ability of the virus to cause damage to the fetus directly, or indirectly, by infection of the mother resulting in an altered fetal environment (eg, fever). The sequence of events that may occur prior to and following viral invasion of the fetus

Figure 30–6.


Virus
Susceptible pregnant woman
Spontaneous abortion
Normal fetus
Maternal infection
Via vagina
Viremia
Amnionic infection
Ova
Placental infection
Fetal infection
Normal fetus
Infected fetus (± disease)
Fetal death (abortion; stillbirth)
Malformation (± death)

primary agents responsible for congenital defects (Rubella virus and cytomegalovirus). Or can occur by (herpes simplex, varicella-zoster, hepatitis B, measles, and mumps virus and with HIV, parvovirus, and some enteroviruses) (Table 30–5).

Table 30–5. Acquisition of Significant Perinatal Viral Infections.

	Frequency of Time of Infection			
Virus	**Prenatal (In Utero)**	**Natal (during Delivery)**	**Postnatal (after Delivery)**	**Neonatal Incidence (Per 1000 Live Births)**
Rubella	+	-	Rare	0.1–0.7
Cytomegalovirus	+	++	+	5–25
Herpes simplex	+	++	+	0.03–0.5
Varicella-zoster	+	Rare	Rare	Rare
Hepatitis B	+	++	+	0–7
Enterovirus	+	++	+	Uncommon
HIV	+	++	Rare	Variable
Parvovirus B19	+	-	Rare	Rare

In utero infections may result (fetal death, premature birth, intrauterine growth retardation, or persistent postnatal infection).

Developmental malformations (congenital heart defects, cataracts, deafness, microcephaly, and limb hypoplasia. Fetal tissue is rapidly proliferating. Viral infection and multiplication may destroy cells or alter cell function. Lytic viruses, such as herpes simplex, may result in fetal death. Less cytolytic viruses, such as rubella, may slow the rate of cell division. If this occurs during a critical phase in organ development, structural defects and congenital anomalies may result.

Viral infections may be transmitted from the mother during delivery (natal) from contaminated genital secretions, stool, or blood. Less commonly, infections may be acquired during the first few weeks after birth (postnatal) from maternal sources, family members, hospital personnel, or blood transfusions.

Effect of Host Age

Host age is a factor in viral pathogenicity. More severe disease is often produced in newborns. In addition to maturation of the immune response with age, there seem to be age-related changes in the susceptibility of certain cell types to viral infection. Viral infections usually can occur in all age groups but may have their major impact at different times of life. Examples include rubella, which is most serious during gestation; rotavirus, which is most serious for infants; and St. Louis encephalitis, which is most serious in the elderly.