Viral Infections of the Newborn

Frances Strodtbeck, RNC, DNS, NNP

Viral infections of the newborn result in significant morbidity and mortality each year. The fetus and newborn are particularly vulnerable to viral infection. The range of expression may vary from no clinical disease to devastating illness and infection occurring before, during, or after birth. Nursing management is determined by the specific viral infection, the severity of the illness, and the unique conditions of the newborn and his/her family. Promising new therapies are on the horizon that may lessen the severity of viral disease. Until such time, the major thrusts of management of neonatal viral disease are prevention of infection and supportive care for the acutely ill newborn.

Viral infections of the newborn are a serious concern; such infections result in significant morbidity and mortality each year. The incidence of viral infections in the newborn is estimated at 6–8% of all live births (Smith, 1993). The fetus and newborn are particularly vulnerable to viral infection for numerous reasons, including a developing immune system that is inadequate for preventing infection and containing the spread of viruses, lack of immunologic experience with viruses, and the presence of rapidly growing cells and tissues (Strodtbeck, 1986). Viral infection in the newborn can vary from no clinical disease to devastating illness.

Infection of the newborn with a virus produces a variety of clinical presentations, ranging from absence of symptoms to infection before (congenital), during (natal), or after birth (postnatal) (Overall, 1992). Because of the affinity of viruses for rapidly growing cells, newborn infection results in multiple outcomes that are determined by the specific virus and the gestational age at the onset of infection. Congenital viral infections may result in miscarriage or stillbirth, congenital defects of various organ systems, clinical infection, or asymptomatic infection. Natal and postnatal infections can result in asymptomatic infection or clinical infection. Outcomes of clinical infection can vary from recovery to persistent infection with or without sequelae to death (Overall, 1992; Smith, 1993).

This article reviews the unique pathogenesis of newborn viral infection and the response of the neonatal immune system to viral attack; current information on the clinical features of specific viral diseases is summarized. The diagnosis, management, and nursing care of the newborn with a viral disease are addressed. The first goal and ultimate management of neonatal viral disease is prevention of the infection.

Pathogenesis of Viral Infection

Viruses are unique microorganisms in that they are obligatory intracellular parasites that must gain access to the inside of a host cell for replication. Physical contact is necessary for this process, and many factors influence the attraction between the host cell and the invading virus. The structure of an individual virus and the presence of certain enzymes determine the mechanism by which the virus replicates inside the host cell. Viral replication follows five basic steps.

1. attachment or adsorption of the virus to the host cell;
2. penetration of the virus into the host cell cytoplasm and uncoating of the viral genome;
3. viral-directed synthesis of viral nucleic acids and proteins using host cell components and metabolism;
4. assembly of new virions in the cytoplasm; and
5. release of the new virions into the intracellular environment. Release of the virus can occur quickly or slowly and frequently results in destruction of the host cell (Strodtbeck, 1986; Voyles, 1993).

Some viruses are capable of integrating their genome into the host cell DNA, producing a new structure known as a "provirus" (Voyles, 1993). The provirus is committed to one of three outcomes: latency, controlled growth,
or lysis. Latency occurs when the provirus reproduces itself along with the host cell such that all daughter cells contain the viral genetic material. These cells can remain latent or be triggered into one of the other two outcomes at a future time. A classic example of latency is the herpes virus, which can remain dormant until a trigger such as stress activates the virus and a characteristic herpetic skin lesion is produced. In the controlled growth outcome, the host cell is not destroyed but becomes committed to the production of new virions, which are gradually released into the surrounding interstitial space. The last outcome, lysis, involves destruction of the host cell upon release of the newly formed virions. This process is referred to as the cytopathic effect and often results in patterned tissue damage that can be used in the clinical laboratory for identification of the virus (Strodtbeck, 1986; Voyles, 1993).

Many of the drugs developed for viral disease also have an effect on rapidly growing cells, which limits their use in the newborn.

From this discussion, it should be clear why viral infections can result in such a wide array of presentations. Because of the intracellular, parasitic nature of viruses and the resulting dependence on host cell metabolic machinery for proliferation, the use of antiviral drug therapy is limited. Many of the drugs developed for viral disease also have an effect on rapidly growing cells, which limits their use in the newborn. Thus, the newborn is not only vulnerable to infection, but also is disadvantaged for viable treatment options.

Newborn Immunologic Response to Viral Infection

The newborn infected with a virus is capable of mounting an immune system response. The response depends upon the gestational age of the newborn and is not as efficient or effective as the response of an adult. Clinical viral infection in newborns usually results in a more rapid progression to full-blown disease and earlier onset of symptomatic organ involvement than would be seen in adults with the same infection (Overall, 1992; Smith, 1993; Strodtbeck, 1986).

Entry of the virus into the newborn triggers activation of the developing immune system. Local antibodies such as IgA are stimulated. Because of their inability to contain the virus, a primary focus of infection is established. The virus replicates itself and releases new virions into the interstitial space, where they are carried away via the lymphatics or the bloodstream to secondary foci of infection. If tissue damage occurs at this point, the inflammatory and cell-mediated responses are activated. Escaping virions in the bloodstream stimulate the production of circulating IgM and IgG antibodies. IgM tends to be produced earlier if the virus is new to the immune system, whereas IgG tends to be produced first if the virus is a repeat challenger. The rise in fetal IgM during intrauterine infection allows fetal or cord blood levels to be used in the diagnosis of congenital viral infection. IgM production peaks early in the infection and rapidly declines. IgG peaks gradually and functions longer than IgM. Both of these immunoglobulins attempt to neutralize the invading virus by binding with the virus to activate complement. This mechanism is designed to prevent virus absorption to host cells (Overall, 1992; Strodtbeck, 1986).

Once the virus gains entry to the host cell, it becomes the responsibility of the cell-mediated system and the inflammatory response to contain the infection. Infected host cells develop new virus-induced, antigenic determinants on their cell membranes that alert these defenses. The new antigens trigger T lymphocytes to respond along with macrophages and other components of the immune system (Overall, 1992; Strodtbeck, 1986). Activation of the immune response at this point may result in the destruction of some host cells, rather than the virus, by the immune system. In the developing fetus, this can worsen the severity of birth defects associated with specific viral diseases, such as rubella or cytomegalovirus (CMV). For example, neurons coated with CMV may be destroyed by lymphocytes which, when added to the number of neurons undergoing viral lysis, increases the risk for microcephaly.

Antigen-antibody complexes also are formed that liberate chemotactic factors and cytokines. Histamine and kinin release results in vasodilation, heat production, and increased capillary permeability. This results in increased temperature (fever) and stagnant anoxia locally, which decreases absorption of the virus because most viruses are susceptible to increased temperature and acidic environments (Strodtbeck, 1986; Voyles, 1993). A side effect of this process is a less-than-ideal environment for the growth of healthy tissue.

The final consideration in the newborn's response to viral infection is the production of interferon. Interferons are highly active proteins produced by virus-infected cells that act upon neighboring cells to prevent the spread of the viral infection. They also are produced by cells of the immune system. Interferons disrupt viral infectivity in a variety of ways that are still being studied. Newborn interferon production appears to be decreased via one pathway and normal via the other (Strodtbeck, 1986). Despite the deficiencies of the newborn immune system to viral attack, only a few of the hundreds of viruses known to cause human disease actually result in infection of the fetus or newborn (Overall, 1992).

Specific Viral Disease in the Newborn

Viral infection of the newborn usually is classified as congenital, acquired, or nosocomial (hospital-acquired) in
Diagnosis of Viral Infection in the Newborn

Diagnosis of viral infection in the newborn usually is prompted by a strong suspicion based on physical characteristics of the newborn, history of exposure or maternal illness, or failure of sepsis testing to yield positive results. Careful review of the maternal history for evidence of viral illness and serum antibody determinations can assist in making the diagnosis; however, definitive diagnosis is based on neonatal studies (Overall, 1992; Smith, 1993).

Accurate diagnosis often is difficult. Because many infected infants have no symptoms or the clinical manifestations are nonspecific, laboratory diagnostic tests must be used to determine the virus responsible for the illness. Laboratory diagnosis may involve direct isolation of the virus in culture; detection of viral antigen or antibodies by immunologic, genetic, or electron microscopic study; or histopathologic methods (Guerina & Goldmann, 1993; Overall, 1992; Smith, 1993). The best method of diagnosis varies, depending upon the specific characteristics of the virus. Recovery of virus from clinical specimens usually is more difficult than recovery of other microorganisms (Strodtbeck, 1986). A negative result may mean an unsuccessful recovery of virus present. Clinical judgment is important for determining the likelihood for additional management and treatment of the suspected viral illness.

Management of Viral Infection

The most common management strategy for newborn viral infection is supportive care. The availability of specific chemotherapies such as antiviral drugs is limited. Promising new therapies are being developed, and several are undergoing evaluation in clinical trials with select populations (Filippell & Rearick, 1993; Kinney & Eiden, 1994; Overall, 1992; Stagno, 1994). Such therapies include ganciclovir for CMV disease, hyperimmune intravenous globulins for CMV and respiratory syncytial virus (RSV) infections, and new vaccinations for rotavirus. Most of these therapies, if approved for use in newborns, will most likely lessen the severity of the disease and subsequent sequelae, rather than cure the infection. Other strategies include improvements in nutrition for the most susceptible newborns, interferon administration to enhance viral-specific immune defenses, and lymphokine enhancement of the neonatal immune system.

The first goal and ultimate management of neonatal viral disease is prevention of the infection. Diligent hand washing; improved screening of visitors and health care providers who come into contact with susceptible newborns; increased efforts to immunize susceptible populations, such as childbearing women and children, to known viral pathogens, such as rubella and measles; and immunization of health care providers who work with immunocompromised patients are strategies that can be used to achieve this goal. The American Academy of Pediatrics (1994) recommendation that all neonatal intensive care unit (NICU) staff members receive annual influenza immunizations to prevent the spread of infection to infants with chronic lung disease is routinely ignored by most units across the country (Eisenfeld et al., 1994).

Nursing Care of the Newborn with a Viral Illness

Nursing care of the newborn with a viral illness is determined by the specific viral infection, the severity of the illness, and the unique conditions of the newborn and his/her family. Newborns with a known viral infection who have no symptoms require no special nursing care while in the hospital. Newborns with viruses such as rubella and CMV who have no symptoms should be followed up closely for the development of late onset se-
Table 1. Features of Specific Viral Infections in the Newborn

| Virus                     | Incidence/Epidemiology | Viral Characteristics | Transmission Risk (+ major route, + minor route, ? suspected route) | Effect on Newborn | Diagnosis | Clinical Features of Infection | Treatment | Prognosis | Prevention |
|---------------------------|------------------------|-----------------------|---------------------------------------------------------------------|------------------|-----------|--------------------------------|-----------|-----------|------------|
| Cytomegalovirus (CMV)     | 1% of all newborns, no seasonal variation | Member of herpesvirus family; causes latent infection, frequent episodes of reactivation or reinfection, prolonged viral shedding for years after infection, has special affinity for the CNS, eyes, lungs, and liver | ++ Congenital + Acquired + Nosocomial (to infant) | + Nosocomial (blood products) | + Day care | +90% asymptomatic; 10% symptomatic with 90%–95% having serious sequelae | Viral isolation from urine | Congenital infection: petechiae, hepatoproliferative, hepatitis, meningitis, jaundice, chorionamnionitis (10%), thrombocytopenia, CNS calcification, acquired infection: hepatitis, nephropathy, malignant lymphoma, worsening respiratory status or pneumonia, gray pallor, atypical lymphocytosis, and "septic" behavior | Supportive care for symptomatic infants; ganciclovir and CMV hyperimmune intravenous globulin are new therapies that have not been well studied in neonatal disease; follow-up hearing testing. | Asymptomatic infants: 5%–15% risk for developmental sequelae, including 5% hearing loss, 2%–7% microcephaly by age 2 years, 6% visual defect, symptomatic: 90%–95% risk of developmental delay or hearing loss, 20% die within first year | Routine hand washing; use CMV-negative blood products for high-risk and premature infants; future strategies may involve use of CMV vaccines currently being used in several clinical trials |
| Enteroviruses (Coxsackie A & B, echovirus, enterovirus, poliovirus) | Seasonal peaks in summer and fall (July-September) | Member of picornavirus family; common cause of human infections in all ages | + Congenital + Congenital (coxsackie B) + Nosocomial | 26% mild disease, 74% severe disease with 1% death rate; 60–70% infected newborns are male | Viral culture from multiple sites; rapid growth in culture but identification of type takes longer; serologic assays | Viral culture from multiple sites; rapid growth in culture but identification of type takes longer; serologic assays | Usual presentation is mild, febrile illness; can mimic severe bacterial sepsis or meningitis; macular or maculopapular rash, vomiting, diarrhea, NEC, fever >39°C, apnea, seizures, DIC, myocarditis | Supportive therapy; intravenous gamma globulin may improve survival | Depends upon virus group, age of infant, severity of illness; mortality highest for coxsackie B group, moderate for echovirus group, low for coxsackie A group | Hand washing; enteric isolation for known/suspected cases; administration of poliovirus vaccine to NICU patients |
| Epstein–Barr virus        | 3% of pregnant women susceptible to virus; infection very rare | Member of herpesvirus family | Not reported | No evidence of congenital or newborn infection | Serologic assay | Not applicable | Not applicable | Not applicable | Not applicable |
| Hepatitis viruses:        |                         |                        |                                                      |                  |                        |                                                      |            |            |            |
| Hepatitis A (HAV)         | HAV: rare reports; HBV: 11% of pregnant women surface antigen positive; incidence varies with population and risk factors; HCV: rare reports; HDV: none reported; HEV: none reported | Five different types of virus with multiple antigens (core, surface); HBV infection most common. | HAV: + Acquired HBV: + Congenital ++ Acquired ++ Nosocomial (blood products) | Most asymptomatic with >90% becoming chronic carriers | Antigen detection assays | Usually asymptomatic; severe disease rare. | Infants of surface antigen-positive mothers should receive hepatitis B immune globulin (HBIG). | 1% mortality risk; increased risk for hepatocellular carcinoma later in life | Screening of all pregnant women; HBV immunization of all newborns, both infants of surface antigen-positive mothers after delivery to reduce risk of transmission |
| Hepatitis B (HBV)         |                         |                        |                                                      |                  |                        |                                                      |            |            |            |
| Hepatitis C (HCV)         |                         |                        |                                                      |                  |                        |                                                      |            |            |            |
| Hepatitis D (HDV)         |                         |                        |                                                      |                  |                        |                                                      |            |            |            |
| Hepatitis E (HEV)         |                         |                        |                                                      |                  |                        |                                                      |            |            |            |
### Herpes simplex virus
- **2-5 infections per 10,000 deliveries annually**
- **Causes latent state**
- **Most infants symptomatic**
- **Viral culture of skin vesicles, nasopharynx, blood, urine, CSF, conjunctiva; antigen detection assays**
- **50-70% present with disseminated disease; 30% develop encephalitis**
- **Acyclovir, vidarabine, supportive therapy**
- **Significant sequelae develop in: 30% localized infection, 50% disseminated disease, 70% encephalitis; mortality 40-80%**
- **Hand washing; avoid contact with skin lesions**

### Human immunodeficiency (HIV) virus
- **30% of infants born to infected mothers will develop disease**
- **Retrovirus**
- **Asymptomatic at birth, onset of symptoms occurs between 4 and 6 months of age**
- **Viral culture; antigen detection assays; polymerase chain reaction (PCR) genetic testing**
- **Usually fatal by 3–5 years of age**
- **Prevent infection in mothers; infected mothers in developed countries should avoid breastfeeding.**

### Influenza viruses
- **Seasonal, with peak during winter months**
- **Very contagious**
- **Mild illness; no documented evidence of congenital infection**
- **Antigen detection assays; viral cultures**
- **Supportive therapy**
- **Passive immunization**
- **Immunize infants with underlying cardiac or pulmonary disease after 6 months of age; NICU staff should receive annual influenza vaccine**

### Measles (Rubella)
- **0.4–0.6 cases per 10,000 pregnancies; seasonal peak incidence March-May**
- **Member of paramyxovirus family**
- **Variable**
- **History of exposure; characteristic rash**
- **Usually good in survivors**
- **Passive immunization of exposed newborns and pregnant women and their contacts; active immunization of children.**

### Mumps
- **1–10 cases per 10,000 pregnancies; seasonal peak incidence March-April**
- **Member of paramyxovirus family**
- **Asymptomatic; no documented evidence of congenital infection**
- **History of exposure; clinical evidence of parotitis**
- **Mild illness**
- **No specific therapy**
- **Routine vaccination of all children older than 1 year**

(continues)
| Virus                 | Incidence/Epidemiology                                                                 | Viral Characteristics                                                                 | Transmission Risk (++, major route; +, minor route; ?, suspected route) | Effect on Newborn                                                                 | Diagnosis                                                                 | Clinical Features of Infection                                      | Treatment                                                                 | Prognosis                                                                 | Prevention                                                                 |
|----------------------|----------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------------|---------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------|
| Parvovirus B19       | 7% of pregnancies with proven infection have adverse outcomes; seasonal peaks in winter and spring | Causes fifth disease or erythema infections; spread by direct contact with respiratory secretions | ++ Congenital infection resulting in abortion, stillbirth, or hydrops fetalis | Nonimmune hydrops                                                            | Serologic testing of mother and infant for antibodies; DNA hybridization; maternal history of rash illness or arthropathy | No infection; viral shedding of subclinical infection; gastroenteritis   | Supportive care                                                        | No adverse sequelae for survivors                                      | Hand washing; avoid exposure to suspected cases                           |
| Respiratory syncytial virus (RSV) | Peak seasonal incidence November-April.                                                     | Member of paramyxovirus family; highly contagious.                                      | + + Nosocomial; no documented evidence of congenital infection            | Significant respiratory disease if symptomatic                              | Viral culture of washings of the nasopharynx; antigen assay               | Asymptomatic; upper respiratory infection without fever; apnea, Pneumonia, Nasal congestion, and rhinorrhea | Aerosolized ribovarin for high-risk newborns (those with underlying lung and heart disorders, immunodeficient diseases, severely ill with oxygen compromise); RSV hyperimmune intravenous immunoglobulin in clinical trials and may be available soon | High mortality for those with underlying conditions                      | Hand washing; isolation of infants with rhinorrhea, unexplained apnea, or nasal congestion |
| Rotavirus            | Marked seasonal variation with peak during dry, colder months; peak incidence occurs in infants 6–24 months of age; 0–22% in US and Canada; higher in Europe | Three groups of rotavirus types: Groups A, B, C, most neonatal disease due to Group A viruses | + + Nosocomial; ++ Day care                                             | No infection; viral shedding of subclinical infection; gastroenteritis       | Direct exam of stool with electron microscope; antirotavirus antibody detection assays | Lethargy, poor feeding, irritability, mild diarrhea; newborns usually asymptomatic | Supportive therapy, rehydration and restoration of electrolyte balance | No sequelae                                                              | Hand washing                                                             |
Rubella

- 5-25% of childbearing women lack rubella antibody; peak incidence March-May
- Single antigenic type of virus resulting in development of effective vaccine; proven teratogen; virus shed for up to 1 year after infection
- ++ Congenital defects occur in 100% of fetuses <11 weeks, 50% between weeks 11 and 20, 37% between weeks 20 and 35, and none >35 weeks; two-thirds asymptomatic in neonatal period
- Congenital heart defects (CHD), cataracts, glaucoma, microphthalmia, radioulnar synostosis, low birth weight, hepatomegaly, splenomegaly, icterus, petechiae; newborns infected at time of birth can develop serious illness
- Rubella-specific IgM antibody; virus culture of nasopharynx
- No specific therapy

Varicella-Zoster

- 7 cases per 10,000 pregnancies
- Member of herpesvirus family; highly contagious; disease occurs throughout the year, with peak incidence in the winter months
- + Congenital, ++ Acquired; +++ Nosocomial
- Asymptomatic; congenital infection, neonatal chickenpox
- Documentation of varicella antigen from skin lesions; clinical diagnosis based on history of exposure and characteristic rash
- Mild illness with typical rash; may present as severe disseminated disease
- Acyclovir for severe disease; Varicella-zoster immune globulin (VZIG) for infants born to infected mothers at time of delivery; infants <1000 g or <28 weeks gestation
- Severe disease often fatal; mild disease no long-term sequelae

Compiled from Arvin & Maldonado, 1994; Cherry, 1994; Cooper, Preblud, & Alford, 1994; Filipell & Rearick, 1994; Gershon, 1994; Giacoia, 1994; Kinney & Eiden, 1994; Mueller & Pizzo, 1994; Overall, 1992; Smith, 1993; Stagno, 1994; Torok, 1994; Whitley & Arvin, 1994.

AZT: zidovudine; CNS: central nervous system; DDC: dideoxycytidine; DDI: dideoxyinosine; DIC: disseminated intravascular coagulation; NEC: necrotizing enterocolitis; NICU: neonatal intensive care unit.
Diagnosis of viral infection in the newborn usually is prompted by a strong suspicion based on physical characteristics of the newborn, history of exposure or maternal illness, or failure of sepsis testing to yield positive results.

quelae (Cooper, Preblud, & Alford, 1994; Overall, 1992). Family members should be provided with available educational materials. Brochures, such as those on CMV from the Children's Biomedical Research Institute in St. Paul, Minnesota, can be helpful for some families (Children's Hospital, 1989).

The first action of nursing care of the newborn with symptoms often is to raise the index of suspicion for viral disease. The presence of physical findings, such as characteristic rashes or vesicles, microcephaly, intrauterine growth retardation or small size for gestational age, should alert the nurse to the possibility of congenital infection. Obtaining a detailed, accurate maternal history, including immunizations received, signs/symptoms of possible viral illness during the pregnancy, and exposure to possible sources of viral infection, is important. Collecting correct specimens for diagnostic tests and cultures is as important as prompt processing by the clinical laboratory (Strodtbeck, 1986).

Nursing care of the newborn with severe viral disease is complex and varies according to the clinical presentation of the neonate. Presentation of specific management plans for each viral infection is beyond the scope of this article. General nursing care may involve ventilator therapy, pharmacologic support of cardiovascular status, correction of acidosis and shock states, correction of coagulopathies, and treatment of severe neurologic disorders, such as meningitis and seizures. Skin care and nutrition are other areas of concern to nursing because many infants may have rashes or be at risk for secondary infections. Care of the family is important and can be complicated by maternal or paternal feelings of guilt regarding congenital infection or infection acquired at the time of delivery. The need for special care such as isolation also can present problems and increase the family's stress. Meticulous attention to hand washing, aseptic technique, and the use of personal protective equipment are additional important aspects of nursing care. These last strategies are important for protecting the ill newborn from other infections and preventing nosocomial spread of the viral infection to other patients or staff members.

Nosocomial Viral Infections

Within recent years, there has been a developing awareness on the national level of the role of viruses as nosocomial pathogens (Giacoi, 1994; Guerina & Goldmann, 1993; Strodtbeck, 1986). Although viral infections are uncommon in the newborn nursery, the potential for outbreaks remains a constant concern, especially during peak seasons of viral infections within the community. Infected visitors and health care workers may unknowingly expose healthy newborns to respiratory viruses such as RSV, influenza, and adenovirus; enteric viruses such as rotavirus and enteroviruses; herpes simplex virus; and varicella virus (Overall, 1992). Early discharge results in newborns being discharged before the end of the minimum incubation period for most viruses. These infants often have symptoms develop at home. Routine telephone follow-up of discharged newborns may alert the nursery staff to the presence of a problem.

The NICU is especially susceptible to nosocomial viral infections for a variety of reasons, including extensive use of invasive technology for the care of sick infants, a fragile and immunocompromised patient population, and large numbers of health care workers and visitors who come in contact with the patients (Guerina & Goldmann, 1993; Strodtbeck, 1986). Newborns with underlying cardiac and pulmonary dysfunction are particularly at risk for the development of nosocomial viral infections, especially RSV and CMV (Guerina & Goldmann, 1993; Smith, 1993).

Many viruses have been implicated as etiologic agents of nosocomial infections in the NICU. The most commonly identified viruses are the respiratory viruses (rhinovirus, adenovirus, RSV, parainfluenza, and influenza virus); the enteric viruses (rotavirus and coronavirus); the enteroviruses; and the herpes viruses (CMV and herpes simplex). Nosocomial viral infections are of concern because they prolong hospital stays and increase health care costs, increase neonatal morbidity and mortality, and are difficult to recognize and diagnose. New evidence suggests that infection with CMV or influenza virus may complicate matters by predisposing the critically ill newborn to bacterial superinfection because of viral-induced changes in polymorphonuclear leukocyte function (Abramson & Wheeler, 1994).

Reports of nosocomial infection range from case studies to unit outbreaks (Singh-Naz, Brown, & Ganeshanathan, 1993; Strodtbeck, 1986; Watson et al., 1993). Risk factors linked to nosocomial viral infections include intubation and assisted ventilation; transfusion with CMV-positive donor blood; ingestion of contaminated breast milk; direct and indirect contact among patients, visitors, and hospital personnel; contact with contaminated objects in the environment; and inadequate hand washing (Guerina & Goldmann, 1993; Strodtbeck, 1986).
Conclusion

Each year significant neonatal morbidity and mortality occur as a result of viral infections. The presence of a developing immune system inexperienced in response to viruses in a rapidly growing host make the newborn especially vulnerable to viral disease. The effects of viral infection are varied and range from no symptoms to mild or severe disease. Although the newborn immune system is capable of mounting a defense to the virus, the response often is inadequate for preventing infection. The outcome of viral infection depends upon the specific virus, the gestational age of the newborn at the onset of infection, and the severity of the infection. In addition to limited specific antiviral drug therapy, many of the available drugs are not recommended for use in newborns. Several new therapies are being tested and show promise; eventually they may lessen the severity of disseminated disease and minimize the long-term sequelae. Until such time, the major thrusts of management of neonatal viral disease are prevention of the infection and supportive care for the acutely ill newborn.

References

Abramson, J. S., & Wheeler, J. G. (1994). Virus-induced neutrophil dysfunction: Role in the pathogenesis of bacterial infections. *Pediatric Infectious Disease Journal, 13*(7), 643–652.

American Academy of Pediatrics Committee on Infectious Diseases. (1994). *The 1994 red book*. Evanston, IL: American Academy of Pediatrics.

Arvin, A. A., & Maldonado, Y. A. (1994). Other viral infections of the fetus and newborn. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 745–755). Philadelphia: WB Saunders.

Cherry, J. D. (1994). Enteroviruses. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 404–445). Philadelphia: WB Saunders.

Children’s Hospital. (1989). *CMV: Diagnosis, prevention and treatment*, 2nd ed. St. Paul: Children’s Biomedical Research Institute.

Cooper, L. Z., Preblud, S. R., & Alford, C. A. (1994). Rubella. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 268–310). Philadelphia: WB Saunders.

Filippell, M. B., & Rearick, T. (1993). Respiratory syncytial virus. *Nursing Clinics of North America, 28*(3), 651–671.

Gershon, A. A. (1994). Chickenpox, measles and mumps. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 565–617). Philadelphia: WB Saunders.

Giacota, G. P. (1994). Uncommon pathogens in newborn infants. *Journal of Perinatology, 24*(2), 134–144.

Guerina, N. G., & Goldmann, D. A. (1993). Neonatal nosocomial infections: Prevention and management. In S. L. Kaplan (Ed.), *Current therapy in pediatric infectious disease*, 3rd ed. (pp. 406–416). New York: BC Decker.

Kinney, J. S., & Eiden, J. J. (1994). Enteric infectious disease in neonates: Epidemiology, pathogenesis, and a practical approach to evaluation and therapy. *Clinics in Perinatology, 21*(2), 317–333.

Mueller, B. U., & Pizzo, P. A. (1994). Acquired immunodeficiency syndrome in the infant. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 377–403). Philadelphia: WB Saunders.

Overall, J. C. (1992). Viral infections of the fetus and neonate. In R. D. Feigin & J. D. Cherry (Eds.), Textbook of pediatric infectious diseases, Volume 1, 3rd ed. (pp. 924–959). Philadelphia: WB Saunders.

Singh-Naz, N., Brown, M., & Ganeshanantham, M. (1993). Nosocomial adenovirus infection. Molecular epidemiology of an outbreak. *Pediatric Infectious Disease Journal, 12*(11), 922–925.

Smith, J. B. (1993). Congenital viral and protozoan infections. In J. J. Pomerance & C. J. Richardson (Eds.), *Neonatology for the clinician* (pp. 173–184). Norwalk, CT: Appleton & Lange.

Stagno, S. (1994). Cytomegalovirus. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 312–353). Philadelphia: WB Saunders.

Strodtbeck, F. (1986). The epidemiology of nosocomial viral infections in infants who are long term residents of the neonatal intensive care unit. Unpublished dissertation. Indiana University: Indianapolis, pp. 25–63.

Torok, T. J. (1994). Human parvovirus B19. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 668–701). Philadelphia: WB Saunders.

Voyles, B. A. (1993). *The biology of viruses* (pp. 27, 197–300). St. Louis: Mosby.

Watson, J. C., Fleming, F. W., Borella, A. J., Olcott, E. S., Conrad, R. E., & Baron, R. C. (1993). Vertical transmission of hepatitis A resulting in an outbreak in a neonatal intensive care unit. *The Journal of Infectious Diseases, 167*(3), 567–571.

Whitley, R. J., & Arvin, A. M. (1994). Herpes simplex virus infection. In J. S. Remington & J. O. Klein (Eds.), *Infectious diseases of the fetus and newborn infant*, 4th ed. (pp. 354–375). Philadelphia: WB Saunders.

Address for correspondence: Frances Strodtbeck, RNC, DNS, NNP, 1743 West Harrison Street, Room 301 SHH, Chicago, IL 60612.

Frances Strodtbeck is the coordinator of the Neonatal Nurse Practitioner Program at the College of Nursing of Rush University of Chicago.