HEPATITIS B (including delta)  
(acute, chronic, delta), not perinatal

REPORTING INFORMATION

- **Class B2 (hepatitis B [including delta], not perinatal):** Report by the end of the work week after the case or suspected case and/or a positive laboratory result to the local public health department where the patient resides. If patient residence is unknown, report to the local public health department in which the reporting health care provider or laboratory is located.

- **Reporting Form(s) and/or Mechanism:** Viral Hepatitis Case Report form, Ohio Confidential Reportable Disease form (HEA 3334, rev. 1/09), Positive Laboratory Findings for Reportable Disease form (HEA 3333, rev. 8/05), the local health department via the Ohio Disease Reporting System (ODRS), or the telephone.
  - Special Notes on Reporting: All new pregnancies for women identified with acute or chronic hepatitis B infection should be reported, even if the case of acute or chronic hepatitis B infection was reported prior to the pregnancy or during a previous pregnancy.

- **Additional reporting information, with specifics regarding the key fields for ODRS Reporting can be located in Section 7.**

AGENTs

Hepatitis B virus is classified in the Hepadnaviridae family, and is a member of the Orthohepadnavirus genus. The hepatitis B virus is a partially double-stranded DNA virus, 40-48 nm in diameter.

Hepatitis D (delta) virus is a defective single-stranded RNA virus that requires the helper function of the hepatitis B virus to replicate.

TEST NAME ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgM anti-HAV</td>
<td>Immunoglobulin M antibody to hepatitis A virus</td>
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<tr>
<td>Anti-HBe</td>
<td>Antibody to hepatitis B e antigen</td>
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<tr>
<td>Anti-HBs</td>
<td>Antibody to hepatitis B surface antigen</td>
</tr>
<tr>
<td>HBeAg</td>
<td>Hepatitis B e antigen</td>
</tr>
<tr>
<td>HBsAg</td>
<td>Hepatitis B surface antigen</td>
</tr>
<tr>
<td>HBV DNA</td>
<td>Hepatitis B virus DNA</td>
</tr>
<tr>
<td>IgM anti-HBc</td>
<td>Immunoglobulin M antibody to hepatitis B core antigen</td>
</tr>
<tr>
<td>Total anti-HBc (IgM/IgG)</td>
<td>Immunoglobulin M and Immunoglobulin G antibodies to hepatitis B core antigen</td>
</tr>
<tr>
<td>Anti-HDV</td>
<td>Antibody to hepatitis D virus</td>
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</table>

CASE DEFINITION

**Hepatitis B, Acute**

**Clinical Case Definition**

An acute illness with discrete onset of symptoms and a) jaundice or b) elevated serum aminotransferase levels (ALT) >100 IU/L.

**Comment**

A documented negative hepatitis B surface antigen (HBsAg) laboratory test result within 6 months prior to a positive test result (either HBsAg, hepatitis B “e” antigen
[HBeAg], or hepatitis B virus nucleic acid testing [HBV NAT] including genotype) does not require an acute clinical presentation to meet the surveillance case definition.

**Laboratory Criteria for Diagnosis**
- HBsAg positive **AND**
- IgM anti-HBc positive (if done)
- IgM anti-HAV negative (if done)

**Case Classification**
- **Suspect**: A case that is IgM anti-HBc positive, or reported by a health-care professional as acute hepatitis B without laboratory results. (ODH)
- **Probable**: A case that does not meet the clinical case definition, but is laboratory confirmed and is a clinically compatible case, as reported by a health-care professional. (ODH)
- **Confirmed**: A case that meets the clinical case definition, is laboratory confirmed, and is not known to have chronic hepatitis B. (CDC)

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### Hepatitis B Case Classifications (Acute)
**Revised April 2012**

<table>
<thead>
<tr>
<th>Reportable Condition</th>
<th>Case Status</th>
<th>Onset Date*</th>
<th>Health Care Prof.</th>
<th>Jaundice</th>
<th>ALT</th>
<th>HBsAg</th>
<th>IgM anti-HBc</th>
<th>Lab Data is Weakly Positive or Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptomatic</strong></td>
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<tr>
<td><strong>Acute</strong></td>
<td>Confirmed</td>
<td>X</td>
<td>X</td>
<td>(+)</td>
<td>(+)*</td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Confirmed</td>
<td>X</td>
<td>&gt; 100 IU/L</td>
<td>(+)</td>
<td>(+)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Probable</td>
<td>X</td>
<td></td>
<td>(+)</td>
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<td></td>
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<tr>
<td></td>
<td>Suspect</td>
<td>X</td>
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<td>X</td>
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</tbody>
</table>

**Case is not known to be symptomatic**

* A documented negative hepatitis B surface antigen (HBsAg) laboratory test result within 6 months prior to a positive test (either HBsAg, hepatitis B “e” antigen (HBeAg), or hepatitis B virus nucleic acid testing (HBV NAT) including genotype) result does not require an acute clinical presentation to meet the surveillance case definition. Source: 2012 Case Definition. CSTE Position Statement Number: 11-ID-03.

**Terms and symbols used in this table:**

- Health Care Prof. = Case is clinically compatible with acute hepatitis as reported by a health care professional
- Lab data is weakly positive or missing = Hepatitis B infection is reported without lab results; or results for any hepatitis B test, (except anti-HBs), may be weakly or borderline positive; or HBV DNA result is below reference range
Symptomatic = Symptoms are compatible with acute hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, and abdominal pain)

(+)= A positive test result
(+)*= A positive test result (if done)

Hepatitis B, Chronic
Clinical Description
No symptoms are required. Persons with chronic hepatitis B virus infection may have no evidence of liver disease or may have a spectrum of disease ranging from chronic hepatitis to cirrhosis or liver cancer.

Laboratory Criteria for Diagnosis
- IgM anti-HBc negative AND a positive result on one of the following tests: hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg), or hepatitis B virus DNA (HBV DNA),
  OR,
- HBsAg, HBeAg, or HBV DNA positive two times at least 6 months apart (any combination of these tests performed 6 months apart is acceptable).

Comment
Cases should be reported in the following manner:

- If a positive test result has a specimen collection date greater than 6 months after an acute specimen collection date, **AND** is in the same calendar year, change the reportable condition from acute Hepatitis B to chronic Hepatitis B.
  - Example: If the first positive sample was collected on February 1, 2012 and the second positive sample was collected on September 12, 2012, change the reportable condition from acute Hepatitis B to chronic Hepatitis B.

- If a positive test result has a specimen collection date greater than 6 months after an acute specimen collection date, **AND** is in a different calendar year, create a new entry with a reportable condition of chronic Hepatitis B.
  - Example: If the first positive sample was collected on October 1, 2011 and the second positive sample was collected on May 12, 2012, create a new entry with a reportable condition of chronic Hepatitis B.

- If a positive test result has a specimen collection date less than 6 months after an acute specimen collection date, update the existing acute Hepatitis B case with additional laboratory results.

Case Classification
**Suspect:** A case that is reported by a health-care professional as chronic hepatitis B without laboratory results. (ODH)
Probable: A case with a single HBsAg positive or HBV DNA positive or HBeAg positive lab result and does not meet the case definition for acute hepatitis B. (CDC)

Confirmed: A case that meets either of the laboratory criteria for diagnosis. (CDC)

Comment:
Multiple laboratory tests indicative of chronic HBV infection may be performed simultaneously on the same patient specimen as part of a “hepatitis panel”. Testing performed in this manner may lead to seemingly discordant results, e.g., HBsAg-negative AND HBV DNA-positive. For the purposes of this case definition, any positive result among the three laboratory tests mentioned above is acceptable, regardless of other testing results. Negative HBeAg results and HBV DNA levels below positive cutoff level do not confirm the absence of HBV infection.

Hepatitis B Case Classifications (Chronic)
Revised April 2012

<table>
<thead>
<tr>
<th>Case Status</th>
<th>Health Care Prof.</th>
<th>IgM anti-HBc</th>
<th>HBsAg</th>
<th>HBeAg</th>
<th>HBV DNA (including qualitative, quantitative, and genotype testing)</th>
<th>Anti-HBe</th>
<th>Total Anti-HBc</th>
<th>Lab Data is Weakly Positive, Undetectable or Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirmed</td>
<td>(-)</td>
<td>+ or + or +</td>
<td></td>
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<tr>
<td>Confirmed</td>
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<td>6M ↔ 6M</td>
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<td></td>
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<tr>
<td>Probable</td>
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<td>+ or + or +</td>
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<tr>
<td>Suspected</td>
<td>X</td>
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<td></td>
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<tr>
<td>Suspected</td>
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<td>X</td>
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<tr>
<td>Suspected</td>
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<td></td>
<td></td>
<td>X</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Comment: If multiple laboratory tests indicative of chronic HBV infection are performed simultaneously, results may be seemingly discordant (e.g., HBsAg-negative and HBV DNA-positive). For the purposes of the case definition, any positive result among the three laboratory tests above is acceptable, regardless of other testing results.

Terms and symbols used in this table:
Health Care Prof. = Case is reported by a health care professional as chronic hepatitis

Lab data is weakly positive or missing = Hepatitis B infection is reported without lab results or lab may be weakly or borderline positive for any hepatitis B test, (except anti-HBs), or HBV DNA result is below reference range

(-) = A negative test result

(+) = A positive test result

6M ↔ 6M = Two positive results with collection dates at least six months apart for any combination of tests: HBsAg, HBV DNA or HBeAg
Hepatitis B, Perinatal Virus Infection Acquired in the United States or U.S. Territories: See Hepatitis B (Perinatal) chapter.

Hepatitis D (delta hepatitis)

Laboratory Criteria for Diagnosis
- HBsAg positive or IgM anti-HBc positive
- Anti-HDV positive

Case Classification
- Suspect: A case that is Anti-HDV positive and reported without hepatitis B virus laboratory results. (ODH)

Probable or Confirmed: Case classification for a hepatitis D virus case that is laboratory confirmed will follow the case classification designated for the associated hepatitis B virus case. (ODH)

Comment:
Anti-HBs positive test result indicates immunity due to vaccination or resolution of a previous infection. Test result may be added to the record of a previously reported case to indicate that the case has resolved.

HEPATITIS B
SIGN AND SYMPTOMS

The onset of acute hepatitis B is generally insidious. Clinical signs and symptoms include various combinations of fever, anorexia, malaise, nausea, vomiting, abdominal pain, dark urine, clay-colored stools, and jaundice. Skin rashes, arthralgia, and arthritis can also occur. Symptoms of acute hepatitis B vary by age. Most children less than 5 years of age and newly infected, immunosuppressed adults are asymptomatic whereas 30-50 percent of persons 5 years of age and older have some initial signs and symptoms. Symptoms begin an average of 90 days (range: 60–150 days) after exposure to HBV. Disease is more severe among persons older than 60 years of age. The fatality rate among acute cases reported to CDC is 0.5%–1%.

Persons with chronic HBV infection might be asymptomatic, have no evidence of liver disease, or have a spectrum of disease ranging from chronic hepatitis to cirrhosis or hepatocellular carcinoma (a type of liver cancer). The majority of chronic hepatitis B virus carriers are asymptomatic, with only 25 percent of those chronically infected in childhood and 15 percent of those chronically infected after childhood developing chronic liver disease.

DIAGNOSIS

Hepatitis B is distinguished from other forms of hepatitis through laboratory testing.

Serologic Diagnosis
Hepatitis B virus antigens and antibodies typically appear and disappear in serum in a predictable sequence over a period of time. Different combinations of these markers are detected in a single serum sample, depending on when during the illness testing is done. The serologic profile contributes to the diagnosis of hepatitis B virus infection and
indicates the stage of illness, degree of infectivity, carrier state or state of immunity.

**Serologic Markers**

**HBsAg.** HBsAg (hepatitis B surface antigen) is the first serologic marker to appear and can be detected in an infected person’s blood an average of 4 weeks (range: 1–9 weeks) after exposure to the virus. Its presence is indicative of active infection.

When HBsAg is positive in patients with apparent acute hepatitis, acute hepatitis B is suggested; however, superimposed hepatitis caused by another agent may give similar symptoms in a patient chronically infected with the hepatitis B virus. In order to differentiate an acute from a chronic infection in a person who is HBsAg-positive, it is necessary to test for IgM anti-HBc. If the IgM anti-HBc is positive, acute hepatitis B infection is suspected.

HBsAg positivity persisting beyond 6 months is indicative of chronic hepatitis B. The risk for chronic infection varies according to the age at infection and is greatest among young children. Approximately 90% of infants and 25%–50% of children aged 1–5 years will remain chronically infected with HBV. By contrast, approximately 95% of adults recover completely from HBV infection and do not become chronically infected.

Chronic hepatitis B virus carriers and are likely to remain HBsAg positive indefinitely. All HBsAg positive persons are potentially infectious, regardless of the presence or absence of any other serologic markers and the duration of infection.

Undetectable levels of HBsAg are present in many patients with subclinical hepatitis.

Note regarding bilirubin levels: Jaundice appears four weeks (range of 1-7 weeks) after the appearance of HBsAg. The severity of the hepatitis, as measured by bilirubin levels, correlates with the duration of HBsAg positivity. As bilirubin clears, HBsAg titers fall, and generally become undetectable within several weeks.

**IgM Anti-HBc.** IgM anti-HBc is often detectable at the time of clinical onset, and declines to sub-detectable levels within 6 months. It is a diagnostic marker for acute hepatitis B virus infection, useful clinically for differentiating acute/recent infection from chronic carrier state or resolved hepatitis B virus infection. It is also useful in the ‘window’ period, when HBsAg has become negative, and the patient has not yet developed the antibody (anti-HBs). A negative test for IgM anti-HBc in association with a positive test for HBsAg, a positive test for total anti-HBc and a negative anti-HBs test usually indicates that an individual has chronic hepatitis B virus infection.

**Total anti-HBc.** Total (combination of IgM and IgG) anti-HBc is generally detectable in serum by the onset of clinical illness. Total anti-HBc persists for many years, both in persons who have cleared the hepatitis B virus and in those who become chronic carriers. In patients with chronic hepatitis B virus infection, both HBsAg and anti-HBc total usually remain detectable for life.

**Anti-HBs.** Anti-HBs titers rise slowly during convalescence, after the disappearance of HBsAg in patients who do not progress to chronic infection. It generally indicates recovery and immunity from infection or previous active vaccination. In approximately 50% of patients with self-limited hepatitis B virus infection, there is a time interval of up to several months between the disappearance of detectable HBsAg and the appearance
of anti-HBs. During this time, only the total anti-HBc is detectable; this period is referred to as the “core window” or “window phase.” Approximately 5% of patients with self-limited hepatitis B virus infection will have cleared the HBsAg by the time they are seen by a clinician. Therefore, the initial diagnostic tests performed on patients presenting with a recent history of symptoms of viral hepatitis should include an IgM anti-HBc, as well as an HBsAg. A positive IgM anti-HBc in the absence of HBsAg is indicative of a recent resolved hepatitis B virus infection. Low titers of total anti-HBc and IgM anti-HBc, and high titers of anti-HBs may be present; their presence in conjunction with liver function abnormalities, appearing in a time frame consistent with the hepatitis B virus incubation period, suggests that hepatitis B virus infection has occurred and is probably resolving. Some persons who are HBsAg-positive will develop detectable anti-HBs; however, these persons are still considered infectious due to the presence of HBsAg. Since seroconversion to anti-HBs indicates immunity, anti-HBs is generally not detected in chronic infections.

**HBeAg.** HBeAg (hepatitis B e antigen) appears a few days after HBsAg becomes detectable and typically disappears before HBsAg is gone, although it might persist for years in a chronic carrier of hepatitis B virus. HBeAg is variably present in patients with chronic hepatitis B virus infection with 25%-50% of patients having detectable HBeAg. The presence of HBeAg correlates with higher titers of circulating hepatitis B virus and increased infectivity. It is not generally necessary to test for HBeAg or its antibody (anti-HBe), unless it is of particular importance that the patient’s relative infectivity be determined. The HBeAg/anti-HBe status should not alter the general recommendations given to patients and their contacts, since all HBsAg-positive individuals are at risk of transmitting hepatitis B. Testing for HBeAg, if done at all, should be reserved for persons who have already been shown to be HBsAg-positive, as HBeAg is not found in the absence of HBsAg.

**Anti-HBe.** Anti-HBe appears at about the time that HBeAg disappears. It generally indicates a good prognosis, and that the risk of infectivity is reduced. Failure of an HBeAg-positive patient to seroconvert to anti-HBe is associated with disease activity and probable chronicity. Anti-HBe is present in 50%-75% of patients with chronic hepatitis B virus infection.

**HBV DNA.** HBV DNA appears soon after HBsAg. It rises to high concentrations during the late incubation period and falls with the onset of clinical disease. Most chronic carriers have high titers of infectious hepatitis B virus in the serum. Detectable hepatitis B virus DNA in the serum is associated with highly contagious state, and undetectable hepatitis B virus DNA indicates that the patient has no detectable infectious virus but does not mean that the patient has resolved the infection.

The table below summarizes interpretation of laboratory findings:

<table>
<thead>
<tr>
<th>Laboratory Findings</th>
<th>Results</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>HBsAg</td>
<td>Negative</td>
<td>Susceptible</td>
</tr>
<tr>
<td>anti-HBc</td>
<td></td>
<td></td>
</tr>
<tr>
<td>anti-HBs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HBsAg</td>
<td>Negative</td>
<td>Immune due to natural infection</td>
</tr>
<tr>
<td>anti-HBc</td>
<td>Positive</td>
<td></td>
</tr>
<tr>
<td>anti-HBs</td>
<td>Positive</td>
<td></td>
</tr>
<tr>
<td>HBsAg</td>
<td>anti-HBc</td>
<td>anti-HBs</td>
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* Four possible interpretations:
1. May be recovering from acute hepatitis B virus infection.
2. May be distantly immune and the test is not sensitive enough to detect very low level of anti-HBs in serum.
3. May be susceptible with a false positive anti-HBc.
4. May have undetectable level of HBsAg present in the serum and the person is actually chronically infected.

**Epidemiology**

**Source**
Hepatitis B virus is found in human blood and blood products, semen, vaginal secretions, and serous fluids. Saliva can be a vehicle of transmission through bites, but other types of exposure to saliva are not likely to transmit hepatitis B virus.

**Occurrence**
Hepatitis B virus infection is a major cause of acute and chronic hepatitis, cirrhosis, and primary hepatocellular carcinoma worldwide. The frequency of hepatitis B virus infection and patterns of transmission vary markedly in different parts of the world. In the United States, Western Europe and Australia, it is a disease of low endemicity, where only 0.2%-<2% of the population are carriers and infection occurs primarily during adulthood. In contrast, hepatitis B virus infection is highly endemic in China and Southeast Asia, sub-Saharan Africa, most Pacific islands and the Amazon Basin; in these areas, 8%-15% of the population carries the virus, and most persons acquire infection at birth or during childhood. In other parts of the world, hepatitis B virus is a disease of intermediate endemicity, where 2%-7% of persons are hepatitis B virus carriers.

It is estimated that the number of chronic carriers in the US is 800,000-1.4 million persons, of which 20%-30% acquired the infection during childhood. These carriers serve as the major reservoir of ongoing hepatitis B virus transmission. In 2008, the estimated number of new infections was 38,000, a decrease of 86% from a peak of approximately 280,000 infected each year during the mid 1980s; this is due primarily to the national strategy to eliminate hepatitis B infection in the US which was implemented in 1991 which recommended routine vaccination of children.

Approximately half of adults become ill or jaundiced with acute illness; less than 10
percent of children under 5 years of age are symptomatic; and approximately 40 persons (1 in 2,000) die of fulminant disease each year. Children less than 5 years of age are more likely to become chronic carriers, although the rate of infection among children has dramatically decreased since recommendations for routine immunoprophylaxis of infants and children have been implemented. Approximately 25% of those who become chronically infected during childhood and 15% of those who become chronically infected after childhood die prematurely from cirrhosis or liver cancer, and the majority remain asymptomatic until onset of cirrhosis or end-stage liver disease. In the United States, chronic HBV infection results in an estimated 2,000–4,000 deaths per year.

The estimated lifetime risk of hepatitis B virus infection in the United States is approximately 5% in the general population, but there are specific groups at greater risk (see “At Risk” Groups). The incidence of hepatitis B varies by race/ethnicity. In 2008, the highest rate of acute, symptomatic hepatitis B was among non-Hispanic blacks (2.2 cases per 100,000 population); rates are higher among Hispanics than non-Hispanics. Additionally, the highest rate of new infections occurs among persons 25-44 years of age (2.6 cases per 100,000 population) and the lowest rate occurs among children less than 15 years of age (0.02 cases per 100,000 population).

The Centers for Disease Control and Prevention (CDC) estimates that over 25,000 infants are born to HBsAg-positive mothers each year in the US and its territories. Post-exposure prophylaxis is highly effective in preventing transmission of hepatitis B virus from mother to infant. It is estimated that 1,250 of these “high risk” infants become chronically infected with hepatitis B virus each year because not all HBsAg-positive mothers are identified and not all infants receive appropriate prophylactic treatment.

Mode of Transmission
The virus is transmitted by percutaneous or mucosal exposure to HBsAg-positive blood and/or body fluids from persons who have acute or chronic HBV infection. Modes of transmission include the following:

1. Sexual contact;
2. Injection-drug use;
3. Other percutaneous exposure, including tattooing, ear piercing, and acupuncture, as well as needlesticks or other injuries from sharp instruments sustained by medical personnel;
4. Exposure through breaks in the skin without overt needle puncture, such as fresh cutaneous scratches, abrasions, burns, or other lesions;
5. Contamination of mucosal surfaces with infective serum or plasma through, for example, mouth pipetting, eye splashes, or other direct contact with mucous membranes of the eyes or mouth, such as hand-to-mouth or hand-to-eye contact when hands are contaminated with infective blood or serum;
6. Contamination of mucosal surfaces with infective secretions other than serum or plasma such as semen;
7. Transfer of infective material to skin lesions or mucous membranes via inanimate environmental surfaces perinatal transmission from mother to infant.

Infection can occur in settings of continuous close personal contact, such as in households or among residents in institutions for the mentally retarded, presumably via inapparent or unnoticed contact of infectious secretions with skin lesions or mucosal surfaces. Less commonly, hepatitis B virus is transmitted through objects such as razors
and toothbrushes, as the virus is stable on environmental surfaces for at least 7 days. Urine and stool are not infectious, and fecal-oral transmission does not occur. There has never been a documented incident of hepatitis B transmission by food. While minute amounts of virus can be found in saliva, it is not an effective vehicle for transmission of disease unless it also contains blood. Hepatitis B is not transmitted through casual contact with an infected person.

**Period of Communicability**
The role of the hepatitis B virus carrier is central in the epidemiology of hepatitis B virus transmission. A person who is HBsAg positive and IgM anti-HBc negative or whose HBsAg positivity persists for six months or more is considered a carrier. Although the degree of infectivity is best correlated with HBeAg-positivity, any person positive for HBsAg is potentially infectious. The likelihood of developing the carrier state varies inversely with the age at which infection occurs. During the perinatal period, hepatitis B virus transmitted from HBeAg-positive mothers results in hepatitis B virus carriage in up to 90% of infected infants, whereas 6%-10% of acutely infected adults become carriers. The highest concentration of hepatitis B virus in carriers and persons with acute infection is in the blood and serous fluids; less is present in other body fluids, such as saliva.

**“At Risk” Groups**
Serologic surveys demonstrate that, although hepatitis B virus infection is uncommon among adults in the general population, it is highly prevalent in certain groups. Those at risk, based on the prevalence of serologic markers of infection, include: visitors or immigrants from high hepatitis B virus endemic geographic areas; infants born to infected mothers; injection drug users who share needles or other drug paraphernalia; prison inmates; sex partners of infected persons; sexually active persons with multiple partners; men who have sex with men; hemodialysis patients; health care and public safety workers with exposure to blood or blood-contaminated body fluids; and household contacts of HBsAg-positive individuals.

**Incubation Period**
The incubation period ranges from 6 weeks to 6 months with an average of 2-3 months.

**HEPATITIS D (delta hepatitis)**

**DIAGNOSIS**

**Serologic Markers**
The serologic course of hepatitis D virus infection varies depending on whether the virus is acquired as a coinfection with hepatitis B virus or as a superinfection of a person with chronic hepatitis B virus infection. In most persons with hepatitis B virus-hepatitis D virus coinfection, both IgM anti-HDV and IgG anti-HDV are detectable during the course of infection. However, in about 15% of patients, the only evidence of hepatitis D virus infection may be the detection of either IgM anti-HDV alone during the early acute period of illness or IgG anti-HDV alone during convalescence. Total anti-HDV generally declines to undetectable levels after the infection resolves and there is no serologic marker that persists to indicate that the patient was ever infected with the hepatitis D virus.

Hepatitis delta antigen (HDag) can be detected in serum in only about 25% of patients with hepatitis B virus-hepatitis D virus coinfection. When HDag is detectable, it
generally disappears as HBsAg disappears and most patients do not develop chronic infection.

In patients with chronic hepatitis B virus infection who are superinfected with hepatitis D virus, several characteristic serologic features generally occur, including:

1. Concentration of HBsAg declines at the time HDAg appears in the serum;
2. HDAg and HDV RNA remain detectable in the serum because chronic hepatitis B virus infection generally occurs in most patients with hepatitis D virus superinfection, unlike the case with coinfection;
3. High concentration of both IgM and IgG anti-HDV are detectable, which persist indefinitely.

**EPIDEMIOLOGY**

**Source**
Hepatitis D virus is found in human blood and blood products, semen, vaginal secretions, and serous fluids.

**Occurrence**
In general, in countries with a low prevalence of chronic hepatitis B virus infection, such as the United States, hepatitis D virus prevalence is generally low among both asymptomatic hepatitis B virus carriers (<10%) and patients with chronic hepatitis B virus-related liver disease (<25%). Hepatitis D virus infection in these countries occurs most commonly among injecting drug users and persons with hemophilia.

Hepatitis D virus infection is acquired either as a coinfection with hepatitis B virus or a superinfection in persons with chronic hepatitis B virus infection. Persons with hepatitis B virus-hepatitis D virus coinfection may have more severe acute disease and a higher risk of hepatitis with rapid liver failure (2%-20%) compared to those infected with HBV alone; however, chronic hepatitis B virus infection appears to occur less frequently in persons with hepatitis B virus-hepatitis D virus coinfection. Chronic hepatitis B virus carriers who acquire hepatitis D virus superinfection usually develop chronic hepatitis D virus infection. In long-term studies of chronic hepatitis B virus carriers with hepatitis D virus superinfection, 70%-80% have developed evidence of chronic liver diseases with cirrhosis compared with 15%-30% of patients with chronic hepatitis B virus infection alone.

**Mode of Transmission**
The modes of hepatitis D virus transmission are similar to those for hepatitis B virus, with percutaneous exposures the most common. Sexual transmission of hepatitis D virus is less efficient than with hepatitis B virus. Perinatal hepatitis D virus transmission is rare.

**HEPATITIS B (including delta)**

**PUBLIC HEALTH MANAGEMENT**

**Case**

**Investigation**
Determine through the patient’s physician if the patient is/was acutely ill and meets the case definition. If the patient is pregnant, follow the detailed guidance in the Hepatitis B (Perinatal) chapter.
Treatment
No therapeutic measure has been proven to have beneficial effect after the onset of acute hepatitis B. The amount of bed rest needed should be determined by the patient’s sense of well-being. Prolonged bed rest should be avoided unless necessary. The choice of food should be dictated by palatability and tolerance of individual patients and an attempt to maintain the best possible nutritional state. No specific dietary alterations appear to affect the outcome of acute viral hepatitis, with the exception of hepatic failure, for which protein and/or salt restriction may be indicated. Corticosteroids are not indicated for treatment of acute hepatitis B. During the acute illness and until liver function studies have returned to normal, the patient should avoid ingestion of alcohol. Persons with acute or chronic liver disease due to viral hepatitis or other causes should avoid eating raw shellfish such as oysters.

Isolation
Because hepatitis B is transmitted only through percutaneous or permucosal inoculation of hepatitis B virus, isolation of infected persons is unnecessary and inappropriate. Hospitalized patients should be placed on Standard Precautions. Physicians, nurses, dentists and others who draw blood or perform surgical procedures should be informed of the patient’s status, but Standard Precautions should always be followed.

Hepatitis B has been transmitted in health care settings by HBsAg-positive health care workers (HCWs) but such cases are rare, and patient contacts of infected HCWs are generally not at risk. Currently available data provide no basis for restricting the practice of HCWs infected with hepatitis B virus who perform invasive procedures not identified as exposure-prone. Exposure-prone procedures should be identified by medical/surgical/dental organizations and by institutions at which such procedures are performed. HCWs infected with hepatitis B virus who are HBeAg-positive should not perform exposure-prone procedures unless they have been advised and counseled by an expert review panel concerning under what circumstances, if any, they may continue to do so (see http://www.odh.ohio.gov/ASSETS/602CC205613D4865A724B1F98AFC70C0/InfHCW.pdf).

There is no evidence that HBsAg-positive food handlers pose a health risk in an occupational setting. Hepatitis B has never been documented as being foodborne; nevertheless, it is reasonable to educate infected food handlers about the sources of hepatitis B virus and routes of transmission and the importance of good personal hygiene, frequent handwashing, and avoidance of hand injuries. Food handlers who are HBsAg-positive should not be restricted from work.

In the community setting, it is important to avoid placing unreasonable restrictions on persons who are HBsAg-positive. Instructions to the patient should include a thorough explanation of the modes of transmission of hepatitis B virus. Personal toiletry items (e.g. toothbrushes, razors) and tools (e.g. nail scissors, nail files) which may potentially cause cutting injuries should not be shared with susceptible individuals. The patient should avoid sexual contact with susceptible individuals and should not donate blood or blood products.

Contacts
Two types of products are available for prophylaxis against hepatitis B. Hepatitis B vaccine provides active immunization against hepatitis B virus infection, and its use is
recommended for both pre- and post-exposure prophylaxis. Immune Globulin (IG) products provide temporary, passive protection and are indicated only in certain post-exposure settings.

See the ODH Vaccine Protocol Manual for the Advisory Committee on Immunization Practices (ACIP) recommended schedule. Information concerning the following subjects is also included in the ODH Vaccine Protocol Manual:
1. HBIG (Hepatitis B Immune Globulin),
2. Hepatitis B vaccine preparation, usage, storage, side effects and adverse reactions,
3. Prevacuation serologic screening for susceptibility, and
4. Serologic confirmation of postvaccination immunity and revaccination of nonresponders.

The current hepatitis B prevention strategy encompasses the following:
1. Screening of all pregnant women for the presence of HBsAg,
2. Providing HBIG and hepatitis B vaccine to infants of HBsAg-positive mothers,
3. Providing routine hepatitis B immunization for all infants,
4. Providing catch-up immunization to all children and adolescents aged < 19 years, and
5. Intensified efforts to immunize high-risk adolescents and adults.

Pre-Exposure Vaccination
Hepatitis B vaccination is recommended for all unvaccinated adults at risk for hepatitis B infection and for all adults requesting protection from hepatitis B infection. See the ODH Vaccine Protocol Manual for the ACIP recommended schedule. Adults at risk for hepatitis B infection include the following:
1. Sex partners of HBsAg-positive persons,
2. Sexually active persons not in a long-term, mutually monogamous relationship,
3. Persons seeking evaluation or treatment for a sexually transmitted disease,
4. Men who have sex with men,
5. Current or recent injection drug users,
6. Household contacts of HBsAg-positive persons,
7. Residents and staff of facilities for developmentally disabled persons,
8. Healthcare and public safety workers with risk for exposure to blood or blood-contaminated body fluids,
9. Persons with end-stage renal disease,
10. International travelers to regions with high or intermediate levels of endemic hepatitis B infection, and
11. Persons with HIV infection.

Post-Exposure Prophylaxis
Recommendations for post-exposure prophylaxis after percutaneous or permucosal exposure to hepatitis B are given in the ODH Vaccine Protocol Manual. The following factors and considerations are important in an assessment for post-exposure prophylaxis:
1. Perinatal exposure
2. Maternal screening
3. Acute exposure to blood that contains (or might contain) HBsAg
a. Exposed person not previously vaccinated
   i. Source known, HBsAg-positive
   ii. Source known, HBsAg status unknown
   iii. Source unknown
b. Exposed person previously vaccinated against hepatitis B
   i. Source known, HBsAg-positive
   ii. Source known, HBsAg status unknown
   iii. Source unknown

4. Sexual contacts of persons with acute hepatitis B virus infection
5. Household contacts of persons with acute hepatitis B virus infection

Comment
Because hepatitis D virus is dependent on hepatitis B virus for replication, hepatitis B virus-hepatitis D virus coinfection can be prevented with either pre- or post-exposure prophylaxis for hepatitis B virus. However, no products exist to prevent hepatitis D virus superinfection of persons with chronic hepatitis B virus infection. Thus, prevention of hepatitis D virus superinfection depends primarily on education to reduce risk behaviors.

SPECIAL INFORMATION
Questions about perinatal hepatitis B should be directed to the ODH Perinatal Hepatitis B Prevention Program at 614-387-7477.

Questions about immunization for hepatitis B should be directed to the ODH Immunization Program at 614-466-4643 or 800-282-0546.

The Division of Viral Hepatitis at the Centers for Disease Control and Prevention (CDC) has a web site with comprehensive information about viral hepatitis: http://www.cdc.gov/hepatitis.
What is hepatitis B?
Hepatitis B is a serious disease caused by a virus that attacks the liver. The virus, which is called hepatitis B virus, can cause lifelong infection, cirrhosis (scarring) of the liver, liver cancer, liver failure and death.

Who is at risk?
Hepatitis B can affect anyone. Each year in the United States, approximately 38,000 people of all ages get hepatitis B and close to 3,000 die of sickness caused by HBV. If you have had other forms of hepatitis, you can still get hepatitis B.

Get vaccinated!
Hepatitis B is preventable.

How great is your risk for hepatitis B?
One out of 20 people in the United States will get hepatitis B some time during their lives. Your risk is higher if you:
- have sex with someone infected with hepatitis B virus
- have sex with more than one partner
- are a man and have sex with a man
- live in the same house with someone who has lifelong hepatitis B virus infection
- have a job that involves contact with human blood
- shoot drugs
- are a patient or work in a home for the developmentally disabled
- have hemophilia
- travel to areas where hepatitis B is common

Your risk is also higher if your parents were born in Southeast Asia, Africa, the Amazon Basin in South America, the Pacific Islands or the Middle East.

If you are at risk for hepatitis B virus infection, ask your health care provider about hepatitis B vaccine.

How do you get hepatitis B?
You get hepatitis B by direct contact with the blood or body fluids of an infected person; for example, you can become infected by having sex or sharing needles with an infected person. A baby can get hepatitis B from an infected mother during childbirth.

Hepatitis B is not spread through food or water or by casual contact.

Who is a carrier of hepatitis B virus?
Sometimes, people who are infected with hepatitis B virus never recover fully from the infection; they carry the virus and can infect others for the rest of their lives. In the United States, about one million people carry hepatitis B virus.

How do you know if you have hepatitis B?
You may have hepatitis B (and be spreading the disease) and not know it; sometimes a person with hepatitis B virus infection has no symptoms at all. Your doctor can do a test to determine if you are infected.
If you have symptoms:
- your eyes or skin may turn yellow
- you may lose your appetite
- you may have nausea, vomiting, fever, and/or stomach or joint pain
- you may feel extremely tired and not be able to work for weeks or months

Is there a cure for hepatitis B?
There is no cure for hepatitis B; this is why prevention is so important. Hepatitis B vaccine is the best protection against hepatitis B virus. Three doses are needed for complete protection. Most people who are infected as adults, however, will clear the virus from their bodies on their own after a period of being very sick; some, though, will go on to have lifelong infection.

If you are pregnant, should you worry about hepatitis B?
If you have hepatitis B virus in your blood, you can give hepatitis B to your baby. Babies who get hepatitis B virus at birth might have the virus for the rest of their lives, can spread the disease, and can get cirrhosis of the liver or liver cancer.

All pregnant women should be tested for hepatitis B virus early in their pregnancy. If the blood test is positive, the baby should receive vaccine along with another shot, hepatitis B immune globulin (called H-BIG), at birth. The vaccine series should be completed during the first 6 months of life.

Who should get vaccinated?
- All babies, at birth
- All children and adolescents aged < 19 years who have not been vaccinated
- Persons of any age whose behavior puts them at high risk for hepatitis B virus infection
- Persons whose jobs expose them to human blood
- Household contacts of HBsAg-positive persons
- Residents and staff of facilities for developmentally disabled persons
- Persons with end-stage renal disease, including predialysis, hemodialysis, peritoneal dialysis, and home dialysis patients
- International travelers to regions with high or intermediate levels (HBsAg prevalence of ≥ 2%) of endemic hepatitis B
- Persons with chronic liver disease
- Persons with HIV infection
- All other persons seeking protection from hepatitis B virus infection