# VIRAL DISEASES AND THEIR DANGER ON ANESTHESIOLOGISTS

An Essay

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By

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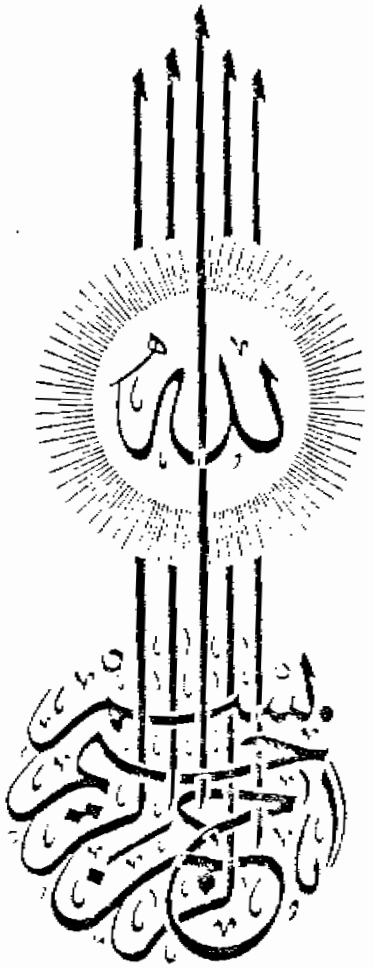


"وقل رب زدني علما"

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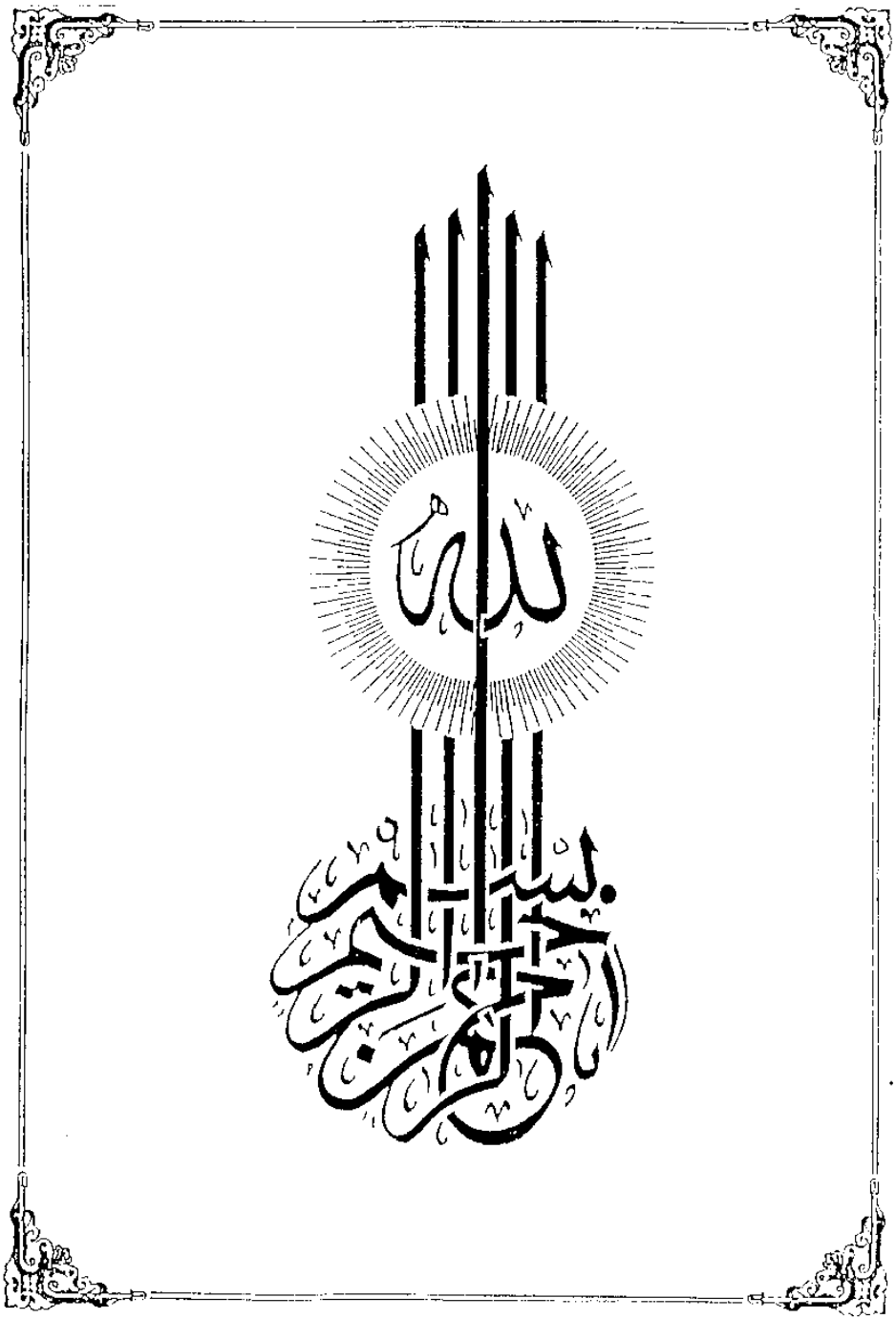

TO MY PARENTS ...

## ACKNOWLEDGEMENT

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# INTRODUCTION

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The extent of hospital acquired viral infection is difficult to define, but it is recognized as a major problem in the modern hospital environment. Although not often considered, the anesthesiologist is at risk for contracting these illnesses and may also serve as a source of transmission to patients as he works near the oral cavity and with invasive monitors.

The list of nosocomial viral agents is extensive and includes various hepatitis viruses, herpesvirus, adenovirus, influenza virus, rhinovirus, and the Creutzfeldt-Jakob disease. In addition, it appears that acquired immune deficiency syndrome [AIDS] is viral in origin.

The purpose of this essay is to consider important aspects of three major viral illnesses that may be encountered by the anesthesiologist or affect his patient. These three are AIDS, hepatitis Non-A, Non-B and hepatitis B.

# HEPATITIS B VIRUS

# 1. HEPATITIS B VIRUS

## I. PATHOGENESIS OF HEPATITIS B VIRUS

### Structure of Hepatitis B Virus (HBV)

Electron microscopy of serum from a patient with HBV infection reveals 3 types of particles (Fig.1). There is the relatively large (43 nm diameter) particle called the Dane particle, now known to represent the complete hepatitis B- virus. In addition there are smaller (22 nm diameter) tubular and spherical particles thought to represent surplus viral coat protein. The Dane particle consists of a central core and outer coat both of which are antigenic. They are known as the hepatitis B-core antigen ($HB_c$ Ag) and the hepatitis B- surface antigen (HBs Ag) respectively. The core also contains a deoxyribonucleic acid polymerase(DNA polymerase) and double stranded circular DNA. The $HB_e$ antigen is a component of the core antigen *(Sherlock, 1984)*

*Course of the Disease*

Acute viral hepatitis, whether due to hepatitis A virus (HAV), hepatitis B virus (HBV), non-A, non-B hepatitis virus (NANB) or hepatitis D virus (HDV), is usually characterized by anorexia, nausea, vomiting and distaste for smoking. These symptoms are followed several days later by jaundice and passage of dark urine and pale stools. Diarrhea may be the initial feature and low grade fever is common. About 15% of patients with hepatitis B may present with maculo-papular rash, petechial or even urticarial rash and about 25% of patients with hepatitis B have arthralgia of multiple joints. Discomfort under the right costal margin is common. In the

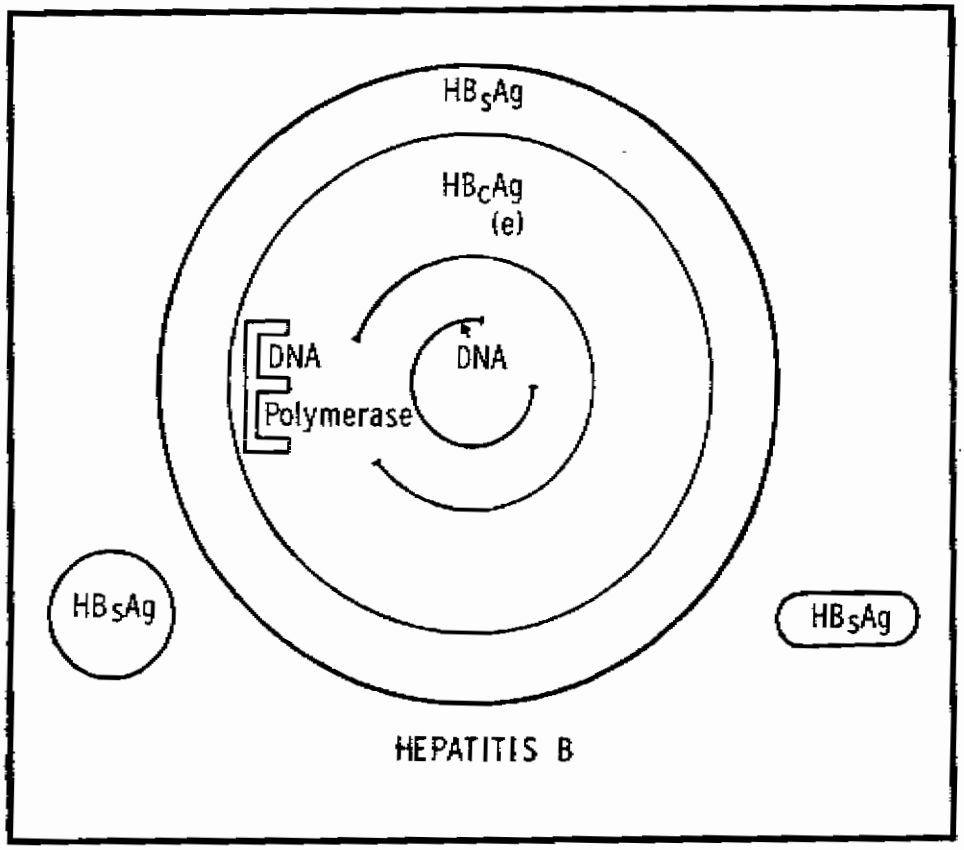


Fig 1. The virion of hepatitis B (Dane particle). The surface antigen (HBsAg) is formed in excess as tubules and spheres. The core contains a core antigen, double stranded DNA, a DNA polymerase and "e" antigen.

(After Sherlock, 1984).

pre-icteric phase the diagnosis may be unclear, and most patients with serologic evidence of previous infection have no clinical history. A history of contact should be sought.

Clinical examination: in addition to jaundice, the most common sign is a smooth enlarged tender liver. About 10% of patients have enlarged spleen *(Cooksley, 1986).*

**Immunologic Features and Diagnostic Tests For Hepatitis B-Virus**

The presence of immunologic markers for hepatitis B-virus constitutes the basis of diagnostic tests used to identify acute or chronic infection with the virus. $HB_s$ Ag is associated with 3 morphological forms: 20-nm spheres, tubules of the same diameter but of variable length, and the surface of the 42-nm virion (Dane particle). Within the Dane particle is a 27-nm core, hepatitis B-core antigen ($HB_cAg$) *(Mathieu and Dienstag, 1983).*

For the detection of these viral antigens and the corresponding antibodies, there is a wide variety of serological techniques of varying sensitivity. Among the more commonly used tests for $HB_sAg$, radioimmunoassay is the most sensitive. Despite its sensitivity, however, radioimmunoassay does not detect $HB_sAg$ in blood at a particle concentration less than $10^6$ per ml therefore fails to identify all $HB_sAg$-positive serum. Other tests for $HB_sAg$ that are occasionally used are, in order of decreasing sensitivity, immune adherence hemagglutination, complement fixation, counterelectrophoresis, and agar gel diffusion. Antibody to $HB_sAg$ can be detected by a number of techniques that include, in order of increasing sensitivity, agar gel diffusion, counterelectrophoresis,

complement fixation, immune adherence hemagglutination, passive hemagglutination, solid-phase radio-immunoassay and radioimmuno-precipitation. Of these, a commercially available radioimmunoassay and passive hemagglutination tests are most commonly used. Free $HB_cAg$ is not found circulating in the blood of either acutely or chronically infected individuals, but antibody to $HB_cAg$ (Anti- $HB_c$) can be detected *(Matthieu, and Dienstag, 1983).*

In addition to $HB_sAg$ and $HB_cAg$, a third antigen system has been identified with HBV infection. Hepatitis B associated e antigen ($HB_eAg$) represents a group of antigens that have no known morphologic counterpart and that are immunologically distinct from $HB_sAg$ and $HB_cAg$. Study of this system is hampered by the crudeness and insensitivity of tests for its identification (agar gel diffusion) and the paucity of potent reagent *(Matthieu and Dienstag, 1983).*

*Interpretation of Serological Assays*

Immunoassay for $HB_sAg$, anti-$HB_s$, and anti-$HB_c$ are now commercially available. The interpretation of results of these tests requires knowledge of the serological events that accompany acute and chronic type B-hepatitis. The various combinations of positive results of these tests and their interpretation are given: *(Zuckerman, 1983).*

*Pattern 1:* $HB_sAg$ alone occurs very rarely in one situation: late in the incubation period of type B-hepatitis, before anti $HB_c$ appears.

*Pattern 2:* $HB_sAg$ with anti $HB_c$ is the most common serologic pattern found in both acute and chronic type B hepatitis. It is not possible to differentiate whether the infection is acute or

chronic from the result of single serum sample without measuring titers of anti $HB_c$IgM.

*Pattern 3:* $HB_s$Ag with both anti-$HB_c$ and anti-$HB_s$: is a serologic pattern which has received increasing attention in recent years. Of interest is that the antigen and antibody do not appear to exist as an immune complex. Rather the anti-$HB_s$ is present in low titer and is heterotypic meaning that it is directed against a subtype of $HB_s$Ag other than with which it coexists. The co-occurrence of both $HB_s$Ag and anti $HB_s$ together in the serum is seen most commonly in chronic type B-hepatitis, especially when the disease is active or advanced.

*Pattern 4:* Anti $HB_s$ and anti $HB_c$ together is the usual serological pattern in the serum of persons who have recovered from acute type B-hepatitis. Such individuals should be considered fully recovered and immune.

*Pattern 5:* Anti $HB_c$ alone, without $HB_s$Ag or anti $HB_s$, can be interpreted in 3 ways, depending on the titer and subclass of antibody.

**First:** It is found in some individuals who have had type B- hepatitis long in the past and who have subsequently lost anti $HB_s$ reactivity. Such persons should have low titers of anti-$HB_c$ that is entirely IgG in subclass.

**Second:** It is serologic pattern found in many persons during the immediate recovery phase of acute type B- hepatitis between the disappearance of $HB_s$Ag and the appearance of anti-$HB_s$ (window period). In these persons the anti-$HB_c$ should be present in high titer and be partially IgM antibody.

**Third:** The finding of high titers of anti-$HB_c$ alone (of IgG type) may indicate a "low level" carrier state on individual who is infected with hepatitis B virus and who produces low undetectable levels of $HB_sAg$. The occurrence of such "low levels" or "silent carrier" may explain why some blood units that test negative for $HB_sAg$ nevertheless seem to transmit type B- hepatitis.

*Pattern 6:* Anti $HB_s$ alone, can occur long after hepatitis B virus infection or immunization with $HB_sAg$.

$HB_eAg$ and anti $HB_e$ testing: Testing for $HB_eAg$ and anti-$HB_e$ should be reserved for persons who have already been shown to be $HB_sAg$-positive. A sensitive immunoassay for $HB_eAg$ and anti $HB_e$ is recently commercially available. The finding of $HB_sAg$ with $HB_eAg$ can be interpreted as an active infection with high titers of circulating hepatitis B-virus. The seroconversion from $HB_eAg$ to anti $HB_e$ is generally a favourable sign for recovery.

**Transmission of Viral Hepatitis B and its Nosocmial Implications**

Most striking is the finding that in approximately 50% of cases without antecedent overt percutaneous inoculation, HBV can be implicated serologically. Because of the high $HB_sAg$ serum titers achieved in HBV infection, because of the development of sensitive serologic tests for detection of HBV infection and because of wide availability of these tests, there is now enormous amount of new information about the ecology of the virus *(Prince et al., 1970).*