antibodies would be developed against the antigenic site, complement would assemble on the antigen-antibody complex, and cell lysis would eventually occur. The organism protects itself from complement assembly and immune surveillance by binding the host’s factor H, thereby thwarting the immune system. The surface binding site for factor H is known as “outer surface protein E” (OspE). Through an elaborate gene-swapping process, the structure of the surface antigen OspE may change, based on external conditions and the degree to which the spirochete is threatened. Regardless of this surface variability, OspE is always able to bind factor H.

The known bacteria that have developed this system of immune protection include the causative agents for gonorrhea, meningitis, pneumonia, and the Lyme disease spirochete Borrelia burgdorferi. The virus responsible for human immunodeficiency disease (HIV) has also developed this protection from immune destruction and may account for some of the difficulty encountered in treating this disease. Other pathogenic bacteria and viruses may have also developed this capability but remain unknown (see Chart 2).

**Microbial Disease Spirochetes, Bacteria, Virus Protected from Immune Destruction by an Identical Mechanism**

Combating HIV by Antibodies to Factor H Binding Site

As indicated, the virus responsible for HIV protects itself from the immune system by binding human serum factor H to antigenic sites (gp41, gp120), thereby affording immune protection by disallowing assembly of complement components at the would-be antigen-antibody complex. Through the administration of antibodies to the factor H binding site, the protection afforded by factor H binding is removed and the virus is