Multiple sclerosis (MS) is a chronic demelinating disease of the central nervous system. Myelin is a fatty material that insulates nerves, acting much like the covering of an electric wire and allowing the nerve to transmit its impulses rapidly. It is the speed and efficiency with which these impulses are conducted that permits smooth, rapid and co-ordinated movements to be performed with little conscious effort. In MS, the loss of myelin (demyelination) is accompanied by a disruption in the ability of the nerves to conduct electrical impulses to and from the brain and this produces various symptoms. The sites where myelin is lost (plaques or lesions) appear as hardened (scar) areas: in MS these scars appear at different times and in different areas of the brain and spinal cord - the term multiple sclerosis meaning, literally, many scars.

In almost all lesions, a variable but usually substantial axonal loss occurs additionally to the loss of myelin. Axonal loss is an important factor in MS because, unlike demyelination, symptoms will not be affected by efforts to improve conduction (Herndon in Burks & Johnson, 2000).

The exact cause of MS has not been unravelled yet, but the disease process is presumed to reflect an auto-immune reaction against myelin antigens and is believed to depend on activated T-lymphocytes. Activated T-cells migrate from the periphery into the central nervous system, where they unite with an antigen-presenting cell and an antigen. This process is accompanied by a release of cytokines that upregulate and augment the inflammatory response. Myelin is damaged by activated microglia and macrophages. The loss of myelin insulation can result in passive ion leakage, slowed or blocked impulse conduction, and the subsequent functional impairments seen in the disease. Frequent sites of demyelination are the white matter adjacent to the lateral ventricles and the floor of the fourth ventricle, the corpus callosum and the periaquetuctal region, the optic nerves, chiasm, and tracts, the corticomedullary junction, and the white matter tracts of the spinal cord (Silwa, 2000).

There appear to be two very different patterns of demyelination (Herndon in Burks & Johnson, 2000).
The first and best-known consists of multifocal discrete inflammatory demyelinating lesions. These occur in both white matter and gray matter and have a predilection for periventricular white matter. They are typical of relapsing-remitting MS and are more common in younger patients.
The second pattern of demyelination is less dramatic but more common. It consists of diffusely scattered demyelination involving individual fibres or small groups of fibres interspersed with normal appearing myelinated fibres. This type

of demyelination is accompanied by much more limited, diffuse inflammatory infiltrates. It is characteristic of chronic progressive MS typically seen in older patients.
Secondary progressive MS involves both patterns of demyelination.

The triggering factor causing the immune system to attack myelin is unknown, but it is thought to be a combination of several factors. Viruses may be important in the development of multiple sclerosis, an infection perhaps occurring in a genetically or immunologically susceptible host. Elevated serum and cerebrospinal fluid antibody titres have been found to varicella zoster, measles, rubella and herpes simplex during relapse. A strong correlation has also been found between virus upper respiratory tract infection and MS exacerbations (Panitch, 1994).
Hereditary or genetic factors appear significant as an increase familial incidence of MS has been found. Studies of histocompatibility antigens have demonstrated an association between multiple sclerosis and A3, B7, B18 and DW2/DRW2. The effects of biochemical factors such as excess of dietary fats or malabsorption of unsaturated fatty acids (Lindsay et al., 1991), or mechanical trauma such as dental procedures, sprains, fractures, head injuries (Silwa, 2000), are unproven.