



## CHAPTER 2: CLINICAL ASPECTS OF HIV/AIDS

### 2.1 Introduction

Despite the dramatic advances made in understanding the natural history of HIV and the development of effective antiretroviral therapies, the AIDS epidemic continues to spread. This growth has displayed some disturbing trends. HIV/AIDS morbidity and mortality increasingly affect the poor, the disenfranchised, and the young.

*The Joint United Nations Programme Report on the HIV/AIDS Epidemic for 2006* (UNAIDS, 2006), estimated the number of people living with HIV/AIDS worldwide at about 39.5 million during 2006; of which 25 million live in sub-Saharan Africa. It further estimated the number of AIDS deaths in 2006 at a total of 2.9 million.

As noted previously, in South Africa, with a population of 47,7 million, the *National HIV and Syphilis Antenatal Sero-Prevalence survey in South Africa* (South Africa, 2005), conducted by the Department of Health, estimated that approximately 5.54 million South Africans were HIV-positive. Women are traditionally over-represented in these groups, as portrayed by the abovementioned survey. The researcher is of the opinion that unless we empower people, and more specifically women, this destructive disease will not be conquered. Of these 5.54 million HIV positive people, 500 000 are estimated to have contracted AIDS and are thus in need of ART's. The said researcher is further of the view that these statistics are an indication of why everything feasible should be done to treat as many people as possible and promote adherence to ART.

Although infection with HIV/AIDS is incurable at present, AIDS is considered to be a manageable chronic disease. Friedland (2003:35) argues that the treatment of patients with HIV infection requires not only a comprehensive



knowledge of the possible disease processes that may occur, but also, the ability to deal with the problems of a chronic, potentially life-threatening illness. Great advances have been made in the treatment of patients with HIV Infection. The appropriate use of a potent combination of highly active antiretroviral therapy (HAART), as well as prophylactic interventions, e.g. giving antiretroviral medications to prevent mother-to-child transmission, occupational injuries, and victims of sexual assault, is of critical importance in providing each patient with the best opportunity to live a long and healthy life despite the presence of HIV infection (Isselbacher, *et al.*, 1999:1853; Van Dyk, 1993:4-16).

In addition to the above, the patients must be educated regarding the importance of adherence and the consequences of non-adherence, for instance the potential development of resistance to antiretroviral drugs and its consequences. The present researcher concurs with Miller (2004:23) that for ART to be successful, education regarding adherence should be at the core of the treatment programme.

In chapter two, the clinical aspects of HIV/AIDS and antiretroviral therapy are discussed. Matters considered in this chapter are the clinical aspects of HIV/AIDS; the aetiology of AIDS; the morphology of HIV; modes of transmission; the human immune system and the effect of the HI virus on the immune system; CD 4 count; viral load; WHO stages and VCT. The said researcher is of the opinion that unless we understand the pathogenesis of the HI virus and antiretroviral therapy, we cannot attempt to treat it.

## **2.2 The Human Immunodeficiency Virus**

The Human Immunodeficiency Virus (HIV) is defined as the virus that causes AIDS. The acronym Acquired Immune Deficiency Syndrome (AIDS) emphasizes that the disease is acquired and not inherited (*Dorland's Illustrated Medical Dictionary*, 1994:826).

HIV is caused by a virus that invades the body, which subsequently attacks the body's immune system and makes it so weak and ineffective that it is unable to



protect the body from both serious and common infections and pathogens. Any HIV-infected individual with a CD4+T cell count of  $<200/\mu\text{L}$  has AIDS by definition, regardless of the presence of symptoms or opportunistic disease (*Dorland's Illustrated Medical Dictionary*, 1994:826; Kasper *et al.*, 2005:1852).

### 2.2.1 Aetiology of Aids

The first recognized cases of the Acquired Immune Deficiency (AIDS) syndrome occurred in America in the summer of 1981, when certain diseases suddenly appeared simultaneously in several patients. These patients all displayed a number of characteristics in common; they were young homosexual men with compromised immune systems. Soon afterwards, a new disease, which undermined the immune system and caused diarrhoea and weight loss, was identified in central Africa in heterosexual people. It was only in 1983 that it was discovered that the disease was caused by a virus which was then named HIV (human immunodeficiency virus).

There are many theories and myths about the origin of the human immune deficiency virus. The researcher supports the theory generally accepted by scientists, that HIV crossed the species barrier from primates to humans. It is not uncommon for a virus to cross from animals to humans as has been demonstrated with HTLV (Human T-cell Lymphotropic Virus) and the influenza viruses, and, as feared, might occur with the Avian Influenza virus.

The human immunodeficiency viruses, HIV type -1 and HIV type 2, which are cytopathic viruses, were identified during 1983. The history of the discovery of the HI virus is both interesting and controversial. Dr. Luc Montagnier and Dr. Francois Barre-Sinoussi and colleagues at the Louis Pasteur Institute in Paris, France, discovered HIV-1 in 1983. A year later Dr. Robert Gallo from the National Cancer Institute in the United States, claimed that he had been the first to discover the virus. Both Montagnier and Gallo are now officially recognized as co-discoverers of the virus (Isselbacher *et al.*, 1999:1853; Abdool Karim & Abdool Karim, 2005:32 and Van Dyk, 1999:4-16).



The HI virus belongs to the family of human retroviruses (retroviridae), and the subfamily of lentiviridae. Two viruses are mainly associated with AIDS: **HIV-1**, which is associated with infections in Central, East and Southern Africa, North and South America, and Europe. The most common cause of HIV diseases throughout the world is HIV-1. In 1999 it was demonstrated that HIV-1 infection in humans was zoonotic and had originated from the pan troglodytes species of chimpanzees (Isselbacher *et. al*, 1999:1853; Van Dyk, 1999:4-16 and Page, Louw, and Pakkiri, 2006:16-27).

**HIV-2** was discovered in West Africa (Cape Verde Islands, Guinea-Bissau and Senegal) in 1986, and is mostly restricted to West Africa. HIV-2 is more closely related to the simian immunodeficiency virus (SIV) found in sooty mangabeys, than it is to HIV-1 (Isselbacher, *et al*,1999:1853; Van Dyk, 1999:4-16; Van Dyk, 2004:5).

The current researcher holds the view that studies on the different types and mutations of HIV indicate that the virus has probably occurred in humans for at least a century, but has been isolated within a community, which had little contact with the outside world. As migration to the cities started to take place, HIV began to spread, which coincided with the decay of morals and values.

### **2.2.2 Diagnosing HIV by means of Voluntary Counselling and Testing**

In the case of individual testing, it is recommended that people make use of voluntary counselling and testing (VCT) services in order to ascertain their status. Internationally, much emphasis is placed on the process of counselling a person when he/she is undergoing an HIV test and the South African Department of Health, similarly, makes special provision for counselling and counsellors, in its regulations (South Africa:2003).

HIV counselling is defined as a confidential dialogue between a client (patient) and a care-provider, aimed at enabling the client to cope with the stress of a positive diagnosis and to assist the client in taking personal decisions related to



HIV/AIDS, for instance, when and to whom to disclose the diagnosis according to the Global Programme on AIDS (WHO, 2006).

An HIV test can be indicated for a variety of reasons: screening donated blood, epidemiological surveillance and mapping of HIV prevalence, and the diagnosis of HIV-infected individuals. The diagnosis of HIV infection is based mainly on the laboratory testing of blood samples. Two broad classes of tests can be distinguished in diagnosing HIV: an HIV antibody test and an HIV antigen test.

### **HIV Antibody Tests**

Antibodies are molecules produced by animals in response to antigens which possess the particular property of combining specifically with the antigen that induced the antibody's formation. HIV antibody tests are therefore based on the principle that the immune system manufactures antibodies against the viral infection and that these antibodies can be quantified.

The major factor limiting the antibody tests is the so-called 'window period': that period between the onset of HIV infection and the appearance of detectable antibodies to the virus. The period is about 3 to 12 weeks and any antibody test performed in this period may give false negative results. This means that although the virus is present in the person's blood, there are, as yet, no (detectable) antibodies in the blood. In such cases, the tests, therefore, erroneously indicate that the person is not infected. This false negative test is particularly dangerous since the person is already infectious and may unknowingly infect others (Foundation for Professional Development, 2006).

The best-known and most-often used antibody test is the ELISA or Enzyme-Linked ImmunoSorbent Assay.

### **Rapid Tests**

Rapid tests - antibody tests that can be performed outside a laboratory in rural and isolated places - have become very useful in the rapid diagnosis of HIV. The advantages of this test are that the results are available immediately, it is easy to use, and is relatively inexpensive and reliable. Blood is obtained from

the patient by a finger-prick, and a drop of blood is placed on the inset with a drop of solution. After 3 to 5 minutes the results are visible. One line indicates a negative result. Two or more lines indicate that the patient is HIV-positive. A positive result must be confirmed with a second test.

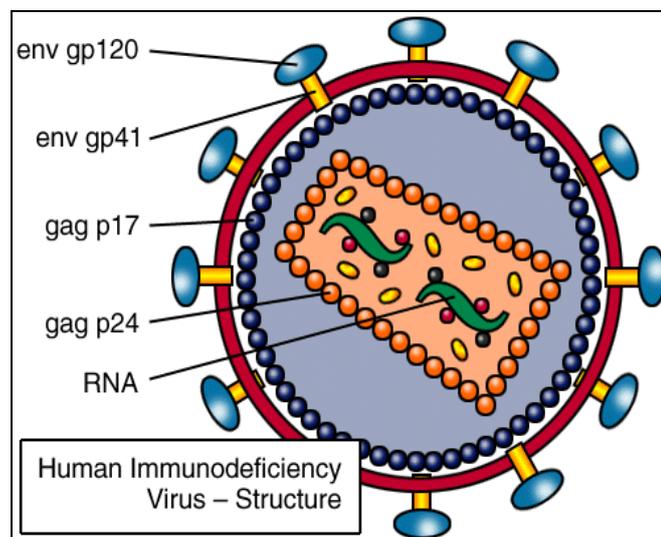
### HIV p24 Antigen Test

In order to minimize the problem of the window period, blood tests that detect the actual virus (HIV) in the blood have been developed to diagnose HIV-infection. The HIV p24 antigen test detects the proteins of the virus and is directed specifically at the predominant HIV antigen (p24). This antigen is usually detectable in the blood approximately 16 days after initial HIV-infection and thus lends itself to an earlier diagnosis of HIV. Viral antigens are detected by the HIV PCR - Polymerase Chain Reaction – technique. Since this test does not rely on the development of antibodies, it is useful in the early diagnosis of HIV. For instance, this test is used in the case of HIV-exposed babies where a definitive diagnosis is required at the baby’s “6 weeks” clinic visit. This test is however very expensive and is reserved for specific cases and settings.

### 2.2.3 Morphology of HIV

The researcher will refer to the following illustration to explain the morphology (study of the form or shape of an organism) of the virus.

**Figure 1: Human Immunodeficiency Virus**



(University of Pretoria, 2006)



The above illustration can be explained as follows:

The HI virus possesses a circular shape and consists of an inner matrix of protein called the core, in which the genetic material (viral RNA) is housed.

The core is surrounded by an outer layer of protein called a nucleocapsid. Electron microscopy shows that the HIV virion is an icosahedral structure containing numerous small glycoprotein projections (external spikes) on its surface, formed by the two major envelope proteins: the external gp 120 and the transmembrane gp41.

The three enzymes that are crucial to viral replication are reverse transcriptase, integrase and protease. Two of these enzymes are currently targeted by antiretroviral drugs (Isselbacher *et. al*, 1999:1853; Van Dyk, 1999:4-16; Foundation for Professional Development, 2006 and Spencer, 2005:6, 7).

#### **2.2.4 Pathophysiology of HIV**

The researcher is of the opinion that the physiology of the HI virus is a very complex issue that can cause great difficulties in communication between social workers, counsellors, patients, and the public in general. The physiology and biochemical functioning of the virus must be understood if antiretroviral and adherence matters are to be addressed.

As illustrated in 2.2.3 above, glycoprotein projections (spikes) are situated on the outer layer of the virus that attach themselves to CD4 receptors, which are present on various types of cells such as: monocytes (a type of white blood cell or leukocyte: part of the human body's immune system that protects against blood-borne pathogens and moves quickly); and macrophages (cells within the tissues that originate from monocytes) and tissue cells in mucous membranes (e.g. the genital and rectal tracts, and certain brain cells). The main role of macrophages is to stimulate an immune response to an infectious agent. When HIV therefore invades the body, and the macrophages attempt to do their usual



job by capturing the invader, the real problem begins (Isselbacher *et. al*, 1999:1853; Spencer, 2005:10 & Van Dyk, 2004:66).

The HI virus incorporates itself in the DNA of the nucleus (a small area inside the cell where genetic material is kept) and becomes integrated in this sanctuary part of the cell, thereby effectively hiding from the body's natural immune system.

The virus, furthermore, uses the body's DNA to replicate itself. This is achieved by employing normal physiological processes, whereby cells copy DNA (the "blueprint" for building living cells) into RNA (the construction foreman), which tells enzymes (the workers) to build new proteins. Proteins are the building blocks used to create living things. This process eventually destroys the body's CD4 cells and it is this unique response that makes HIV so dangerous (and ultimately fatal) to human beings (Van Dyk, 2004:15: Foundation for Professional Development, 2006:34).

The specific events in the replication cycle of the HIV are explained by Spencer (2005:6, 7):

- Free virus (HIV);
- Fusion with CD4 receptor/co-receptor and cell membrane;
- Penetration and entry of HIV into cytoplasm of cell;
- 'Uncoating' of virus and liberation of 'free' virus and its associated viral enzymes;
- Transformation of viral RNA into viral DNA;
- Reverse Transcription;
- Penetration of the nucleus of the cell and integration of viral DNA into host (genomic) DNA to form proviral DNA;
- Activation of the CD4 cell leads to the transcription of proviral DNA into its original (genomic) viral RNA and messenger, mRNA;



- Viral RNA leaves the nucleus together with viral mRNA. mRNA is translated into appropriate viral proteins (structural, enzymic) on the ribosomes of the endoplasmic reticulum;
- Translated viral proteins and genomic viral RNA are processed, assembled, packaged and released in the form of a new infectious virus; and
- New viral assembly

In summary, HIV hijacks the most important defensive cell in the immune system (the CD4 cell) and turns it into an efficient virus factory to manufacture replicas of it. Although several antibodies are formed during this process, they are completely powerless against HIV, because HIV hides inside the CD4 cells. The body is then left defenceless because the antibodies will certainly not attack and kill their own CD4 cells.

### **2.2.5 Viral Load**

The HIV RNA viral load quantification or HIV RNA assay is a measurement of the number of viral particles in the blood. The viral load is expressed as “copies per millilitre” of blood. The test measures the viral RNA, since this is the form in which HIV particles carry their genetic material.

A rising viral load is indicative of very active HIV disease. The higher the viral load, the more rapidly a person’s immune system will be damaged by CD4 cell destruction. It is important to know the estimated number of HI viruses in the blood in order to manage HIV infection effectively, since this indicates the degree of viral activity and the subsequent pace of development of immune deficiency. A high viral load is furthermore associated with an increased risk of developing mutations that might afford resistance to anti-viral medication (University of Pretoria, 2006 & Bartlett, Gallant, & Joel, 2005:40).

The ultimate purpose of antiretroviral therapy is to achieve an undetectable viral load (less than 50 viral copies per millilitre of blood) and sustain it as such. Measurement of the viral load is thus very useful in monitoring the response to antiretroviral therapy. This is, however, a very expensive test and is unavailable



in many developing countries, and, therefore, is not an absolute requirement for the management of patients on anti-retroviral treatment.

The viral load changes logarithmically. Only a change of more than 0.5 log<sub>10</sub> is regarded as clinically significant and an ideal response would be a decrease in the viral load of 1 log after 8 weeks of antiretroviral treatment. The following example illustrates the point: a change in viral load from 100 000 to 10 000 copies/ml represents a 1 log<sub>10</sub> change and is regarded as clinically significant, whereas a change in viral load from 100 000 to 30 000 copies/ml represents a 0.48 log<sub>10</sub> change and is not clinically significant. The final aim of treatment is to reduce the viral load to fewer than 50 copies per millilitre after 24 weeks of ARVs and to maintain that level (Isselbacher *et. al*, 1999:1853; Spencer, 2005:10 & Van Dyk, 2004:66).

## **2.3 Modes of Transmission**

For infection with HIV to occur, the virus must find a way to enter the bloodstream. This is more likely to happen if there are sufficient quantities of the virus in the fluid (i.e. semen, vaginal fluid, and blood or breast milk), and one is exposed to the virus for a long time.

### **2.3.1 Sexual**

HIV infection is primarily sexually transmitted when the virus enters a person's bloodstream via the body fluids of an infected individual. HIV is spread most commonly by unprotected vaginal or anal penetrative, sexual contact with an infected partner and possibly by oral sexual contact under certain conditions, e.g. in the presence of ulcerations of the buccal mucosa.

The membrane linings of body cavities, especially in the ano-rectal area and the vagina, are very delicate and can be torn as a result of friction generated during sexual intercourse. The virus can enter the body through the lining of the vagina, vulva, penis and rectum during sex. This makes it easy for the virus to enter the sex partner's bloodstream – either through the tears or by mixing with blood from larger injuries. There are other body fluids, including saliva, sweat,



tears and urine that do not contain sufficient quantities of the virus to be transmitted.

### **2.3.2 Blood**

HIV can also be spread by means of contact with infected blood. For instance, drug- users frequently spread HIV by sharing needles or syringes contaminated by the blood of someone infected with the virus.

Healthcare workers can also become infected with HIV when injured with needles, syringes, razor blades or other sharp instruments contaminated with HIV-infected blood. Similarly, a person can be infected when s/he receives HIV-contaminated blood via a blood transfusion (Evian, 2000:20 & Van Dyk, 2004:19).

### **2.3.3 Mother-to-child Transmission (MTCT)**

Mother-to-child transmission (MTCT) of HIV is the major causes of HIV infection in children. HIV can be transmitted from an infected mother to her baby either via the placenta during pregnancy, by blood contamination during childbirth or by breastfeeding (Evian, 2000:32 & van Dyk, 2004:19).

## **2.4 Preventing the Spread of HIV**

HIV/AIDS is a life-threatening disease. In the absence of a cure, or vaccine to prevent transmission of the virus, the only strategy would be prevention.

Prevention strategies include:

- Behaviour intervention by means of public education campaigns and outreach, testing and counselling;
- prevention technology by using condoms, or sterile needles;
- post-exposure prevention with antiretrovirals and
- mother-to-child transmission (PMTCT) by administering antiretrovirals.



#### **2.4.1 Behaviour Intervention by means of Education**

Behaviour interventions, where the focus specifically falls on preventing and spreading the HI-virus, have the biggest chance of limiting the epidemic. Public health education campaigns to raise awareness of high-risk activities have proven to be effective if successfully implemented as part of an integrated education prevention programme.

Abdool Karim & Abdool Karim (2005:268) corroborate this perspective on HIV/AIDS and education as follows: “Widespread education and associated high levels of knowledge have done little so far to contribute to a decline in HIV prevalence. There is increasing recognition that public health approaches that promote abstinence, fidelity and condom use, in the absence of wider societal changes, are not effective”. There is a distinct gap between cognitive knowledge of safe sex and risky sexual behaviour and behavioural outcome (actual refraining from risky behaviour) (Kasiram, Dano & Partab, 2006:54-55). The researcher is of the opinion that little correlation exists between knowledge and refraining from unprotected sexual encounters, since knowledge is cognitive and sexual behaviour is usually an emotional act and a basic human need.

- Ross and Deverell (2004:16) opine that cultural considerations definitely influence whether members of a population choose to participate in prevention campaigns and whether they choose to believe, internalise and accept the messages propagated by such campaigns, such as adhering to antiretroviral treatment.

#### **2.4.2 Voluntary Counselling and Testing (VCT)**

Voluntary counselling and testing (VCT) is a process whereby people in a community, industry or business receive counselling which enables them to understand the advantages of knowing their HIV status and more about the process of HIV antibody testing. Using this knowledge they can make an informed decision about being tested for HIV.



The results of an HIV test can have enormous psychological and social implications for the person being tested. As daunting as the barriers to HIV testing can be, it is important for everyone to confront these. When people learn their HIV status early, important benefits result, both in terms of prevention and care (Page, Louw, & Pakkiri, 2006:60).

### **2.4.3 Prevention Technology**

#### **Condoms**

Studies have shown an increased acceptance of condom use, especially among young people. It is important, however, that consistent messages are provided which promote and support the proper and continued use of condoms. The male condom, when used consistently and correctly, is an effective means of preventing HIV infection.

The first record of condom use comes from Egypt, where hieroglyphics from before 1000BC show men wearing sheaths over their erect penises. Condoms were used during the Roman Empire and the word condom is probably derived from the Latin word "condon", meaning receptacle. In Europe, during the seventeenth and eighteenth century condoms made from linen or animal intestines were available for the prevention of pregnancy as well as prophylaxis against sexually transmitted infections. The rubber condom, as we know it today, was first widely produced after the vulcanization of rubber was patented in 1844 (Abdool Karim & Abdool Karim, 2005:172).

#### **Microbicides**

A new experimental method that might expand our current armamentarium to prevent HIV infection is the development of microbicides. The term "microbicide" refers to a range of different products that share one common characteristic: the ability to prevent the sexual transmission of HIV and other sexually transmitted infections (STI's), when applied topically in the vagina before sexual intercourse. These come in many forms including gels, creams, suppositories, films, sponges or rings that release the active ingredient over time. Phase 2 and 3 clinical trials of microbicides, are currently underway in the



developing world; yet no safe and effective microbicide is yet available to the public (Rossouw, 2007).

## **Vaccines**

It is widely believed that a vaccine will be the only effective way to control the pandemic globally. The different subtypes and variants characteristic of the virus, however, pose a major scientific challenge in the development of single or multiple vaccines effective against all major subtypes of HIV. The HIV vaccine undergoing phase III, trials currently in the USA and Thailand (AIDSVAX), was designed to reduce susceptibility only to HIV subtype B, and hence its efficacy in sub-Saharan Africa, where subtype C predominates, is questionable.

### **2.4.4 Post-exposure Prophylaxis (PEP)**

Post-exposure prophylaxis (PEP) is a method of attempting to prevent HIV infection in a person who has been exposed to infected blood or other body fluids, as in the case of occupational exposure or rape. With PEP, anti-HIV drugs should preferably be started within an hour of the injury and then be continued for a month thereafter (Evian, 2000:30; Van Dyk, 2004:19).

### **2.4.5 Mother-to-child Transmission (PMTCT)**

Great advances have been made in the prevention of mother-to-child transmission of HIV. Antiretroviral therapy is administered to women during pregnancy, labour and delivery in order to reduce their viral load and lessen the risk of transmission to the baby (Evian, 2000:31; Van Dyk, 2004:19).

Since there is no cure or vaccine for HIV infection, the only way to prevent infection is by prevention itself. The social worker's role in prevention is pivotal, especially in facilitating education regarding sexual behavioural change. Behavioural change is a complex task requiring integrated inter-sectoral approaches, implemented at all levels of society, and sustained over years.



## 2.5 Immune System

The researcher is aware that, in order to understand HIV/AIDS and ARV's, it is also important to understand the working of the human immune system. The immune system is defined by Van Dyk (2004:8) as being the defence force that defends the body from external threats and invasions.

Disease can be caused by a variety of factors, including genetic defects, hormone imbalances, nutritional deficiencies, and infection. Transmission of disease via infection involves the invasion of the body by pathogenic microbes and their subsequent growth in various parts of the body. In HIV infection, the profound immune suppression caused by the virus renders patients vulnerable to opportunistic infections and malignancies.

Opportunistic infections are caused by organisms that would, under normal circumstances, and in the presence of a normally functioning immune system, not cause disease; but, in the presence of the HIV-depleted immune system, do cause significant disease processes.

The median time from HIV infection to AIDS is approximately 8-10 years in developed countries. Different people, however, respond differently to HIV infection. Some people remain healthy and active for as long as 10 to 20 years, with little or no sign of immune suppression, while other people deteriorate rapidly and develop full-blown Aids within 5-7 years, or even sooner.

Botes (2005:119-124) postulates that the reasons why people respond differently to HIV infection are:

- Differences in the strains of HIV;
- Differences in the infective dose – people are infected by different 'dosages' of the virus;
- Differences in the human immune response – people respond differently to the virus; and



- Differences in the general health status of the person concerned, affect the course of the disease.

HIV causes a viral infection but elicits an immunological disease. The most important cell involved in the immune attrition is the CD4 T-lymphocyte since it plays a central coordinating role in the immune response (Isselbacher *et al.*, 1999:1853 and Van Dyk, 1999:4-16).

The immune system, which protects the body against viral, bacterial and parasitic infections, is directly attacked and destroyed by the HIV; this makes the virus so unique and fatal. No other virus known in the history of humankind has been manifested in this manner. In order to understand the danger of AIDS fully, the functioning of the immune system must also be understood by patients and counsellors.

### **2.5.1 CD4 Cell Count**

CD4 lymphocytes are a subset of the white blood cells and play an important role in keeping the immune system healthy. The CD4 lymphocyte count is the single, most important test for determining an individual's immune status.

The CD4 count is used as a reflection of the damage incurred by the immune system as well as immune system restoration in patients on antiretroviral therapy. It is the best predictor of the risk of opportunistic infections in HIV-infected people. The CD4 cell count has been shown to be an independent risk factor for progression to AIDS and death (Van Dyk, 2004:423).

The CD4 count of a patient is highly variable and dependent on the individual's immune system, his/her general state of health and the presence of concurrent diseases (South Africa, 2004:2). The normal CD4 count in adults ranges from 600 to 2000 cells/mm<sup>3</sup>. In children, the absolute CD4 count is variable and age dependent, and CD4% should rather be used. A CD4 value below 500 cells/mm<sup>3</sup>, is usually an indication of immune suppression and vulnerability to opportunistic infections. People generally tend to become symptomatic when the CD4 is below 400 cells/mm<sup>3</sup>. According to the World Health Organization



(WHO: 2005), patients with a CD4 count of less than 200 cells/mm<sup>3</sup> are classified as having AIDS.

**Table 4: The CD4 count, CD4% and immune suppression in adults**

CD4 Count	CD4 %	The Immune System
>500	>29	Normal immune function
200-499	14-28	Moderate immune suppression
<200	<14	Advanced immune suppression
<50		Severe immune suppression

(South Africa, 2004)

The above Table reflects the relationship between CD4 count, CD4% and immune suppression in adults. The CD4 count is expressed as an absolute number or a percentage of T-lymphocytes.

Thus, the level of CD4 cells in the peripheral blood is the key parameter to use in monitoring any changes within the immune response. It possesses the following well-defined roles:

- Indicates the degree of immune suppression;
- Establishes the risk of specific HIV-associated complications;
- Determines the need for prophylaxis against opportunistic infections; and
- Assesses response to antiretroviral therapy (WHO: 2006).

The researcher argues that in practice, the CD 4 count can fulfil a very important role in motivating patients to adhere to ART. Patients tend to be motivated when the repeat CD 4 count, taken four to six months after initiation of ART, indicates an increase; this signifies a good response to therapy. The CD4 count can be presented to patients as a practical proof of their response to ART. This value should, however, never be seen in isolation. The precondition, therefore, demonstrates that counsellors, as well as patients, should command an in-depth knowledge and understanding of the effect the HI virus has on the immune system, as well as of the interpretation of both the CD4 count, and the viral load.



It should, therefore, be clear that one of the key tasks of the social worker lies in educating counsellors and patients alike regarding the importance of a proper understanding of the function of the immune system, in general, and the CD4 count, in particular.

### **2.5.2 WHO Staging**

The immunodeficiency that develops during HIV infection forms a continuum, but several discrete clinical phases can be identified. According to the *CDC Staging AIDS Surveillance Case Definition for Adolescents and Adults* (Centres for Disease Control and Prevention, 1993) as well as the *Comprehensive Care and Treatment of HIV/AIDS Antiretroviral Treatment guidelines* (South Africa, 2004), all HIV-infected individuals with either a CD4 count  $<200\text{cell/mm}^3$  or an AIDS-defining condition (WHO stage 4), qualify for the initiation of antiretroviral therapy.

*The Adult HIV and AIDS Staging System of The World Health Organization* (WHO, 2005) uses standardized criteria to clinically stage HIV infection and allows stratification of individuals in terms of clinical criteria and performance status. This staging accommodates facilities where CD4 testing is not freely available, and thus only takes into account clinical determinates and symptoms of the patient. Once a patient has been staged, he/she remains in that stage, unless his/her condition deteriorates, in which case he/she will move to a more advanced stage according to the *HIV/AIDS management for Professional Nurses Manual*, (Foundation for profession Development, 2004:90 and Botes and Levay 2004:13).

The researcher contends that WHO staging could be linked to quality-of-life issues. For example, a person with WHO stage 4 would suffer from severe AIDS-related symptoms, which will impair his/her, quality of life severely. Similarly, a person's WHO stage might improve to stage 1 or 2 after the initiation of ARVs, with concurrent improvement in his/her quality of life. According to the researcher, this improvement is the most important goal of ARV therapy.



The stages can be employed to motivate patients not only to commence ART but, also, to continue with the treatment, long-term according to *the Comprehensive Care and Treatment of HIV/AIDS Antiretroviral Treatment Guidelines* (South Africa, 2003:4). Staging, however, is generally more often used by the medical profession and the terminology is often, not well understood by counsellors.

**Table 5: WHO (2006) staging**

<p>The WHO (2005) proposed staging system for HIV infection and disease in adults and adolescents is:</p> <p><b>Clinical Stage I:</b> Performance scale 1: Asymptomatic, normal activity</p> <ol style="list-style-type: none"><li>1. Asymptomatic</li><li>2. Persistent generalized lymphadenopathy (PGL)</li></ol> <p><b>Clinical Stage II:</b> And/or Performance scale 2: symptomatic, normal activity</p> <ol style="list-style-type: none"><li>3. Weight loss, &lt; 10% of body weight</li><li>4. Minor mucocutaneous manifestation (seborrheic dermatitis prurigo, fungal nail infection, recurrent oral ulcerations, angular cheilitis)</li><li>5. Herpes Zoster, within the last 5 years</li><li>6. Recurrent upper respiratory tract infection (i.e., bacterial sinusitis)</li></ol> <p><b>Clinical stage III:</b> And/or performance scale 3: bed-ridden &lt; 50% of the day during the last month</p> <ol style="list-style-type: none"><li>7. Weight loss &gt; 10% of body weight</li><li>8. Unexplained chronic diarrhoea &gt;1 month</li><li>9. Unexplained prolonged fever (intermittent or constant) &gt; 1 month</li><li>10. Oral candidiasis (thrush)</li><li>11. Oral hairy leukoplakia</li><li>12. Pulmonary tuberculosis, within the past year</li><li>13. Severe bacterial infections (i.e./ pneumonia, pyomyositis)</li></ol> <p><b>Clinical Stage IV:</b> And/or Performance scale 4: bed-ridden &gt; 50% of the day during the last month</p> <ol style="list-style-type: none"><li>14. HIV wasting syndrome, as defined by CDC</li><li>15. Pneumocystis carinii pneumonia</li><li>16. Toxoplasmosis of the brain</li><li>17. Cryptosporidiosis with diarrhoea &gt; 1 month</li><li>18. Cryptococcus, extra pulmonary</li><li>19. Cytomegalovirus (CMV) disease of an organ other than liver, spleen or lymph nodes</li><li>20. Herpes simplex virus (HSV) infection, mucocutaneous &gt; 1 month, or visceral any</li></ol>
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	duration
21.	Progressive multifocal leukoencephalopathy (PML)
22.	Any disseminated endemic mycosis (i.e. histoplasmosis, coccidioidomycosis)
23.	Candidiasis of the oesophagus, trachea, bronchi or lungs
24.	Atypical mycobacteriosis, disseminated
25.	Non-typhoid Salmonella septicaemia
26.	Extra pulmonary tuberculosis
27.	Lymphoma
28.	Kaposi's sarcoma (KS)
29.	HIV encephalopathy, defined by CDC

(WHO: 2006)

An explanation of the above table, illustrating the clinical staging (WHO: 2006) of HIV and AIDS and its link to the viral load, CD 4 count as well as quality-of-life, follows:

A patient in Clinical Stage 1 (Asymptomatic), will possibly have a low viral load and a high CD 4 count (>200) and function according to the Karnofsky score (WHO, 2005) at 90% to 100%: which is independent, with minimal symptoms. While, on the other hand, a patient in Clinical Stage 4 (AIDS-related complex or full-blown AIDS), would possibly show a high viral load and a low CD 4 count (<200) and function according to the Karnofsky score, possibly at 20% to 50%, is dependent or needs hospitalisation.

## 2.6 Summary

In the present chapter, the researcher discussed the clinical aspects of HIV/AIDS: the aetiology of AIDS, modes of transmission, the human immune system, the morphology of HIV, the pathophysiology of HIV, the CD4 cell count, WHO staging, viral load, and diagnosing HIV by means of VCT.

HIV/AIDS and specifically, its effects on the immune system are complex issues that should be understood by the team members who deal with HIV/AIDS patients. In order to render effective counselling and treatment services, aspects such as the CD 4 count, viral load and WHO staging, should be linked



to quality-of-life issues in order to motivate patients to commence, and continue, ART.

In the following chapter, the author discusses the goals of antiretroviral therapy (ART), different antiretroviral drugs, response to, and prognosis of antiretroviral treatment, treatment failure, side-effects, and drug interactions.