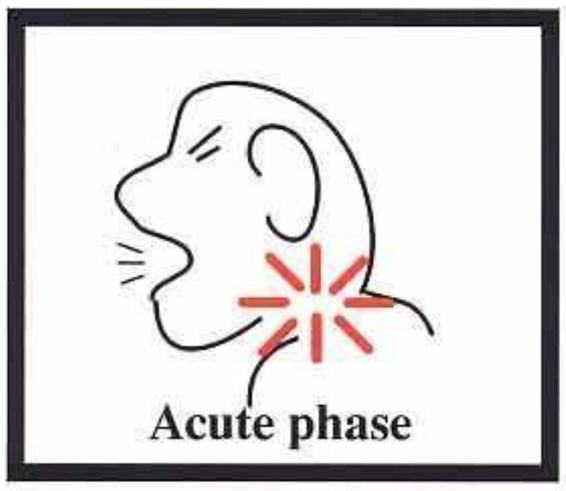
CHAPTER SEVEN

AIDS

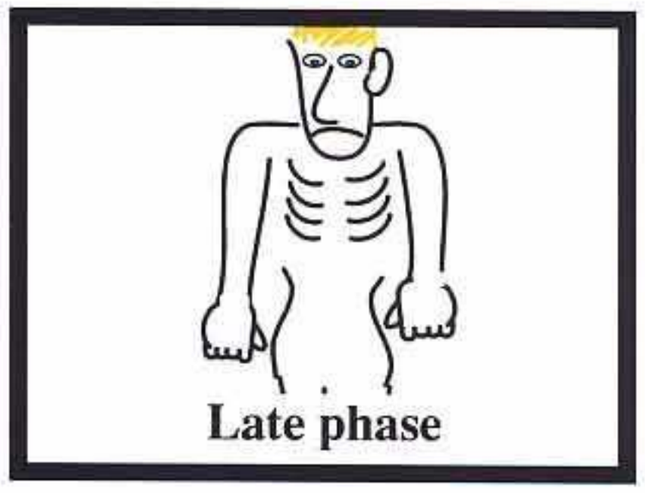
FROM HIV INFECTION TO 'FULL BLOWN' AIDS

HIV : Human Immunodeficiency Virus
AIDS : Acquired Immunodeficiency Syndrome



For a few weeks there are strange rashes and flu-like symptoms.

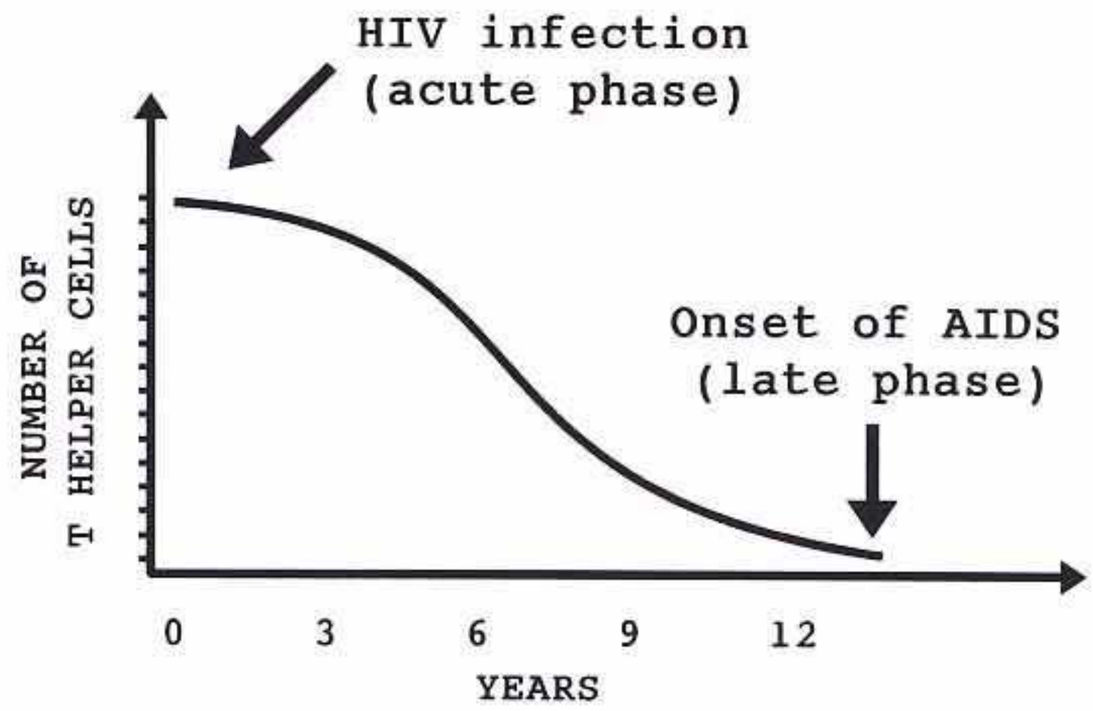
Years can now follow with few, if any, signs or symptoms.



Suddenly, rare cancers and opportunistic infections start to appear.

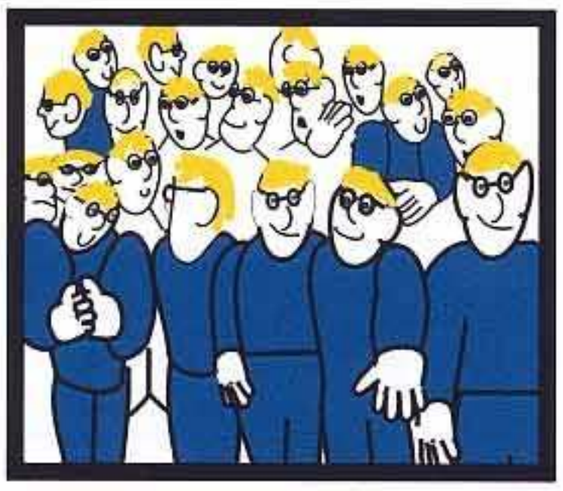
Easy reading

Technical information

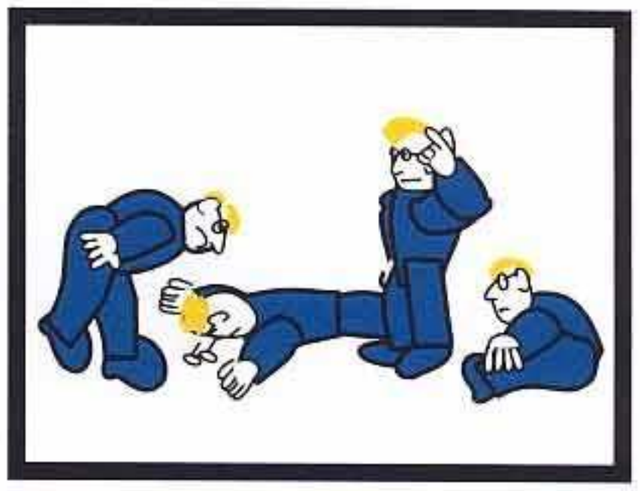


Following HIV infection, T helper cell numbers gradually dwindle to nothing.

A PHOTOCALL OF T HELPER CELLS



Just prior to
HIV infection.

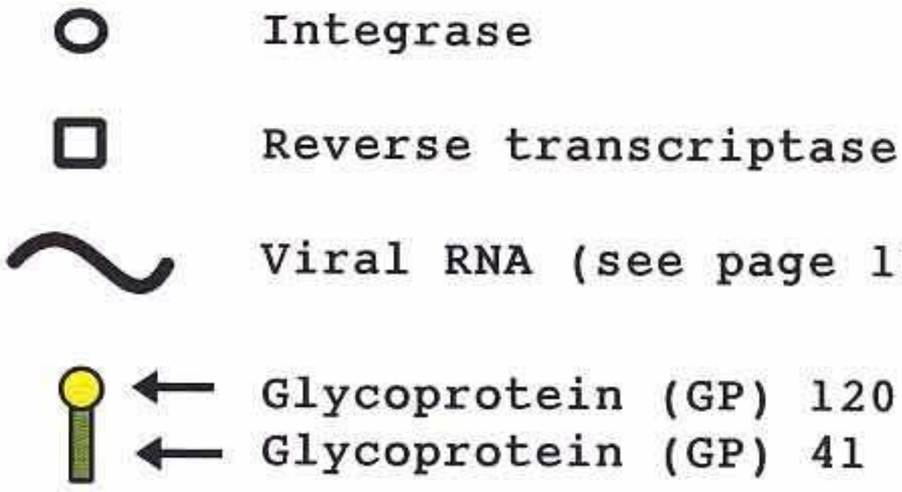


Many years later and few
T helper cells are now left.

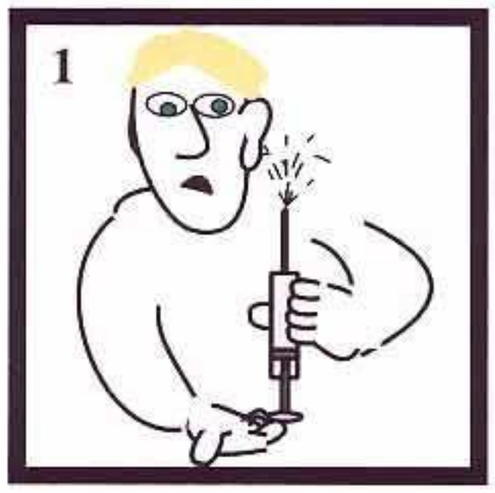
**With few T helpers left to defend the body,
this heralds the onset of 'full blown' AIDS.**

THE HUMAN IMMUNODEFICIENCY VIRUS (HIV)

Inside the
AIDS virus.



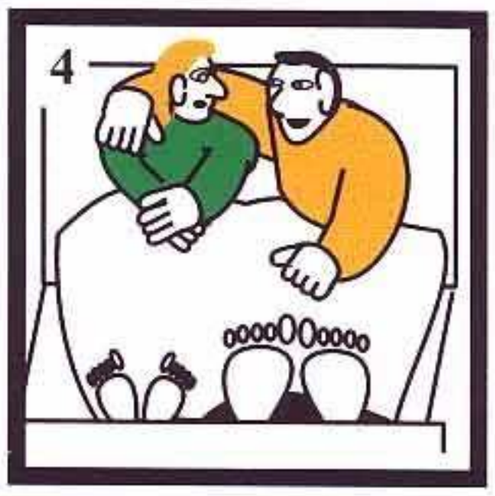
SOME OF THE WAYS PEOPLE BECOME INFECTED BY THIS VIRUS



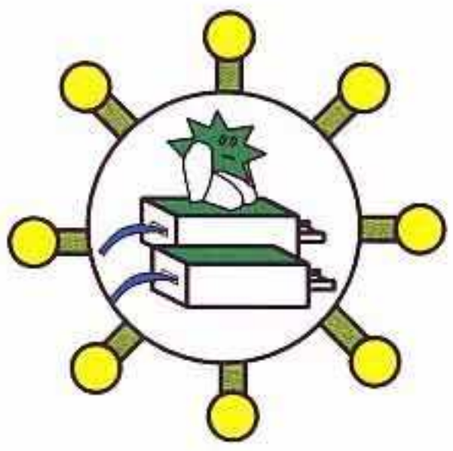
Using a contaminated (infected) needle.

The virus can pass from a mother to her unborn baby.

Receiving infected blood products.

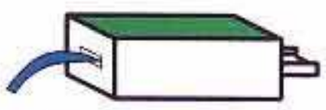


Unprotected sex!!



Inside the AIDS virus.

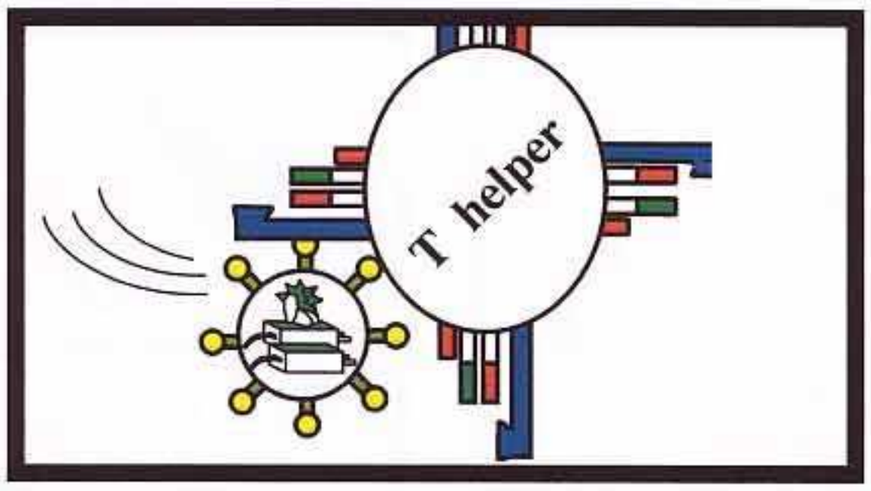
Integrase



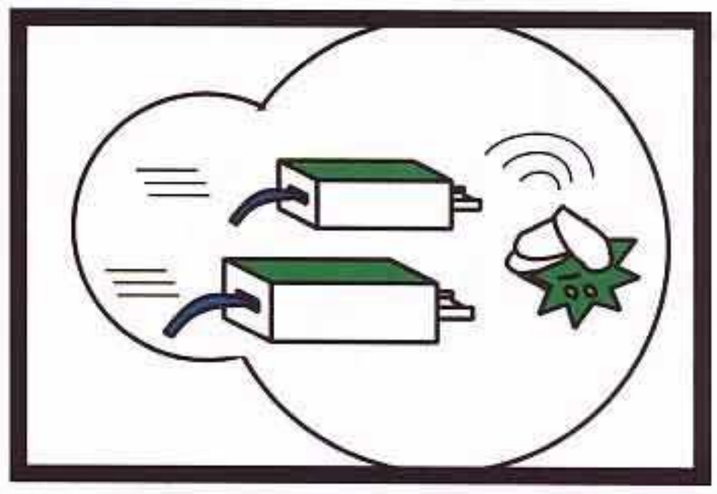
A photocopier (reverse transcriptase)

Viral RNA

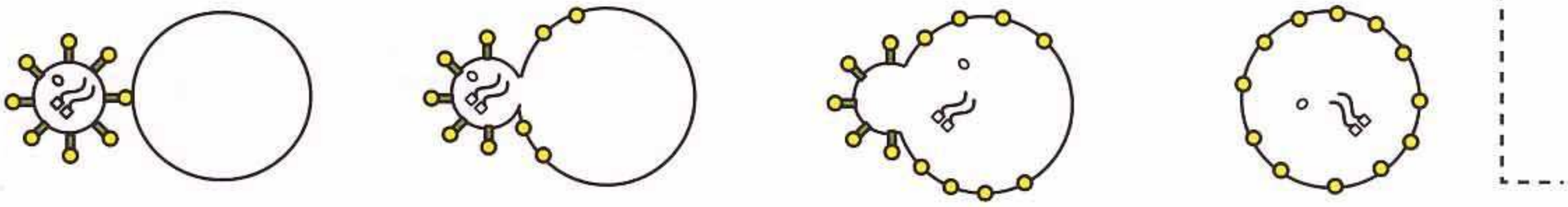
The anti-AIDS drug AZT, works by inactivating reverse transcriptase (the viral photocopiers).



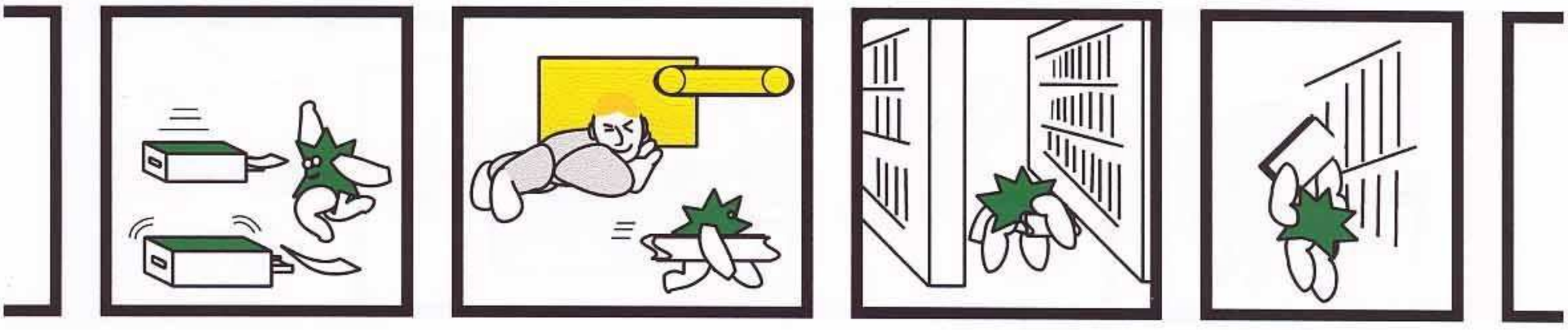
The AIDS virus uses its GP120 protein, to attach onto the T helper's CD4 surface molecule.



Soon its contents have flowed into the T helper cell.



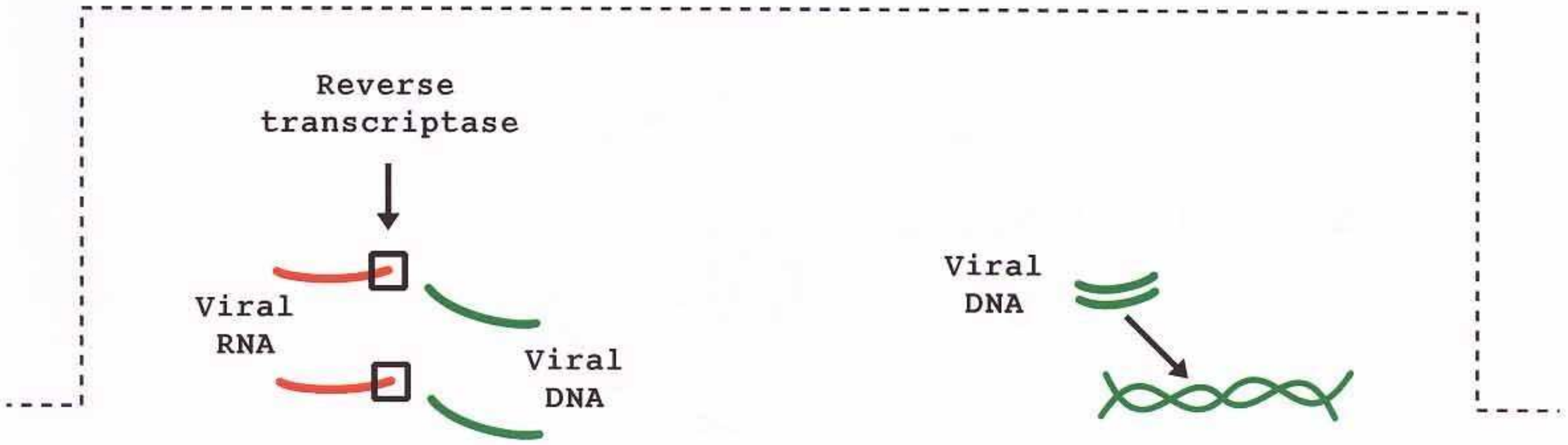
As the 2 membranes fuse together and viral contents flow into the T helper, note how the viral membrane becomes incorporated into the T helper cell's membrane.



The photocopiers start working and release viral DNA.

Integrase now picks up the DNA and takes it into the design centre.

Making his way over to where all the reference books are kept, he slots in his DNA with that belonging to the host.

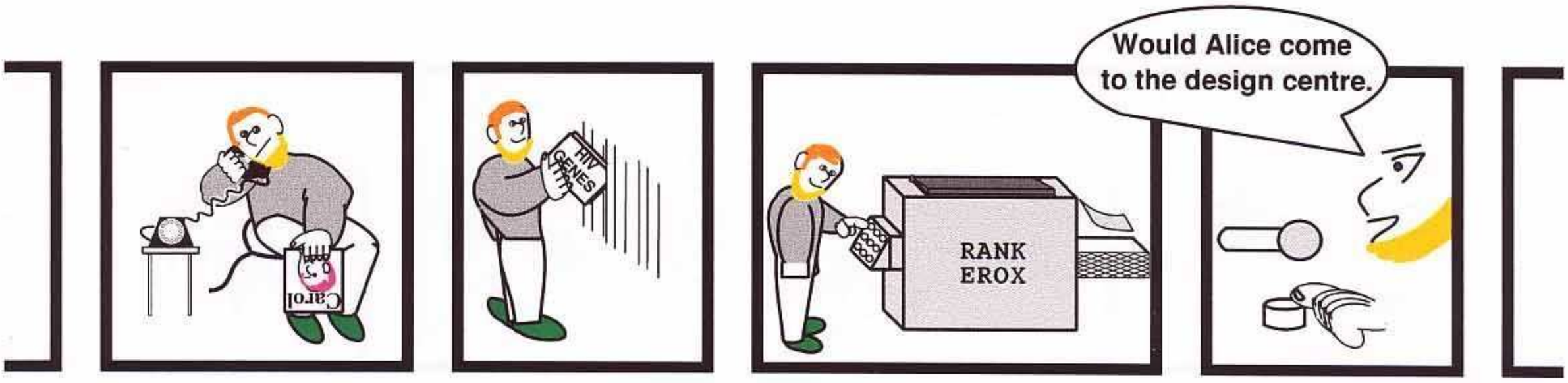


Reverse transcriptase produces viral DNA which is transported into the nucleus.

Integrase then inserts the viral DNA onto one of the host's chromosomes.

WEEKS, MONTHS OR EVEN YEARS LATER

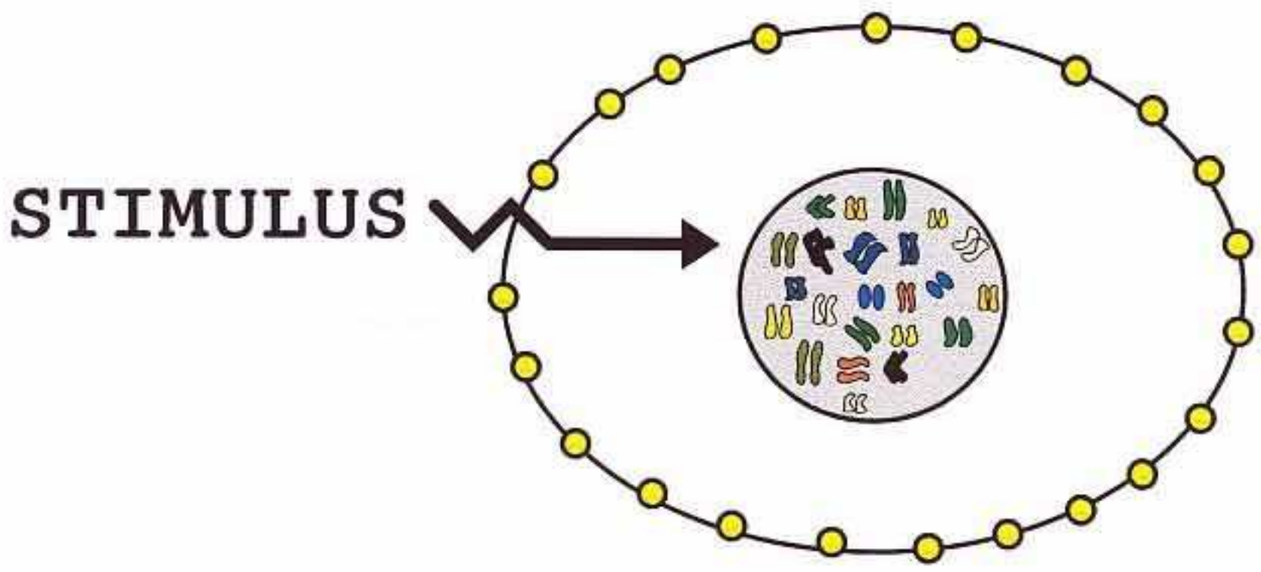
Back inside the infected T helper cell.



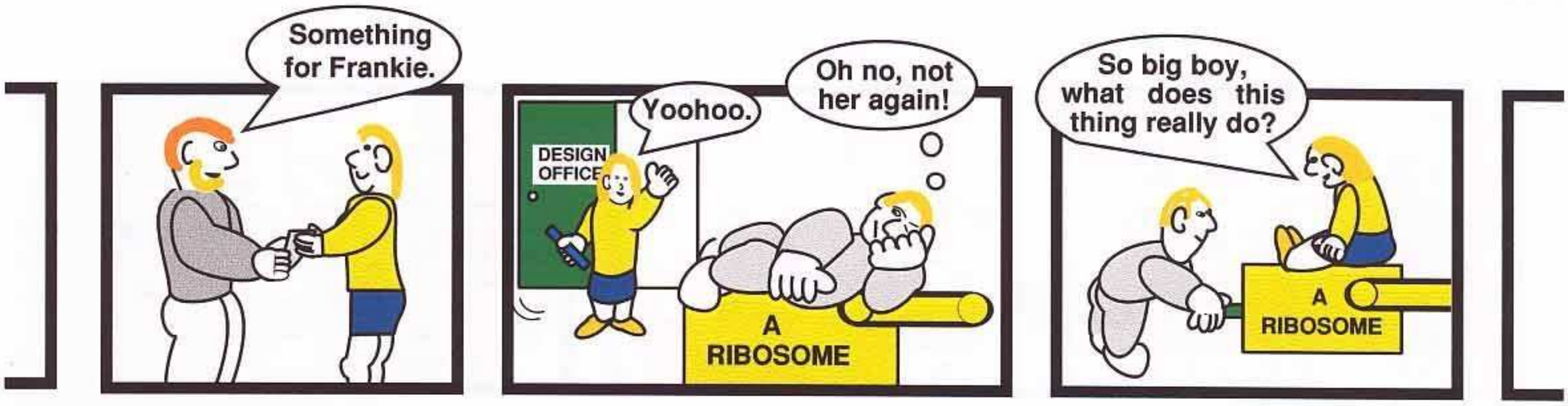
Nick receives a call telling him to start HIV production.

First he finds the HIV manual.

Then it's off to his photo-copier for a messenger RNA copy.



At present it is not known what triggers an infected T helper cell into commencing HIV gene transcription.

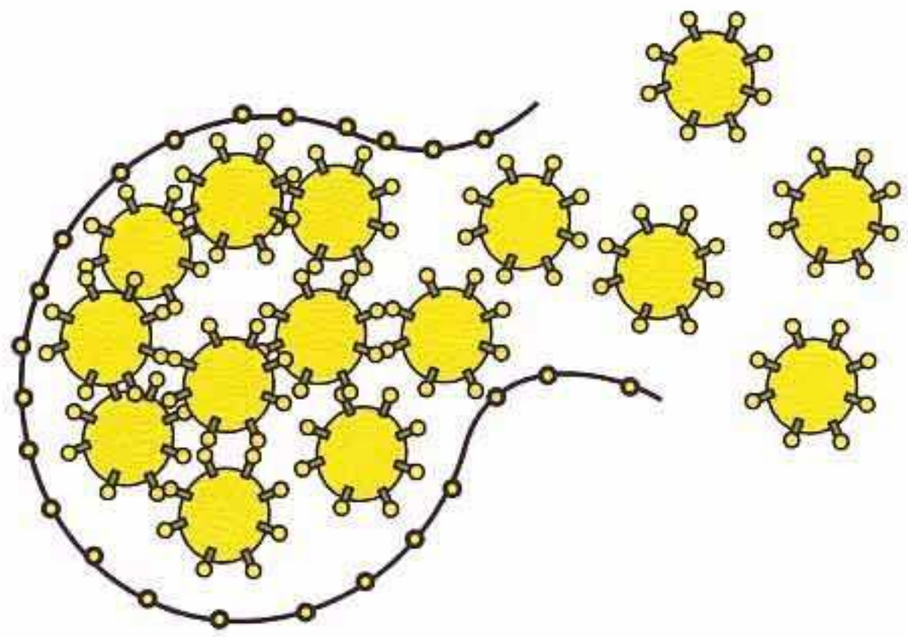


As Nick hands Alice the RNA, he's unaware it will be their death sentence.

On receiving the RNA, Frankie feeds it into a ribosome.

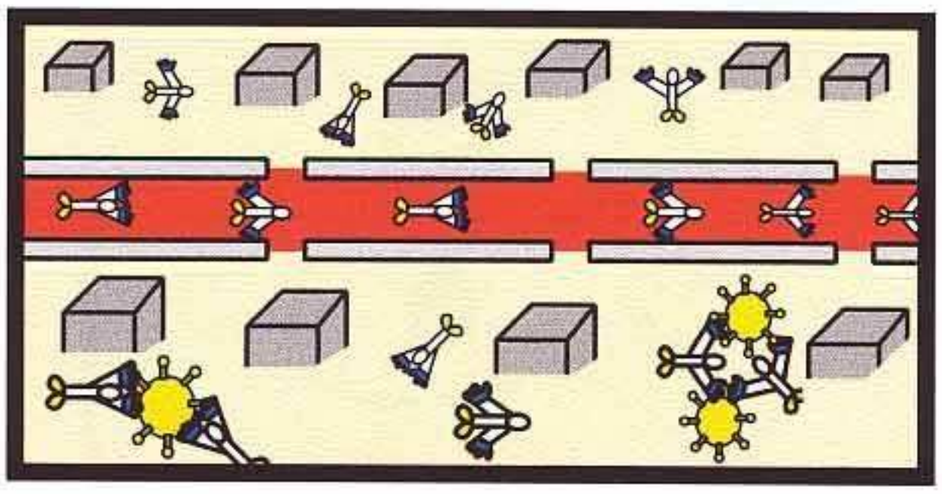
For ribosomal translation, turn back to page 132.

Suddenly from the ribosome, parts of the AIDS virus start appearing.

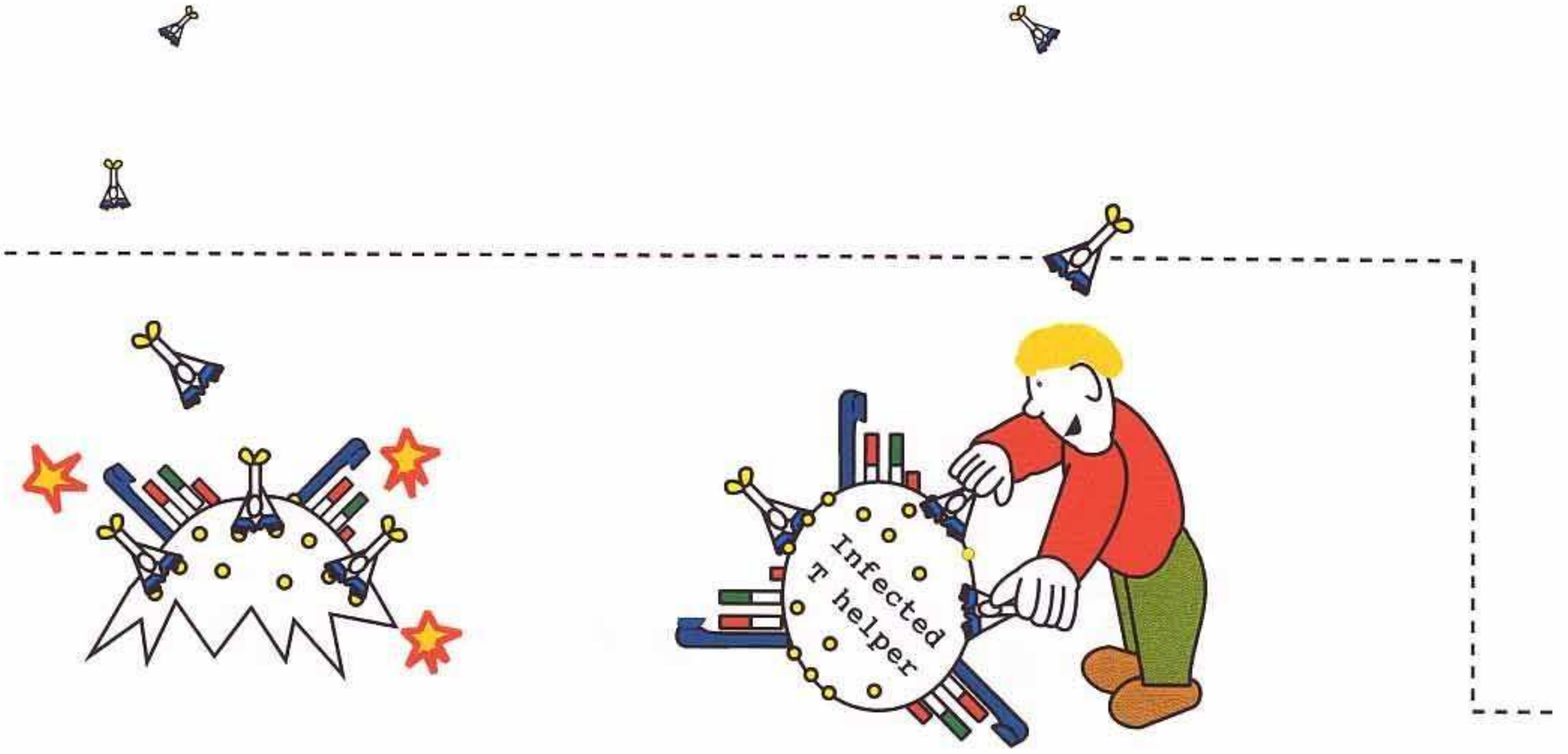


Once HIV production starts, it is not long before
the infected T helper is filled to bursting point!

OTHER POSSIBLE CAUSES FOR DECLINING NUMBERS OF T HELPER CELLS



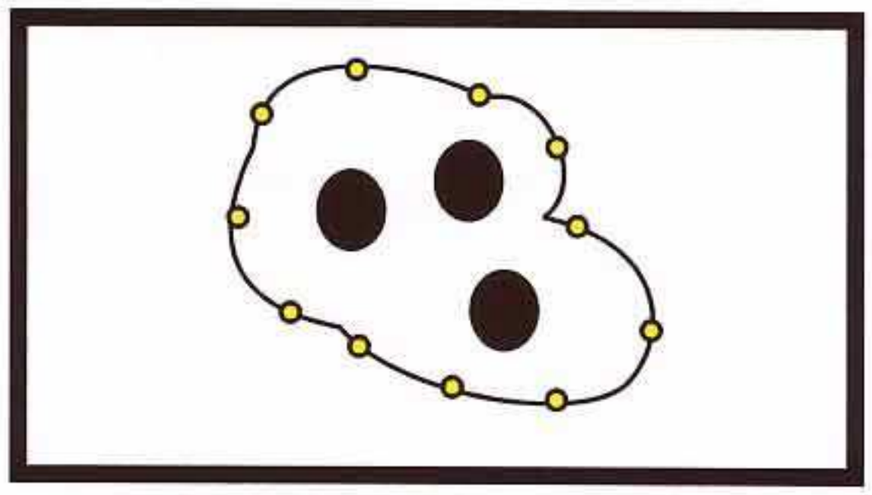
A couple of weeks after becoming infected, anti-HIV antibodies start to appear and quickly mop up any exposed HIV's.



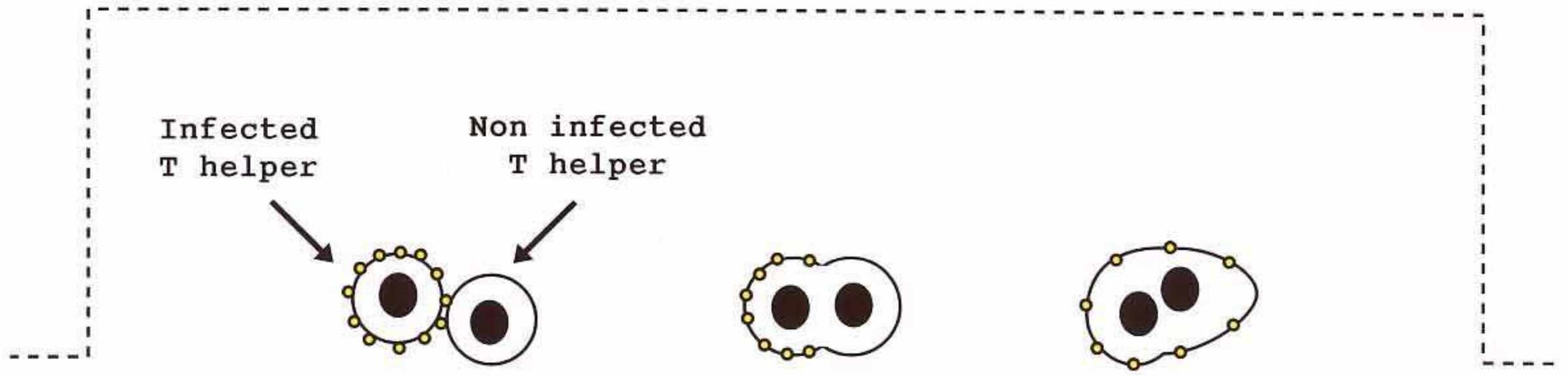
The anti-HIV antibodies may 'grab' viral proteins found on the surface of infected T helper cells. This could then activate complement or encourage macrophages to kill them.

SYNCYTIA

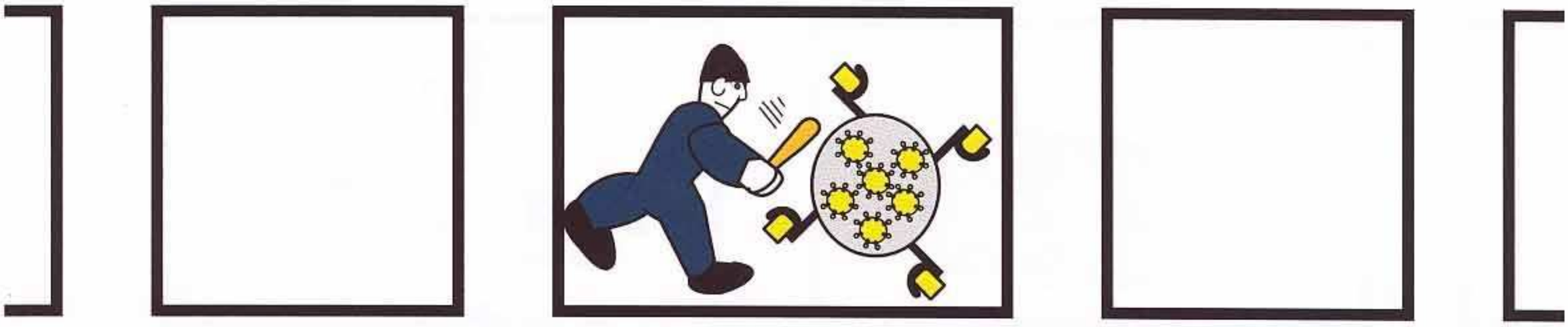
These useless cells, are found in HIV positive (infected) patients.



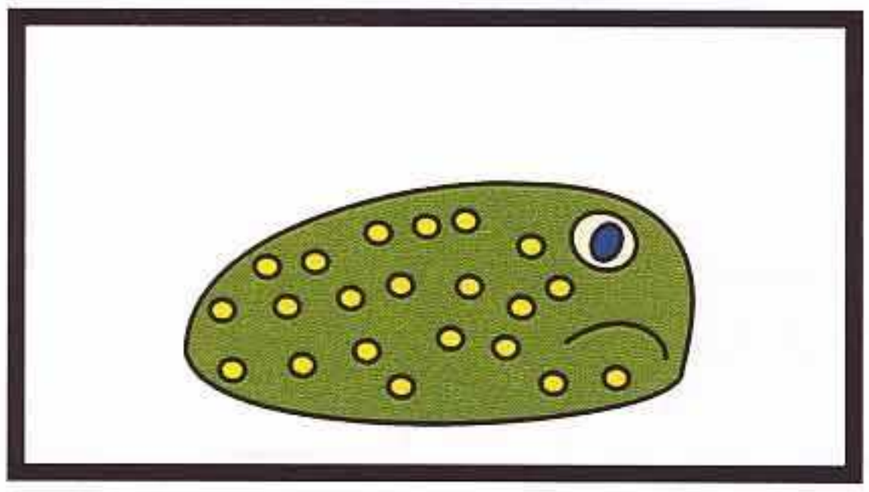
Syncytia are abnormally large multi-nucleated useless T helper cells, which survive for only a short period of time.



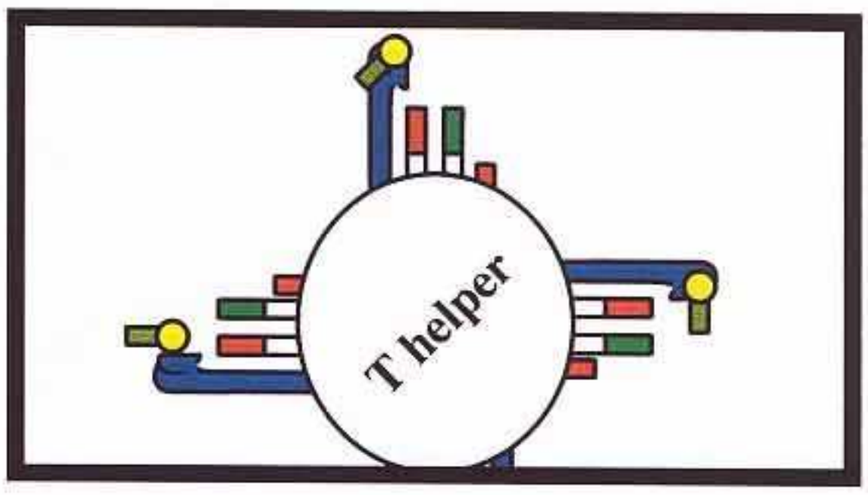
Infected and non infected T helper cells appear to coalesce. This may be due to the HIV proteins found on the surface of infected T helper cells.



Infected T helpers may well be killed off by T cytotoxic cells.



As macrophages can harbour the AIDS virus, this could be a factor in causing declining T helper cell numbers.

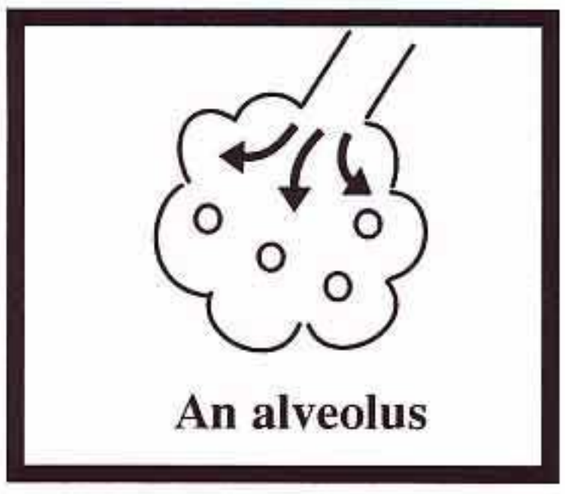


'Free' viral proteins like GP 120, could become attached to uninfected T helper cells and stop them functioning.

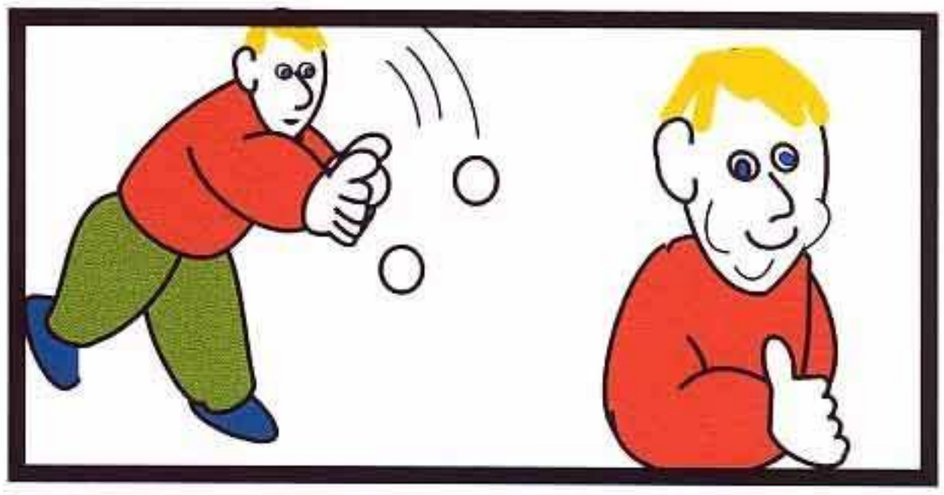
Note that both macrophages and T helper cells express the CD4 surface molecule.

PNEUMOCYSTIS CARINII

Although this organism is harmless to most of us, it will kill many AIDS patients.



The organism is inhailed into the lungs.



Although these parasites are then 'eaten' by local macrophages, to 'digest' them, they will now need help from the T helper cells.

THE LIFE CYCLE OF PNEUMOCYSTIS CARINII

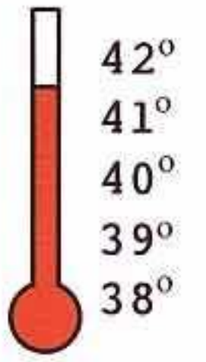
The parasite enters the lungs as a trophozoite (1). It then enlarges into a cyst with a thick protective coat (2). Sporozoites now develop inside it (3). Soon these burst out to start a new life-cycle as trophozoites (4).

SOON THE LUNGS START TO CLOG UP

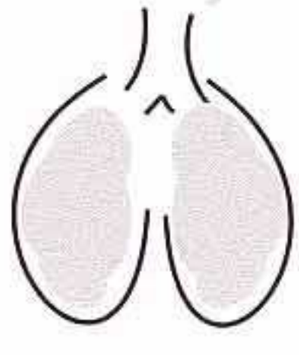
AIDS patients have few if any T helper cells to now assist the alveolar macrophages. So the trophozoites and cysts are not eradicated and these begin to 'fill up' the victims lungs. Soon a life-threatening pneumonia develops, which unfortunately, is frequntly fatal.

ASSOCIATED SIGNS AND SYMPTOMS

Rapid breathing.



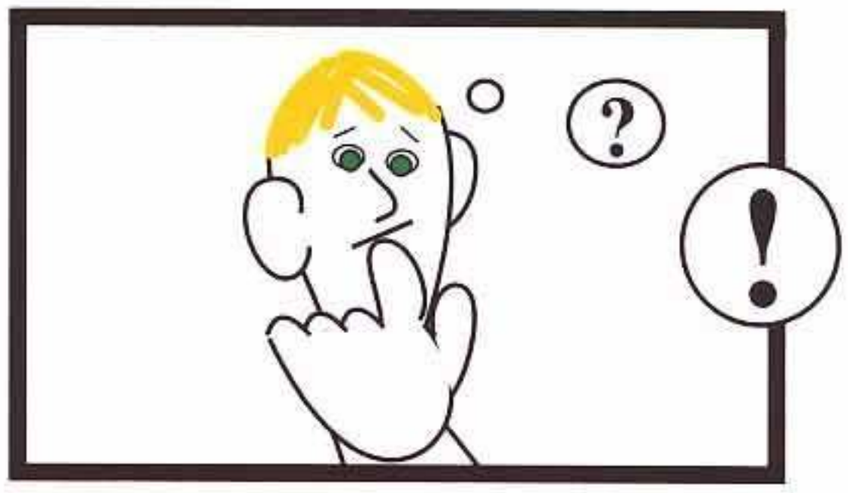
A raised temperature.



X-rays show both lungs are affected.

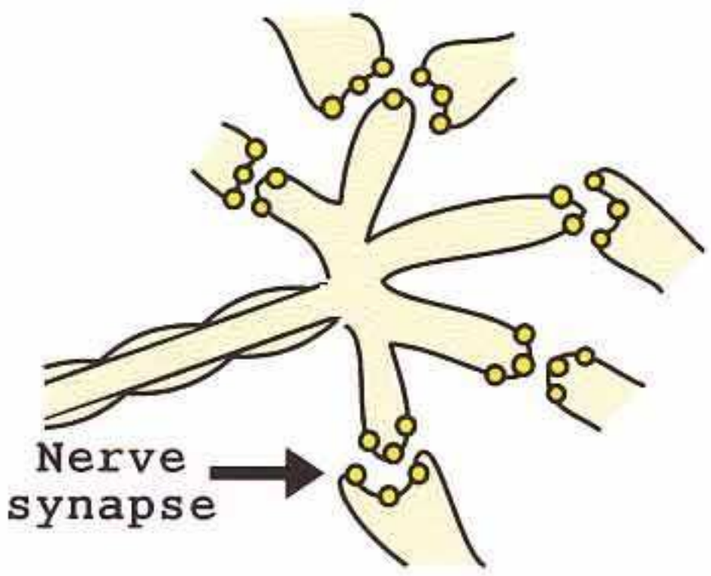
A lung biopsy.

NEUROLOGICAL COMPLICATIONS

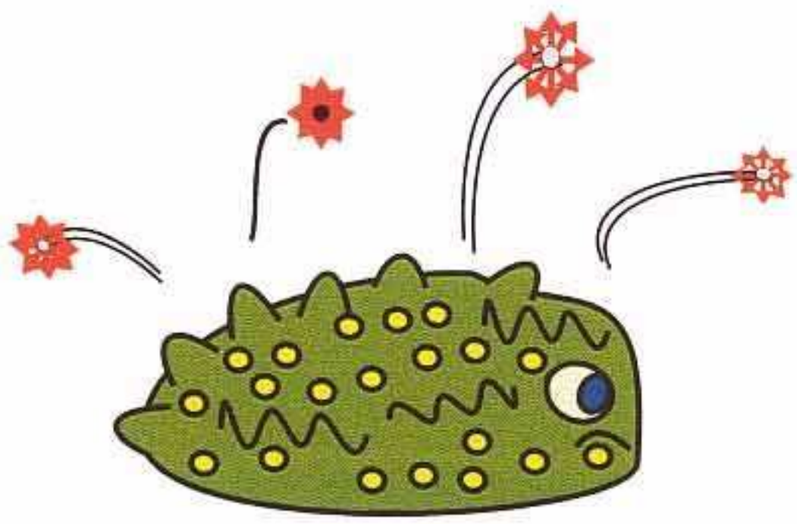


Many people suffering from AIDS develop memory loss and dementia.

POSSIBLE CAUSES FOR THE MEMORY LOSS AND DEMENTIA



Viral particles like GP 120, might become attached to nerve synapses in the brain, disrupting normal neurological function.



Infected brain macrophages might release excessive amounts of inflammation, which could harm the brain.