**VIGS in Suid-Afrika – wie, wat en waar?**

Aan die Redakteur: Gerhard Ras* het in sy onlangs brief verduidelik dat die krediet vir die diagnose van die eerste VIGS-gevalle in die RSA nie aan hom of professor Prozesky nie, maar aan drie kollegas toekom. In sy meegangig kommentaar het die professor Prozesky geskryf dat hy die viriloog in die span was wat die eerste geval in Suid-Afrika gediагnosee het. Dit sou miskien die indruk kon skep dat 'n spesifieke viriologiese diagnose bevestig was.

Die HIV ofwel VIGS-virus is egter vir die eerste keer 'n jaar later in 1983 in Frankryk gekweek. In Suid-Afrika is HIV vir die eerste keer in November 1984 in die Departement Geneeskundige Virologie by die Tygerberg-hospitaal se opleidingskompleks gekweek, wat dit toe moontlik gemaak het om 'n baie doeltreffende diagnostiese immunofluoroesensiesetoets te ontwikkel wat vir etlike jare in die departement gebruik is totdat kommersiële toetsstelle beskikbaar geword het.6,7

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**Aan die Redakteur:** Ek wil graag u aandag daarop vestig dat die persoon wat werkelik verantwoordelik was vir die diagnose van die eerste geval van VIGS in Suid-Afrika, professor I. W. Simon, hoof van die Departement Anatomiese Patologie, Universiteit van Pretoria, is.

Volgens alle inligging tot my beskikking was die diagnose nooit tydens lewe in die eerste pasiënt gemaak nie. Enkele weke na die voltooiing van die lykskouing het die oordelende se familie profesor Simon geskakel om te verneem wat die lykskouingbevindings en oorsaak van dood was. Tydens die gesprek hy besef dat die pasiënt homoeksaauel was, die spreekwoordelike 'pennie' het geval en profesor Simon het die diagnose van VIGS gemaak en dit ook so aan dr. T. Hamersma oorgeda.

Na my mening kom die eer dus profesor Simon toe.

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**Non-cirrhotic portal hypertension**

To the Editor: In their paper entitled 'Non-cirrhotic portal hypertension – a new entity in South Africa?', Jaskiewicz and Robson* report on 6 patients. I would like to point out that non-cirrhotic portal hypertension (NCPH) is **not** uncommon in this country, especially in the Transvaal, where schistosomiasis is endemic in the north-eastern region. We published a paper* on this problem in 1988, emphasizing that the two important causes of NCPH in South Africa are bilharzia (7 of our patients) and portal vein thrombosis (8 patients). These 15 patients were treated between 1 June 1986 and 31 May 1987.

Splenectomy is not recommended, since this precludes the performance of a distal splenorenal shunt (DSRS), which should cure the condition permanently. These patients have a good prognosis, although they fare very well with regular sclerotherapy and ablation of varices alone.2


**Proteinuria and the acute mononucleosis-like illness associated with seroconversion in HIV infection**

To the Editor: During December 1988 a 32-year-old woman was admitted to Somerset Hospital with a pyrexial illness of 1 day's duration. Examination revealed conjunctivitis, mild neck stiffness, a diffuse erythematous macular rash and tender cervical lymphadenopathy. A progressive lymphocytosis developed, peaking in the 2nd week of illness at 74% of the total white cell count of 26,1 x 10⁹/L. Many atypical lymphocytes were noted. Mild proteinuria (1+ on dipstick examination) was present on admission. The serum creatinine value was normal at 87 μmol/L.

During the next 2 weeks the patient developed splenomegaly, generalised lymphadenopathy and oral and vaginal candidiasis. At this stage the 24-hour urinary protein excretion was 2.9 g. During the 5th week of illness the serum creatinine value peaked at 185 μmol/L, occasional granular casts were seen in the sediment, and 24-hour urinary protein excretion was 0.83 g.

The patient was initially assessed as having a septicaemic illness, but all blood cultures were negative and she was found to be HIV-positive (Wellcozyme HIV Recombinant enzyme-linked immunosorbent assay (ELISA)) during the 3rd week of illness. Although Western blot testing at that stage was negative, the p24 antigen was shown to be present a week later. A third specimen 17 days later was strongly positive on ELISA and positive on Western blot testing (all viral bands except p17 were present – LavBlot; Pasteur Diagnostics).

The illness, characterised by persistent pyrexia and lassitude, began to resolve during the 4th week, by which time the serum creatinine value had returned to normal. The patient had muscle pain for 2 weeks with a persistently raised creatinine kinase (CK) value, but did not have other biochemical features of rhabdomyo-