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Provinsiale hospitale, staatshospitale en mediese skemas

Aan die Redakteur: Ek verwys na twee berigte in *Die Burger* van 1 Mei 1990 onder die opskrifte '[Provinsiale] Hospitaalgelde styg skerp' en 'Strenger beheer oor privaathospitale, toerusting kom'.

As die provinsiale hospitale nou gaan 'siekefondsfooie' vra, beteken dit dat die provinsiale hospitale besig is om te privatiseer en dat die Staat dus in direkte kompetisie met die privaatsektor tree. 'Strenger regeringsbeheer by die oprigting van privaathospitale en veral duur toerusting' is 'n terugwaartse stap vir privatisering en demp private inisiatief. Uit hierdie twee berigte kan afgelei word dat staatshospitale geprivatiseer en privaathospitale genasionaliseer gaan word!

'Staatsgeriewe word ten volle benut'. Daarenteen moet ons dikwels hoor dat die privaatsektor personeel weglok van die openbare sektor, dat daar nie genoeg personeel is nie en dat sale gesluit moet word. As gevolg van die inisiatief van die privaatsektor is dit soms nodig dat pasiënte van staatshospitale tydelik na privaathospitale moet gaan vir sekere prosedures.

As sake teen hierdie tempo ontwikkel, sal ons dit moeilik vind om pasiënte te kry vir ons opleidingshospitale. Opleiding moet ook deur die privaatsektor onderneem word. Dit is die enigste oplossing vir ons huidige tekort aan opgeleide personeel. As dit gebeur, sal die koste egter nie so radikaal besnoei kan word soos wat in die berigte voorsien word nie. Tweedens sal privaat- of siekefondspasiënte net moet aanvaar dat hulle beskikbaar moet wees vir kliniese materiaal waar nodig. Daar moet groter samewerking wees tussen die openbare en privaatsektore. Dit is noodsaaklik dat die antipatie, en soms nydigheid, wat dikwels tussen die twee sektore waargeneem word, aangespreek word en met positiewe benadering uitgeroei word.

Volgens *Die Burger* sal siekefondse 'n deurslaggewende rol speel in die beheer van koste deur hul voordele aan lede te bepaal. Dis ou nuus — hulle doen dit alreeds. Die Wet op Mediese Skemas No. 72 van 1967 soos gewysig gee by implikasie 'n vereniging (Verteenwoordigende Vereniging van Mediese Skemas) byna dieselfde magte as dié waarop 'n statutêre organisasie kan staatmaak. Hierdie situasie is mettertyd as 'n voldonge feit aanvaar. Siekefondse word nêrens die wetlike reg gegee om as monitor ('big brother') op te tree nie. Hulle is bloot instansies wat die werkgewer en werknemer moet probeer tevrede hou en in dié proses hul eie boeke laat klop. Die Wet op Mediese Skemas is vandag uitgedien en is slegs 'n relikwie wat sedert die depressie van die dertigerjare geskep, herskep en misvorm is. Die wet behoort geskrap of in sy geheel hersien te word.

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Absence of cardiovascular disease in a rural community using soft water

To the Editor: With reference to the article by Derry *et al.*[1] we report some apparently contradictory findings in the traditional-living rural community of Tshikundamalema in Venda.

The Hans Snyckers Institute is involved in a long-term research project in this remote area in Venda in an effort to monitor the development of some diseases associated with a Western lifestyle in an isolated tribe, living very traditionally but being exposed gradually to a changing lifestyle. Their diet is very simple and contains very little in the way of salt, sugar, meat, milk and eggs, while extensive use is made of natural foods from the veld, and water, untreated, from natural sources.

The quality of the drinking water obtained from four different sources, namely the river, a spring, a mountain stream and a ravine, has been analysed and it has been found to be extremely soft, containing $CaCO_3$ levels of 21, 5, 5 and 13 mg/l respectively, compared with 87 mg/l for Pretoria. Iodine was undeterminably low in all the sources and fluoride extremely low (0,05 parts per million).

In spite of this apparently unhealthy drinking water, cardiovascular disease appears to be very uncommon in this population. Only 5% of 276 persons over the age of 40 years (median 59 years) examined in a house-to-house survey had a sitting blood pressure above 140/90 mmHg (Korotkoff phase 1 and 5). Taking age into account no person had real clinical hypertension. The absence of hypertension is *not* an ethnic feature among Venda people, since the condition occurs in other communities in the region.[2]

ECGs were performed on a representative group of adults and found to be normal in 80% of subjects. The 20% of deviations did not represent ischaemia or lengthening of the Q-T interval but only those changes regarded as typical in blacks.[3] No case of peripheral vascular disease was detected.

It would appear that soft drinking water, in contrast to the findings of other authors[4-8] and as a single factor, is not a cause of hypertension or cardiovascular disease in a community where the other factors associated with westernisation are virtually absent.

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Aberrant in vitro *HLA-DR expression in patients with chronic fatigue*

To the Editor: The chronic fatigue syndrome (CFS) is a clinical entity characterised by chronic fluctuating fatigue associated with a multitude of related symptoms, which may vary between patients.[1] It is of unknown causation, but usually follows a presumed acute viral infection.[2]

Several workers have reported on immunological abnormalities in CFS patients[3-5] and recently the activated state of the immune system in some of these patients has been stressed.[4] It was suggested that much of the symptomatology of the syndrome may be the result of cytokine action secondary to this activated state.[2,4]

We previously demonstrated reduced *in vitro* proliferative responses of peripheral blood mononuclear cells (PBMNCs) from some CFS patients when the cells were exposed to the mitogens concanavalin A and phytohaemagglutinin (PHA) (unpublished observation). These reduced responses usually manifested as a combination of higher spontaneous incorporation of [^{3}H]-thymidine in unstimulated cultures as well as a lower increment in the level of radio-isotope incorporation by stimulated cultures, when compared with controls.

TABLE I. PERCENTAGE OF sIg-NEGATIVE PBMNCs EXPRESSING HLA-DR ANTIGEN

	Unstimulated (uDR)	Stimulated (sDR)	Expression index
Patient group ($N = 10$)	**6,1**	**13,4**	**1,2**
Control group ($N = 10$)	**3,5**	**12,4**	**2,54**
Patient 1	**25**	**28**	**0,12**
Control 1	**2**	**14**	**6,0**
Patient 2	**7**	**6**	**−0,14**
Control 2	**2**	**10**	**4,0**
Patient 3	**4**	**3**	**−0,25**
Control 3	**5**	**13**	**1,6**

On account of this observation and because an increase in the number of unstimulated peripheral lymphocytes expressing HLA-DR has been reported for CFS patients,[4] we investigated the expression of HLA-DR on the non-B-lymphocyte PBMNCs of these patients before and after *in vitro* PHA stimulation.

We tested 10 patients referred to us with histories of unexplained chronic fatigue and conforming to the proposed criteria for the CFS.[1] Controls, recruited from the hospital staff, were matched for age and sex. The tests were carried out blind and the laboratory had no knowledge of the origin of a particular sample.

PBMNCs were obtained by Ficoll-Hypaque density-gradient centrifugation of venous blood and were incubated in two groups for 24 hours in RPMI 1640 with 10% fetal calf serum, one group in medium only and the other group in medium and PHA at the final concentration of 4 μg/ml. The percentage of PBMNCs expressing HLA-DR antigen and surface immunoglobulin (sIg) was determined using monoclonal antibodies (Ortho Diagnostics) and indirect immunofluoresence microscopy or FITC-conjugated anti-human immunoglobulin antiserum (Cappel, Organon Technika) and direct microscopy respectively.

The percentages of sIg-negative PBMNCs expressing HLA-DR in the unstimulated (uDR) and stimulated (sDR) cultures were calculated by excluding the sIg-positive cells. An HLA-DR expression index was determined using the equation: index = (sDR − uDR) / uDR.

The mean expression index for the patient group was lower than that for the control group, but this difference was not statistically significant. However, 3 out of the 10 patients tested exhibited expression indices more than 10 times lower than the mean for the patient group (Table I). This was mainly due to the inability of PHA to induce a significant increase in HLA-DR expression (sDR − uDR), although a high background level of expression in uDR was a contributory factor in patient 1 and to a lesser extent in patient 2 (range of uDR for control group: 1,14 - 6,06%).

Larger studies will help to confirm or refute the above data, and because of the cyclical nature of the clinical disease longitudinal studies may define the proportion of patients involved more clearly.

The basis for the observed phenomenon is unknown, but investigating it may possibly help elucidate the role of the immune system in the pathogenesis of the syndrome. The exact phenotype of PBMNCs involved remains to be determined.

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HIV and granuloma inguinale in Durban

To the Editor: Dr Freinkel[1] questions whether granuloma inguinale (donovanosis) disappeared from South Africa for half a century and re-emerged in the late 1970s or was present all the time but remained unrecognised. Following the introduction into routine use of a rapid test for the detection of Donovan bodies in tissue smears,[2] the numbers of cases of granuloma inguinale diagnosed at this clinic increased immediately; 313 new cases (256 men, 57 women) were seen in 1988, a caseload surpassed only in west New Guinea[3] in modern times.

Freinkel cites a report of granuloma inguinale in 1939 and further cases are recorded in the annual reports of the Medical Officer of Health (MOH) for Durban from 1959, when the present sexually transmitted diseases (STD) classification was introduced. In these reports the numbers of new cases ranged from 195 in 1973 to zero in 1979, when old cases only were diagnosed. One explanation for this fluctuation in the recognition of granuloma inguinale may have been confusion with lymphogranuloma venereum (LGV) caused by *Chlamydia trachomatis* L serovars. The terms lymphogranuloma inguinale and granuloma venereum are mentioned in the MOH's report of 1956, and in 1959 attenders with lymphogranuloma inguinale are reported as serving as a source of antigen for the Frei test at the South African Institute for Medical Research in Johannesburg. In 1964 Davis[4] reported 5 patients with lymphogranuloma inguinale caused by *Donovania granulomatis* responding to streptomycin, the standard treatment for granuloma inguinale at that time. However, in all cases inguinal buboes were present and genital ulcers absent and it is more likely that LGV was the correct diagnosis.

Recently granuloma inguinale has been identified as a risk factor for HIV infection among local Zulu men with genito-urinary disease (GUD),[5] who are a key core group in the spread of HIV-1 in Durban.[6] Some countries have virtually eradicated granuloma inguinale, but its lack of recognition and poor control locally is probably a reflection of an overworked STD service with limited resources and an ever-increasing workload. A World Health Organisation consensus statement[7] has stressed the importance of GUD and STD control in reducing HIV-1 transmission. The document also emphasises the need for increased support for programmes of STD prevention and research and clearly identifies an area to be addressed by the Advisory Group on AIDS.

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