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Africa's AIDS problem

SIR,—I welcome the article by Dr F I D Konotey-Ahulu (20 June, p 1593) advocating a deeper research interest in the clinical epidemiology of the acquired immune deficiency syndrome (AIDS) in Africa rather than the current concentration on the seroepidemiology of the infection alone. In addition, may I make a plea for more clinicopathological research.

Very little has been published about the morbid anatomy of AIDS in Africa,[1] and perusal of the abstracts of the third international conference on AIDS (Washington, 1-5 June 1987) yields no more. Despite the known deficiencies of laboratory facilities in Africa[2] local pathologists are looking at AIDS material that I would encourage them to write up. In addition to this department, other laboratories in developed countries help African hospitals with histopathology, and a realistic picture could thus be composed.

There are some points worth concentrating on in particular, where the pathology of AIDS in Africa could illuminate not only African medicine but also medicine in the more fortunate developed nations. Firstly, with regard to tuberculosis, there seems to be an association between infection with the human immunodeficiency virus (HIV) and presentation with extrapulmonary tuberculosis (unpublished observations in Uganda); the histological picture is different from that of classical tuberculosis in being highly necrotic, multibacillary, and non-reactive, as is also seen at necropsy. (There is still, however, no bacteriological proof that this infection is actually *Mycobacterium tuberculosis*.) As tuberculosis is, at present, an even more important global disease than AIDS research into the combined pathology of these two infections is needed.

Secondly, in relation to diarrhoea, "slim" disease is a common form of AIDS in Africa,[3] and a similar picture is seen in developed countries, yet the pathogenesis is unknown. In Kampala a study showed that most patients had associated cryptosporidiosis and isosporiasis,[1] but there is no firm proof that these are the causes of enterocyte damage and diarrhoea rather than being "passengers" while other agents—undetected through lack of careful microbiology—are the cause. Improved correlation of gut morphology, function, and infection in African AIDS would provide invaluable information relevant everywhere in the world.

Thirdly, with regard to malignant lymphoma, Epstein-Barr virus infection and immunosuppression from malaria induced Burkitt's lymphoma in African children long before AIDS arrived, and Burkitt's lymphoma is well described in patients with AIDS in developed countries. The conjunction of HIV and latent Epstein-Barr virus infection in Africa should provide much information about lymphomagenesis in AIDS but there is no published evidence yet[4]; in this department we have seen only three cases of Burkitt like lymphoma in Africans infected with HIV-I and HIV-II.

The list of deficient clinicopathological information about African AIDS could be extended indefinitely (what about paediatric AIDS and neuropathology?). Morbid anatomists may be accused of hyperbole in calling their subject the centre of the medical universe, but there is some justification in thinking that without pathological data proper descriptions of diseases, let alone epidemics, are incomplete.

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2 Hutt MSR, Spencer H. Histopathology services for developing countries. *Br Med J* 1982;285:1327-9.
3 Serwadda D, Sewankambo N, Carswell JW, *et al.* Slim disease: a new disease in Uganda and its association with HTLV-III infection. *Lancet* 1985;ii:849-52.
4 Lucas S, Wamukota W. HIV and local African population. In: Pounder RE, Chiodini P, eds. *Advanced medicine conference 23.* London: Baillière Tindall, 1987.

Diagnosing pulmonary thromboembolism

SIR,—I often wonder if those writing about diagnosing pulmonary thromboembolism appreciate the distinction between massive pulmonary embolism and the much more common phenomenon of pulmonary infarction resulting from smaller pulmonary emboli. Dr W J Windebank (30 May, p 1369) states that "pulmonary embolism" is diagnosed and treated in only 29% of episodes and that of the remainder one third are fatal. That may be the case in major pulmonary embolism, but it is certainly not true of pulmonary infarction caused by fairly small emboli that have no appreciable effect on right ventricular output.

Dr Windebank confuses the issue further by citing the "classic triad" of dyspnoea, pleural pain, and haemoptysis. The first is certainly the dominant symptom in massive pulmonary embolism, but the two others are manifestations of

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