diagnosis of anencephaly may usually be confirmed by amniocentesis and ultrasonography. A termination of pregnancy may then be offered to the parents. Because of the antenatal surveillance programme in Britain the number of anencephalic babies who survive to gestational age is probably much smaller than that in the United States. The royal colleges' report estimates that no more than 20 such donors might be available for heart donation.

It might be thought that parents of an anencephalic baby would find the question of organ donation repugnant. The North American experience suggests otherwise. Dr Stiller reports: "I have been approached by several women carrying anencephalics and their physicians, who have sought to redeem the pregnancies by the use of the organs from these babies after birth to give life to others." Professor Bayley, a professor of surgery from California, has stated: "My experience at a transplant centre has been that most parents who give birth to an anencephalic infant are not only willing for their infant's organs to be donated but are usually insistent." The generally favourable attitude to transplantation suggests that similar sentiments would be found in Britain.

If anencephalic babies are to be used as organ donors then we must establish precisely when they may be regarded as being brain dead. After much deliberation the British and Canadian groups arrived at the same conclusion—that absence of spontaneous respiration would signify death. The British document states that "organs for transplantation can be removed from anencephalic infants when two doctors who are not members of the transplant team agree that spontaneous respiration has ceased." In the adult the diagnosis of brain death together with apnoea is recognised as death.³ The colleges thought that by analogy the absence of the forebrain in these infants together with apnoea might similarly be recognised as death. In practice, respiration is likely to stop within 24 hours after birth, and only a few infants survive for up to seven days. Once respiration has stopped the infant may be ventilated and the organs removed. Diagnosing death in other infants such as those with brain injuries remains a problem. The colleges advised that organs should not be removed within the first seven days of life from neonates with

beating hearts even if they satisfy the brain stem death criteria for older children and adults. In effect, therefore, a moratorium remains on these donors. Both groups emphasised that no pressure should be put on parents to continue with a pregnancy solely for organ retrieval.

Using abortion material for transplantation into the brains of patients with Parkinson's disease should not be confused with transplanting neonatal organs as the legal considerations are different: an aborted embryo will never have been legally viable whereas a neonate of more than 28 weeks' gestation will have been. The clinical implications are, however, similar and complex. At the Canadian conference Caplan addressed the topic as it related to anencephaly and concluded that: "The fact that anencephalic infants lack the minimal capacities and abilities requisite for generating a duty of respect for their humanness does not thereby allow those in medicine to do anything they like with such children. The interest and sensibilities of parents and of society must still be taken into account in considering whether or not such children should be used as tissue or organ sources. . . . If the parents of an anencephalic infant wish to donate their child as a tissue or organ source, and if this can be accomplished in a manner that does not violate the sensibilities and values of the medical profession or society concerning the treatment of human materials, then it would seem desirable to amend existing laws governing organ procurement to include anencephalics as possible organ and tissue sources." In Britain there is no legal definition of death, and therefore no change in the law is required. The colleges' report, nevertheless, gives a strong lead on what is permissible, and its proposals are likely to become accepted practice.

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Genital ulcers, other sexually transmitted diseases, and the sexual transmission of HIV

The first two may be important risk factors for the third

Several gaps remain in our understanding of the spread of HIV in the population. Firstly, there is considerable variation in its infectivity by sexual transmission: partners of people infected with HIV show widely different infection rates. Thus while some people remain uninfected after hundreds of penetrative sexual exposures with a partner with HIV infection others may become infected with HIV after only one or several such exposures. Several studies have shown no correlation between the number of sexual acts with an infected person and the risk of infection in a heterosexual relationship.^{1,2} Secondly, in contrast to the rarity of its occurrence among heterosexuals in Europe, the cause of the rapid heterosexual spread of HIV infection in some populations in sub-Saharan Africa remains an enigma, though several partial explanations have been suggested.³

Hence besides behavioural variables, biological factors

possibly also influence the sexual transmission of HIV. Evidence is growing that genital ulcerations, and perhaps other sexually transmitted diseases as well, increase the risk of sexual transmission of HIV infection. Thus a history of sexually transmitted diseases was significantly more common in both homosexual men and in heterosexual men and women with HIV antibody.^{4,7} This association was independent of the number of sexual partners and became stronger with an increasing number of episodes of sexually transmitted diseases.⁶

Several cross sectional studies in various populations, including homosexual men and heterosexuals, have found a strong association between a history of genital ulcer disease or markers for syphilis, or both, or antibody to *Haemophilus ducreyi*, or antibody against herpes simplex virus type 2, or both, and HIV infection, which is independent of sexual

exposure (R O Cannon *et al*, fourth international conference on AIDS, Stockholm, 1988; P Piot *et al*, unpublished data).⁸⁻¹⁰ Whereas most studies had been performed in sexually transmitted diseases clinics or in prostitutes the same association has also been found in a community based study in Zaire (N Nzila *et al*, third international conference on AIDS, Washington, DC, 1986). Nevertheless, these retrospective data do not permit any conclusions about causality, given that genital ulcers may be a marker for sexual activity and that HIV infection may be complicated by persistent ulceration, particularly with genital herpes. The association found in several studies between actual genital ulceration and HIV antibody supports the hypothesis that it may complicate HIV infection (N Nzila *et al*, third international conference on AIDS, Washington, DC, 1986).^{7,9}

Two prospective studies in the United States have shown that genital herpes precedes seroconversion for HIV antibody in homosexual men,^{10,11} and a cohort study in Nairobi showed that genital ulcerations were an independent risk factor for incident HIV infection in female prostitutes (L F Plummer *et al*, fourth international conference on AIDS, Stockholm, 1988). The study in Nairobi showed that the risk for HIV seroconversion increased with the number of episodes of genital ulceration, which was mostly due to chancroid.

Hence there is convincing evidence that genital ulceration increases susceptibility to HIV infection. Nevertheless, the case for increased infectiousness of people infected with HIV who also have genital ulceration is less clear. In a prospective study in Kenya 293 men were studied who presented with a sexually transmitted disease and a history of recent unprotected contact with a female prostitute from an urban area where over 80% of the prostitutes were infected with HIV (D W Cameron *et al*, international conference on AIDS, Stockholm, 1988). Overall 8% (24) of the men showed seroconversion after a mean of 9.1 weeks. Compared with men with other sexually transmitted diseases those with genital ulceration (mostly chancroid) had an increased risk for seroconversion with HIV antibody (adjusted risk ratio 4.7, 95% confidence interval 1.3 to 17). One interpretation of these data is that most men concomitantly acquired their HIV infection and genital ulceration and that women infected with HIV with genital ulcer disease may be more infectious because they have increased shedding of the virus in the genital tract.

The interaction between genital ulcer disease and HIV transmission is biologically plausible. Not only may HIV penetrate more easily through the disrupted mucosa or skin but the lymphocytes and macrophages associated with the inflammation may represent an increased pool of target cells in a seronegative person and of HIV infected cells in a seropositive one.

The evidence for a role for non-ulcerating sexually transmitted diseases as cofactors in HIV transmission, however, is not so clear cut. Genital chlamydial infection but not gonorrhoea was an independent risk factor for HIV acquisition in a cohort of female prostitutes in Kenya (F Plummer *et al*, fourth international conference on AIDS, Stockholm, 1988) whereas a history of gonorrhoea correlated significantly with HIV infection in the spouses of seropositive subjects (M Fischl *et al*, fourth international conference on AIDS, Stockholm, 1988). In general, genital infections causing an inflammatory response or erosions may potentiate HIV transmission: Van de Perre *et al* detected HIV antigens in lymphocytes from cervicovaginal secretions but not in the genital epithelial cells.¹²

The extent of the role of genital ulcer disease in the population as a cofactor for HIV transmission is not known but may be considerable in areas where ulcerating sexually transmitted diseases such as chancroid are common. The presumed high incidence of genital ulceration in some urban populations may be one reason for the fast spread of HIV infection in sub-Saharan Africa. Nevertheless, sexual transmission of HIV may occur without any concomitant sexually transmitted disease, and other biological factors, such as the stage of disease, and behavioural variables, such as the type of sexual intercourse, also contribute (M Laga *et al*, fourth international conference on AIDS, Stockholm, 1988).⁵⁻¹³

What are the implications of these observations? Firstly, programmes to control sexually transmitted diseases should be strengthened or initiated where they do not exist. Not only will this reduce the incidence of severe complications and sequelae of sexually transmitted disease but it may also interfere with the spread of HIV. The two things not to do are to take the resources for AIDS prevention away from the general budget to control sexually transmitted diseases and to isolate the programmes to prevent AIDS from those to control sexually transmitted diseases. Secondly, the control programmes should focus more on genital ulcer disease, particularly in areas where chancroid is endemic, as this disease is easily treatable with cheap antibiotics and may not be particularly contagious. Thirdly, we need to define the impact of reducing the incidence of genital ulceration on the spread of HIV and to evaluate any interventions. Fourthly, as patients with genital ulcer disease are at higher risk for HIV infection they should be counselled about the infection and its prevention. Finally, more research is needed about the role of other sexually transmitted diseases in the transmission of HIV.

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