AIDS, and there is no evidence for the transmission of hepatitis B by mosquitoes. In one extensive laboratory study seven species of mosquitoes were fed artificially on human blood containing hepatitis B. Radioimmunoassay showed that all the ingested hepatitis B surface antigen disappeared in parallel with digestion of the blood meal by the mosquito. Furthermore, the infectivity of LAV/HTLV-III is less than that of hepatitis B, and the titre of circulating virus is substantially lower. Fourthly, perhaps only one in 10000 infected (T4) lymphocytes produces complete virus (personal discussion with R C Gallo), and it seems unlikely that virus would be present in the blood meal.

What about other human ectoparasitic insects? J Maunder (personal communication) considers that the mouthparts of fleas might be better adapted than most for the mechanical transmission of the infection, but the habits of the flea, including host fidelity and infrequent feeding, make it an unlikely vector. The habits of bedbugs are such that these are not efficient vectors of disease in the community. Lice are even less likely to transmit AIDS, partly because of the effective one way valves of the mouthparts, partly because the mouthparts are unwettable and are emptied and cleaned after use, and partly because lice have not been implicated in the transmission of viral diseases.

Nevertheless, research is needed into the survival of virus introduced by blood meals and by inoculation into mosquitoes, and this work is in progress at the London School of Hygiene and Tropical Medicine. Equally careful surveillance and collection of data on AIDS must continue in all countries. At present, however, it is evident that the time picture of the epidemiology of AIDS does not include transmission by blood sucking insects.

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- 1 Curran JW, Morgan M, Hardy AM, Jaffe HW, Darrow WW, Dowdle WR. The epidemiology of
- AIDS: current status and future prospects. Science 1985;229:1352-7.

 Melbye M. The natural history of human T lymphotropic virus-III infections: the cause of AIDS. Br Med 7 1986:292:5-12
- Biggar RJ. The AIDS problem in Africa. Lancet 1986;i:79-82
- 4 World Health Organisation. Acquired immunodeficiency syndrome—update. World Health Organisation Weekly Epidemiological Record 1986;61:85-8.
- World Health Organisation. Atelier sur le SIDA en Afrique Centrale, Bangui. Geneva: World Health Organisation, 1985.
- 6 World Health Organisation. AIDS: the search for clues. WHO Chron 1985;39:207-11
- Byrom NA, Davidson G, Draper CC, Zuckerman AJ. Role of mosquitoes in transmission of hepatitis B antigen. J Infect Dis 1973;128:259-60.

Sarcoid heart disease

The first case of sarcoidosis affecting the heart was reported by Bernstein et al in 1929, and since then many case reports and reviews have been published. The most recent of these, in the Mayo Clinic Proceedings, highlighted the characteristic features.² Analysis of a large series has provided a comprehensive picture of the variations of this fascinating disease. 3-6 In our own series we now have 290 cases seen in Britain, 100 with necropsy confirmation.

Though cardiac lesions may occur in any patient with sarcoidosis and at any stage, sarcoid heart disease most often presents with cardiac symptoms—and only when sarcoidosis is suspected and sought is the aetiological diagnosis established. This is done by seeking clinical evidence of sarcoidosis, preferably with histological confirmation by biopsy of the skin, a lymph node, bronchus, or lung or a Kveim test. Endomyocardial biopsy is diagnostic if the result is positive, but the patchy distribution of the lesions makes a negative finding of absolutely no value.⁷ At necropsy the characteristic pattern is massive heart lesions with minimal sarcoidosis in other organs. The classic giant cell granulomas with fibrosis may be confluent and occupy enormous areas of the myocardium or may be microscopic and diffuse. Haemodynamically important valve lesions are rare, and the pericardium is more commonly affected than is clinically apparent. The frequency of extensive replacement of the conducting system by granuloma is consistent with the clinical presentation of heart block, sudden death, and complex arrhythmias.

The common patterns of presentation in order of frequency are complete heart block, ventricular extrasystoles or ventricular tachycardia, myocardial disease with failure, sudden death, first degree heart block or bundle branch block, especially right, supraventricular arrhythmia, mitral valve dysfunction simulating myocardial infarction, and pericarditis. Many of these features may appear in any one case, and sarcoidosis should be suspected in any patient younger than the usual with complete heart block or unexplained heart failure, particularly with complex arrhythmias difficult to control.

The age range of patients is wide—in our experience from 18 to 88 with a peak between 25 and 55. We now know that the prognosis in the individual case is not necessarily bad, particularly with energetic treatment. Many patients have survived serious illness for 10 years and a few for even 20 years.

Of the many methods of investigation, 24 hour electrocardiographic monitoring and echocardiography are perhaps the most rewarding. The efficacy of the treatment of arrhythmias must be carefully monitored. Echocardiography may show left ventricular dysfunction, localised dyskinesia, mitral valve dysfunction, septal thickening, and bright echoes—particularly from the ventricular septum and the left ventricular free wall—consistent with fibrogranulomatous infiltration.

Treatment for the cardiac condition will include the whole gamut of drugs. Amiodarone has proved helpful in cases of resistant arrhythmia.8 Heart block must be treated by pacing. In all groups energetic use of steroids can be very beneficial—which explains the importance of the physician making the aetiological diagnosis.

The widely quoted paper of Roberts et al deserves further attention. Regrettably, it omitted reference to the two earlier reports of large numbers of cases3-4 and in its analysis therefore perpetuated the myth that this disease is much more common in blacks. In our series of 290 only 21 were not white. The suggestion that steroids might encourage development of left ventricular aneurysm has inhibited their use. We have frequently found these aneurysms in patients who have received no treatment at all, and we believe the possibility of aneurysm formation need not be considered before deciding to treat with steroids. The prognosis is by no means as bad as suggested by Roberts et al and the outcome is not necessarily related to the extent of the sarcoidosis of the heart. Hearts massively affected by transmural granulomas may continue to perform astonishingly normally, while microscopical lesions can lead to sudden death or, if widespread, to congestive heart failure.6

Several transplant centres have found previously undiagnosed sarcoid of the heart in occasional cases of "cardiomyo-

pathy." In other cases transplantation has been avoided by accurate diagnosis and successful treatment with steroids.

Much remains to be learnt about sarcoidosis, and its effects on the heart are no exception. We do now know a great deal more about sarcoid of the heart and its behaviour, but further study is essential. We would be very pleased to hear of additional cases to include in our study.

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- 1 Bernstein M, Konzellman FW, Sidlick DM, Boeck's sarcoid: report of a case with visceral involvement. Arch Intern Med 1929;44:721-34.

 2 Lemery R, McGoon MD, Edwards WD. Cardiac sarcoidosis: a potentially treatable form of
- myocarditis. Mayo Clin Proc 1985;60:549-54.
- Fleming HA. Sarcoid heart disease. Br Heart J 1974;36:54-68.
- 4 Matsui Y, Iwai K, Tachibana T, et al. Clinicopathological study of fatal myocardial sarcoidosis. Ann NY Acad Sct 1976;278:455-69.
- 5 Roberts WC, McAllister HA, Ferrans VJ. Sarcoidosis of the heart. A clinicopathological study of 35 necropsy patients (group i) and review of 78 previously described necropsy patients (group ii). Am 7 Med 1977:63:86-108.
- 6 Fleming HA, Bailey SM. Sarcoid heart disease: report of 197 United Kingdom cases with necropsy confirmation in 62. J R Coll Physicians Lond 1981;15:245-53.
- 7 Sekiguchi M, Numao U, Imai M, Furuie T, Mikami R. Clinical and histological profile of sarcoidosis of the heart and acute idiopathic myocarditis. Concepts through a study employing endomyocardial biopsy. I. Sarcoidosis. *Jpn Circ J* 1980;44:249-63.

 8 Fleming HA, Bailey SM. 250 cases of sarcoid heart disease in the United Kingdom: heart block and
- other arrhythmias. In proceedings of tenth international conference on sarcoidosis and other
- granulomatous disorders. Baltimore: 1984. Ann NY Acad Sci (in press).

 9 Fleming HA, Bailey SM. The prognosis of sarcoid heart disease in the United Kingdom. In proceedings of tenth international congress on sarcoidosis and other granulomatous disorders Baltimore 1984. Ann NY Acad Sci (in press).

Systemic candidiasis in heroin addicts

Systemic infections with Candida albicans or related species present in different ways depending on the site of invasion, the effectiveness of the host's immune response, and the presence of underlying disease. For example, in neutropenic patients C albicans and (in some centres) C tropicalis cause a widely disseminated infection affecting many sites including, particularly in the latter infection, skin and muscle, whereas patients with infections associated with parenteral nutrition may show a different pattern of disease affecting sites such as the eye.³ Specific forms of systemic candidiasis may occur with new therapeutic practices, such as candidal peritonitis in patients receiving chronic ambulatory peritoneal dialysis. And in recent years reports have come from widely separated parts of Europe and Australia describing an apparently new candidal syndrome in heroin addicts. 4-6

Characteristically, the complete syndrome includes an early "septicaemic" phase with a high swinging fever and chills three to eight hours after injection of the drug. This is followed at varying intervals ranging from one day to three weeks by pain in the joints, ribcage, or back, painful folliculitis of the scalp or beard area, and visual disturbances. Bone or joint lesions occur in almost one fifth of those affected, and the most common sign is costal osteochondritis. Erosion of the vertebral bodies has also been reported.8 Folliculitis may occur in many patients; it consists of relatively large pustules or nodules centred on dermal candidal abscesses with secondary invasion of the hair follicle and shaft. In the eye several different abnormalities have been described, ranging from vitreous abscesses and retinal deposits to hypopyon and conjunctivitis.6 The clinical features of the complete syndrome are distinctive, but individual

patients may show only one or more of these components, the most common being the skin or eye lesions. The diagnosis may be confirmed by isolation of candida from infected site \$\overline{\mathbb{g}}\$ but blood cultures are seldom positive in the late stages of the disease.

A further important feature of this syndrome has been the clustering of cases suggesting exposure to a common source Contamination of heroin itself has not been shown to occur—though "brown" heroin was originally suspected and recent work suggests that closely related compounds such as diacetylmorphine inhibit the growth of C albicans (B 1106). Nevertheless, the habit of dissolving some forms of heroin, including the brown variety, in acid solutions such as lemon juice before injection may be relevant. Lemons can support the growth of candida and, even though contamination nated, appear normal. In the Glasgow outbreak the source appeared to be several plastic lemons that had been kep? beyond the expiry date and contained juice contaminate with candida (p 1106).6 While this seems the most promising hypothesis to date, some aspects of the problem remain puzzling—such as the fact that heating the lemon juice to dissolve the drug, a common practice, is likely to destroy the organism.

Nor do we know why the injection of candida shoul₩ produce this particular syndrome. Endophthalmitis mag occur after colonisation of intravenous lines by candida ig patients without overt immune defects, and even though various immunological abnormalities have been detected in intravenous drug abusers' there is no evidence that they are implicated in the pathogenesis of this syndrome. Nor immunocompromised guinea pigs injected intravenously with heavy inocula of candida develop similar skin lesions.

The results of treatment of this syndrome are usually good, though retinal lesions may cause permanent visua loss. Successes have been recorded with a range of different drug regimens, including amphotericin B, flucytosine, an& ketoconazole. The use of oral flucytosine in addition to one of the other antifugals seems logical in view of its superior penetration of the eye." Cure of candidal endophthalmitis is by no means assured, however, and alternative methods such as vitrectomy or intraocular injection of amphotericin B may be needed. The ideal approach to management of this condition is prevention, and it may be possible to limit the use of potentially contaminated additives by judicious coun selling. Certainly the dangers of injecting lemon juice shoul be made known to addicts themselves.

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1 Myerowitz RL, Ouzin GJ, Allen GM. Disseminated candidiasis. Changes in incidence underlying diseases and pathology. Am J Clin Pathol 1977;68:29-38.

2 Jarowski CI, Fialk MA, Murray HW, et al. Fever, rash and muscle tenderness. A distinctive clinical presentation of disseminated candidiasis. Arch Intern Med 1978;138:544-6.

3 Henderson DK, Edwards JE, Montgomerie JZ. Hematogenous candida endophthalmiris on patients are:

3 Henderson DK, Edwards JE, Montgomerie JZ. Hematogenous candida endophthalmitis patients receiving parenteral hyperalimentation fluids. 7 Infect Dis 1981:143:655-61.
 4 Mellinger M, de Beauchamp O, Gallien C, Ingold R, Taboada MJ. Epidemiological and clinic approach to the study of candidiasis caused by Candida albicans in the Paris region. Analysis of

- 35 observations. Bull Narc 1982;34:61-81.

 5 Collignon PJ, Sorrell TC. Disseminated candidiasis. Evidence of a distinctive syndrome in herogabusers. Br Med J 1983;287:861-2.
- abusers. Br Med J 1983;287:861-2.
 6 Servant JB, Dutton GN, Ong-Tone L, Barrie T, Davey C. Candidal endophthalmitis
 Glaswegian heroin addicts: report of an epidemic. Trans Ophthalmol Soc UK 1985;104:297-306
 7 Dupont B, Drouhet E. Cutaneous, ocular and osteoarticular candidiasis in heroin addicts: newclinical and therapeutic aspects in 38 patients. J Infect Dis 1985;152:577-91.
- clinical and therapeutic aspects in 38 patients. J Infect Dts 1985;152:577-91.

 8 Newton-John HF, Wise K, Looke DFM. The role of lemon juice in disseminated candidiasis of heroin abusers. Med J Aust 1983;140:780-1.
- 9 McDonough RJ, Madden JJ, Falek A, et al. Alteration of T and null lymphocyte frequencies in the peripheral blood of human opiate addicts: in vivo evidence for opiate receptor sites on A lymphocytes. J Immunol 1980;125:2539-43.
- 10 Van Cutsem J. The antifungal activity of ketoconazole. Am 7 Med 1983;74:9-15
- Edwards JE. Candida endophthalmitis. In: Bodey GP, Fainstein V, eds. Candidiasis. New York Raven Press, 1985:211-25

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