to the young in Britain is already curtailed, but little evidence exists to show that this is particularly effective.

The main message that emerges from looking at problems with alcohol among women and the young is that they are not really very different from those among the whole population. More alcohol is being consumed and more problems are resulting in all sections of the population, and strategies for responding to this problem must be much the same for all those sections.

This is the sixth in a series of articles on alcohol.

References

1 Special Committee of the Royal College of Psychiatrists. *Alcohol and alcoholism.* London: Tavistock Publications, 1979.
2 Ferrence RG. Sex differences in the prevalence of problem drinking. In: Kalant OJ, ed. *Alcohol and drug problems in women.* New York and London: Plenum Press, 1980.
3 Shaw S. The causes of increasing drinking problems among women. In: Camberwell Council on Alcoholism. *Women and alcohol.* London: Tavistock Publications, 1980.
4 Sclare AB. The fetal alcohol syndrome. In: Camberwell Council on Alcoholism. *Women and alcohol.* London: Tavistock Publications, 1980.
5 Lemoine P, Harousseau H, Borteyru JP, Menuet JC. Children of alcoholic parents: anomalies observed in 127 cases. *Quest Medical* 1968;**25**: 476-82.
6 Clarren SK, Smith DW. The fetal alcohol syndrome. *N Engl J Med* 1978; **298**:1063-7.
7 Rosett HL. A clinical perspective of the fetal alcohol syndrome. *Alcoholism: Clinical and Experimental Research* 1980;**4**:119-22.
8 Rosett HL, Weiner L, Zuckerman B, McKinlay S, Edelin KC. Reduction of alcohol consumption during pregnancy with benefits to the newborn. *Alcoholism: Clinical and Experimental Research* 1980;**4**:178-84.
9 Anonymous. Surgeon General's advisory on alcohol and pregnancy. *FDA Drug Bulletin* 1981;**11**:9-10.
10 Little RE. Moderate alcohol use during pregnancy and decreased infant birth rate. *Am J Public Health* 1977;**67**:1154-6.
11 Kline J, Shrout P, Stein Z, Susser M, Warburton D. Drinking during pregnancy and spontaneous abortion. *Lancet* 1980;ii:176-80.
12 Harlap S, Shiono PH. Alcohol, smoking and incidence of spontaneous abortions in first and second trimester. *Lancet* 1980;ii:173-6.
13 Streissguth AP, Barr HM, Martin DC, Herman CS. Effects of maternal alcohol, nicotine and caffeine use during pregnancy on infant neural and motor development at 8 months. *Alcoholism: Clinical and Experimental Research* 1980;**4**:152-64.
14 Jahoda G, Cramond J. *Children and alcohol.* London: HMSO, 1972.
15 Davies J, Stacey B. *Teenagers and alcohol.* London: HMSO, 1972.
16 Aitken PP. *Ten-to-fourteen-year-olds and alcohol.* London: HMSO, 1978.
17 Hawker A. *Adolescents and alcohol.* London: Edsall and Co, 1978.
18 Smart RG. *The new drinkers: teenage use and abuse of alcohol.* Toronto: Addiction Research Foundation, 1980.
19 Smart RG, Gray G, Bennett C. Predictors of drinking and signs of heavy drinking among high school students. *International Journal of Studies on Addiction* 1978;**13**:1079-94.
20 Plant MA, Peck DF, Stuart R. Self-reported drinking habits and alcohol-related consequences among a cohort of Scottish teenagers. *Br J Addict* (in press).
21 Fillmore KM. Drinking and problem drinking in early adulthood and middle age: an exploratory 20-year follow-up study. *Quarterly Journal of Studies on Alcohol* 1974;**35**:819-40.
22 Annis HM, Liban CB. Alcoholism in women: treatment modalities and outcomes. In: Kalant OJ. *Alcohol and drug problems in women.* New York and London: Plenum Press, 1980.
23 Tuchman E. Rehabilitation of alcoholics at Kalksburg (Austria). *Br J Addict* 1965;**61**:59-70.

The Six Diseases of WHO

Human trypanosomiasis in Africa

J R FOULKES

The penalties of having vast tracts of land in Africa denied to man and his domestic animals because of the various species of the tsetse fly are incalculable. These flies of the genus *Glossina* have been described as the "bane of Africa" since the trypanosomes that they host effectively exclude man and his animals from one quarter of the continent. It has been estimated that without *Glossina* the cattle population could double. Apart from the staggering economic implications to the continent's agriculture the trypanosome also causes human sleeping sickness.

Any general discussion of African trypanosomiasis must start with the realisation that there really are two fairly consistent clinical types of sleeping sickness: West African or Gambian and East African or Rhodesian. The West African disease is largely transmitted by tsetse flies of the *Glossina palpalis* group that feed along the rivers and harbour *Trypanosoma* (*Trypanozoon*) *gambiense*. The East African disease is largely transmitted by tsetse flies of the *G morsitans* group that inhabit the savannah country and host *T* (*T*) *rhodesiense*. Table I outlines some of the major differences between the two forms and illustrates the fallacy of thinking about African trypanosomiasis as a single entity. The sharp lines of distinction in the chart are only generally true, and there are many exceptions. For instance, there is a geographical overlapping of the two forms of the disease on the shores of Lake Victoria, and cases of Rhodesian sleeping sickness have on occasion been transmitted by *G palpalis*. Even the separation between the species of the trypanosome is debatable, and some workers, such as Ormerod,[1] conclude that *T* (*T*) *gambiense*, *T* (*T*) *rhodesiense*, and even *T* (*T*)

TABLE I—*Differences between Gambian and Rhodesian disease*

Gambian (West African, *T* (*T*) *gambiense*)	Rhodesian (East African, *T* (*T*) *rhodesiense*)
Chronic—university student in England who developed symptoms two years after leaving Ghana	Acute—progresses rapidly to death within a few months—almost universally fatal if untreated
Man—fly—man cycle	Wild game—fly—wild game cycle with man only a temporary interloper
Healthy carriers common	Healthy carriers uncommon
Transmitted largely by *G palpalis* group of riverine tsetse flies	Transmitted largely by *G morsitans* group of savannah tsetse flies
Infected flies are close to villages—many women and children infected	Occupational hazard to hunters and fishermen. Very few women and children infected (except in an epidemic)
Lymphadenopathy a prominent feature, and node aspiration highly productive in making the diagnosis	Lymphadenopathy much less prominent and node aspiration less productive than blood slide in making diagnosis
Prophylactic injections valuable	Prophylactic injections completely unreliable

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brucei (traditionally an animal-only species) are not distinguishable as distinct species. I propose to concentrate on the Rhodesian form.

Presenting signs and symptoms

The typical presenting picture seen in Zambia is of a moderately debilitated man who admits to having hunted in a high-risk area two to four months earlier. Because he lives on the periphery of an endemic zone it is assumed that he has acquired a partial immunity and therefore experiences surprisingly few symptoms in the first stage of the disease while the parasites are limited to his haemolymphatic system. He does not seek medical help at this time. It is several months after the bite of an infected tsetse fly before the trypanosomes begin to penetrate the blood-brain barrier and cause symptoms in the central nervous system (second-stage disease). It is only after developing a headache, intermittent fever, some lethargy, and possibly a tremor that he makes the long trek to a hospital. The most common presenting signs and symptoms of 685 patients seen in Zambia are compiled in table II. Notice that the rather non-specific symptoms and signs of headache, cyclic fever, dizziness, debility, and anaemia are the most common. The signs and symptoms in the central nervous system of tremor, dullness, change of gait, slurred speech, and coma are less common but point much more clearly to the diagnosis of sleeping sickness. For the 12% who present in semi-coma the prognosis is very poor, and most deaths occur in this group.

TABLE II—*Presenting signs and symptoms (%) of 685 patients seen in Zambia*

Headache	66	Splenomegaly (apparently a higher incidence of malaria in E Province)	12–40
Lymphadenopathy (small shotty nodes not counted in N-W Province)	20–80	Hepatomegaly	33
Fever	31–54	Change in gait	18
Dizziness	50	Oedema	30
Debility	52	Comatose or semi-comatose	12
Anaemia (< 10 g)	48	Diarrhoea	6
Tremor	14–44	Slurred speech	4
Dull and lethargic	40	Jaundice	1–2
Hand-chin reflex (only occasionally elicited in N-W Province)	40	Chancre—seen only once—in a non-Zambian	

Compiled from patients seen in the Eastern Province[2] and North-western Province.[3] When a considerable discrepency occurs both figures are given.

The disease is quite different for those who are not resident on the periphery of an endemic zone and who totally lack immunity. About eight to 10 days after the bite of an infected tsetse fly the trypanosomes have multiplied enough to cause symptoms that are indistinguishable from acute malaria. Often the possibility of trypanosomiasis is not thought of until the high fever and rigors are still present 24 hours after a loading dose of chloroquine. A high level of parasitaemia exists by this time, and the thick blood slide would be teeming with trypanosomes. A further clue could be the presence of a trypanosomal chancre, which appears about one week after the bite of an infected fly—at the very site where the trypanosomes were injected. When this is present it is helpful in establishing an early diagnosis since the chancre is characteristic in appearance, and the trypanosomes may be found in the chancre several days before they are detectable in the blood. The chancre is rare among the partially immune and has never been detected in the more than 1000 cases diagnosed in the North-western Province of Zambia.

Diagnosis

The diagnosis is completely dependent on identifying the parasite. Certain screening tests, such as IgM determination on serum and cerebrospinal fluid, and the fluorescence antibody test help to raise the level of suspicion, but the toxic drugs required for treatment are not used until the diagnosis is confirmed parasitologically. The thick blood slide is the most helpful means of identifying *T rhodesiense*. In West Africa, however, node aspiration is generally considered to be better (table II). If the parasitaemia is scanty a blood concentration method is required. The microhaematocrit centrifuge tube technique of Woo[4] is the most suitable for rural hospitals. Centrifugation of filtrates of blood passed through miniature columns of anion-exchange diethylaminoethanol-cellulose is the most efficient of available techniques for the direct demonstration of trypanosomes. This method is not yet fully adapted for use in rural centres and is, at present, suitable only for urban hospitals. Animal inoculation is a very helpful technique—using citrated blood or cerebrospinal fluid, or both, from a human suspect and injecting it intraperitoneally into a white mouse or a white rat. The white mouse is extremely susceptible to trypanosomal attack, and some of the virulent strains in Zambia multiply fast enough to be found in tail blood in just three days after inoculation. In late-stage disease the parasites are scanty in the peripheral blood but are found in greater numbers in the cerebrospinal fluid. A search in the counting chamber shows them darting around in the spinal fluid with their typical rapid movement.

Treatment

It is lamentable that the only reliable trypanocidal drug for eradicating the parasite from the central nervous system is arsenic, which is a very toxic compound and holds many pitfalls for the unwary. The trivalent arsenical melarsen, which in combination with British antilewisite forms melarsoprol (Mel-B), is the drug of choice for meningoencephalitic disease. It is responsible for a cure rate of about 90% in most institutions, and most deaths occur in tardy patients, who, on admission, are unable to walk and who are quite possibly already untreatable. A further unique feature of treating second-stage sleeping sickness is that it is necessary when treating virulent strains to start the initial dose at very low levels. For all but the most fit patients this is one-tenth of the full dosage. This is slowly worked up to full dosage by the ninth injection (out of a total of 12 intravenous injections over four weeks). The danger of causing drug resistance by low initial dosages is largely hypothetical and must be balanced by the unacceptable mortality of large initial doses given to the severely ill. The preferred course was described by Robertson.[5]

Pretreatment with a non-arsenical drug is essential in advanced cases. The toxic effects of using arsenic on a fresh case make it mandatory to eliminate first the parasites from the haemolympathic system with a non-arsenical compound. The choice here is between suramin (Antrypol) and berenil. Both drugs are quite capable of eradicating the parasites outside the blood-brain barrier. A further requirement before giving melarsoprol is to correct severe anaemia by transfusion. Patients who are moribund on admission have a better chance of survival if corticosteroids are started initially and used concomitantly with melarsoprol.[6]

The treatment of first-stage disease (haemolymphatic) is much less complicated and dangerous; it may be cured with 1 g of suramin given intravenously on days 1, 3, 7, 14, and 21.

Special problems contributing to the lack of progress in controlling trypanosomiasis

Several features about the geography and epidemiology of sleeping sickness contribute to the slow advances in diminishing the disease and its consequences. It would be just as dangerous to walk across the Luangwa Valley in Eastern Zambia today as it was in 1909, when a prospector named Armstrong trekked along that route, contracted sleeping sickness, and thereby contributed

the name *rhodesiense* to the "new" strain of trypanosome found in his blood. Why hasn't there been more progress?

Geography—Certainly the East African disease is principally a threat in remote areas where man chooses to invade the fly country in his pursuit of game, honey, and fish. The people at risk live many miles from medical help and are likely not to be well orientated towards Western medicine. The herbs and potions used by the traditional healers are totally ineffective as trypanocidal agents. The most dangerous part of spending several weeks under the care of a witch doctor is the time that is lost, and a treatable case might be converted into an incurable one.

Small number of human cases—The number of people who actually contract sleeping sickness is quite small compared with the five other diseases under special investigation in the United Nations Development Programme/World Bank/WHO Special Programme on Tropical Diseases. Even though the veterinary and economic repercussions of trypanosomiasis are huge, it is unrealistic to expect a government to become very active about a disease that might affect fewer than 100 of its citizens a year, as it does, for instance, in Botswana, Zimbabwe, Sudan, and Mozambique. The research facilities of the big drug firms are not focused at all on a disease that attacks only about 10 000 people a year and offers such a limited market.

Economics—The vast tracts of land that belong to the tsetse fly make the task of eradication a daunting one. Five years ago, the cost of eradicating *Glossina* from one square kilometre was about $300-500. Astronomical sums would be required to reclaim any appreciable amount of the 10 million square kilometres of Africa that still belong to *Glossina*. The cost of control measures is also punishing. Maintaining fly pickets, tsetse guards, veterinary staff, surveillance teams, and bush clearing are all large items in the budget of less affluent countries. In countries where thousands of children die each year because the medical services are unable to deliver chloroquine to them it is not surprising that control measures for sleeping sickness have low priority.

Politics—It seems almost too obvious to mention, but the medical infrastructure necessary to contain the huge problem of sleeping sickness is dependent on a stable government. In July 1972 I travelled to Tororo, Uganda, to repay a vist to the director of the prestigious East African Trypanosomiasis Research Organisation. It was a great disappointment to learn that he had found it necessary to flee the country after a military coup. In the years that followed not just a research institute but the entire structure of medicine was shaken to its foundations. In 1965 WHO[7] estimated that only 10% of the workers who had previously been engaged in controlling trypanosomiasis and tsetse fly in Africa were still in their posts. It would be unkind to list the countries that 20 years ago had an effective control programme but are no longer able to maintain one. Conversely, it would only be fair to mention that there are countries, such as Zambia, that now have more experts undertaking research and control than ever before. In Zambia this has largely resulted from the WHO-sponsored Tropical Disease Research Centre, whose experts undertake extensive field projects and teaching.

References

1 Ormerod WE. Taxonomy of the sleeping sickness trypanosomes. *J Parasitol* 1967;**53**:824-30.
2 Buyst H. The treatment of T rhodesiense sleeping sickness, with special reference to its physio-pathological and epidemiological basis. *Ann Soc Belg Med Trop* 1975;**55**:95-104.
3 Foulkes JR. Human trypanosomiasis in Zambia. *Med J Zambia* 1970;**4**:167-77.
4 Woo PT. The haematocrit centrifuge technique for the diagnosis of African trypanosomiasis. *Acta Trop* 1970;**27**:384-6.
5 Robertson DH. The treatment of sleeping sickness (mainly due to Trypanosoma rhodesiense) with melarsoprol. *Trans R Soc Trop Med Hyg* 1963;**57**:122-33.
6 Foulkes JR. An evaluation of prednisolone as a routine adjunct to the treatment of T rhodesiense. *J Trop Med Hyg* 1975;**78**:72-4.
7 Anonymous. Problems of trypanosomiasis control in Africa. *WHO Chron* 1965;**19**:152-3.

MATERIA NON MEDICA

Who will buy?

It is a moot point whether one receives more experience of life during the official terms of the medical course, or in the long holidays in between. For me the latter was true, and nothing was more instructive than the time I spent on the markets in the cloth business. My credentials were hardly attractive. "Do you know anything about cloth?" "Do you know the different types of tapestries?" "Are you familiar with the varieties of ladies' dress material?" "Curtain materials?" "Handkerchiefs?" "Are you physically strong and can you drive large trucks?" To all those pertinent questions the answer was no. "Can you talk to a large crowd and hold their interest?" I swallowed to hide my terror and stammered that I was sure I could easily learn to do so. Perhaps out of pity for my impecunious state, they took me on.

The origins of markets reach back into history to the Phoenicians and Arabs, who shipped food and goods to different trading centres. China had developed chains of trading centres some hundreds of years BC. Roman and Anglo-Saxon Britain saw the development of markets around London; the *Institutae Londoniae* in 1000 AD mentions Billingsgate and its dealing with timber, wine, and fish. Fairs enabled the peasants and cultivators to meet and barter goods, and later some became regular fixtures. During the Renaissance many markets became regular shopping districts. We visited northern cities and towns such as Bolton, Oldham, Bury, the port of Fleetwood, and Bakewell in Cheshire on bank holidays, all of which have thriving markets.

My arrival was hardly auspicious; half asleep from the exertion of helping to load the huge vans in the early hours of the morning, I promptly stood on and broke in half a yardstick and tripped over a bale of cloth. The regular staff hastily put me at the back of the stall to keep me out of harm's way, with instructions to observe and learn but do nothing. Later on I was allowed to measure a few yards of cloth with the yardstick, but this seemed to have a life of its own and took every opportunity to leap out of my hand. The first cloth that I ever cut was so crooked that, amid the barely suppressed laughter of the customers and the staff, it had to be discarded.

Stall owners buy large quantities of materials from various warehouses and are therefore able to sell the goods more cheaply than the shops. Customers know that they can thus save money but they do expect a performance from the trader as well. I used to watch the experienced "pitchers," as they were called. They would stand on a dais, and to no one in particular start a monologue about the history and merits of the goods they wished to sell. A crowd would soon gather. After some ribald humorous exchanges with the women in the audience, they would offer the article at a low price, but above the asking price. Inevitably somebody would offer the money. "As you are such a sport," the pitcher would say, "I will treat you." He would then lower the price further, but still above the asking price. More of the audience would then offer money. "Go on, let's give it all away for nothing," he would say, recklessly, and offer it at the intended price. By now most of the audience would be fighting to buy. It was at this point, when I had learnt the trade, that I would start an argument with him, complaining that he had gone mad, and was giving it all away too cheaply. The crowd all knew it was an act, and we knew they knew, but it was nevertheless great fun and an integral part of the whole performance.

I left the markets with an insight into crowd psychology and an ability to speak in front of large audiences without fear or embarrassment.—R E GOODMAN, general practitioner, Northenden, Manchester.