trypanosomes. Serial specimens should be examined as parasitaemia levels may vary. Concentration methods increase the chances for detection (quantitative buffy coat, Becton-Dickinson, NJ). Treatment decisions should be based on demonstration of the parasite since serological assays have variable sensitivity and specificity.

In vivo inoculation of mice distinguishes between EAT and WAT as *T. b. rhodesiense* but not *T. b. gambiense* trypanosomes will be detected.

The diagnosis of WAT in our first patient was surprising, as he had not travelled to Western endemic areas. This raises the possibility of cross-migration of tsetse fly species to other areas.

A lumbar puncture is mandatory after parasite clearance of peripheral blood to exclude stage II (neurological) disease and to individualise treatment. Suramin and pentamidine do not penetrate the central nervous system (CNS) adequately and are only used for the haemolymphatic stage.

The arsenic-based drug melarsoprol is currently the only effective treatment for second-stage EAT but causes an encephalopathic syndrome in 5 - 10% of patients, with a case fatality rate of 50%, and resistance has also been described. Efflornithine should be used in WAT patients with CNS disease.

Development of treatment options is limited. Nifurtimox, a drug used to treat Chagas disease, may be effective, but has not yet been validated for use. Oral eflornithine, the diamidine prodrug DB 289, and the pre-clinical development of megazol seem promising.