March last year. Several research teams have been busy in the oil polluted waters and on the beaches stretching from the spill site itself to Kodiak Island 300 miles south in the Gulf of Alaska. But the investigators scarcely talk to each other and they are certainly not being allowed to publish their results. No journals or symposia will hear of these data until well into the 1990s, if ever. Instead, lawyers and economists are beginning to use the findings to assess the cash costs of both the initial damage and the subsequent restoration work. The United States and Alaskan governments are expected to present Exxon with a claim for up to \$2 billion—which, in turn, the company will contest on the basis of results compiled by its scientists, lawyers, and economists. Meanwhile, even this remarkable opportunity for peer review and quality control by parallel study is being wasted.

"Under ordinary circumstances, one team of scientists chancing upon another in the remote Alaskan wilds would exchange greetings, compare notes, and trade tales of methodology and findings," writes Mark Holman Turner.¹ But in today's litigious circumstances these things do not happen. Although craft carrying "rival" research teams pass each other and occasionally land on the same stretch of blackened

beach to conduct analyses and take samples, "biologists from different boats measure their words carefully. The legal aftermath of the spill has forced the likes of Stephen Maclean, of the University of Alaska's Institute of Arctic Biology, to maintain distance from scientist colleagues working for Exxon. 'We know they're out there,' Maclean says. 'But we have been told by the [government] lawyers that we are not to cross paths in any substantive way.'"

The practical repercussions of severe restrictions in publication are bound to include substantial delays in other parties learning lessons from the *Exxon Valdez* incident and applying that knowledge in dealing with spills elsewhere in future. There has also been unnecessary and costly duplication of effort between the 300 scientists working for the *Exxon Valdez*'s owners and a comparable number ranged against them. But one of the saddest consequences, surely, is the divisiveness which the legal process has introduced among researchers engaged on what both sides would no doubt like to see as a common goal of environmental protection and improvement.

1 Holman Turner M. Oil spill: legal strategies block ecology communications. *Bioscience* 1990;40:238-42.

Letter from Brasilia

Cutaneous leishmaniasis

P D Marsden

Leishmaniacs, as we call them, for they are so enthusiastic about this fascinating disease, are the world experts in the use of the heavy metals—the mainstay of nineteenth century treatment. More use is made of pentavalent antimonial drugs to treat leishmaniasis than of all the other heavy metal treatments combined, including arsenical drugs in third stage African sleeping sickness, gold in rheumatoid arthritis, and platinum in testicular tumours. Yet this is nothing to be proud of as the treatment of leishmaniasis has not changed for 40 years. I reviewed antimonial treatment a few years ago when I realised that I probably would not use anything else. Only two pentavalent antimonials are available: Pentostam (Wellcome) comes in a 100 ml flask containing 100 mg antimony/ml, which when I last asked cost £72 in England, and Glucantime (Rhodia, Specia, and Rhone Poulenc) comes in a 5 ml ampoule (85 mg/ml) and costs the Brazilian government about US\$1 per ampoule. Although the government bought well over 1 million ampoules in 1989, it is not even beginning to meet the problem. Only the Brazilian and Venezuelan governments in South America supply the drug to practitioners free. Infected patients cannot afford the drugs, and this is one reason why development of new treatments is so slow—there's no market. The companies tried to stop manufacture of their respective pentavalent antimonial compound, presumably because they were not making much profit. In India and China the governments make their own drug, but the one attempt to do this in Brazil failed because the product was too toxic.

Pentavalent antimonial drugs are the recommended treatment for all forms of leishmaniasis, but I will consider here the cutaneous form. It is essential that doctors know the types of cutaneous leishmaniasis present in their area so that they can manage patients effectively. In Brasilia more than 90% of our patients are infected with *Leishmania viannia braziliensis* so we

treat all patients with maximal antimony treatment to promote early healing and avoid metastasis. Occasionally we see patients from the Amazon region with *L viannia guyanensis* infection, which causes clinically distinct skin lesions. In *L viannia braziliensis* infections ulcers are deep, usually single, and slow to heal even with treatment, whereas with *L viannia guyanensis* they are superficial, small, and often multiple. Distribution of the guyanensis subspecies is said to be restricted to that of its animal reservoir, the two toed sloth (*Choloepus didactylus*), which is Amazonian. Our laboratory has also identified *L mexicana amazonensis* infections, but these are rare because the sandfly vector, *Lutzomyia flaviscutellata*, rarely bites man and the infection is maintained in rodents. When this infection occurs in man it usually produces a limited skin granuloma, but it is also responsible for diffuse anergic leishmaniasis (Convits' syndrome), which results in massive skin invasion with multiple nodules resembling lepromatous leprosy. In 15 years we have never seen Convits' syndrome in Brasilia or in our field area in Tres Bracos Bahia but there are many reports from other parts of Brazil. Why only this subspecies of leishmania seems to produce this syndrome requires investigation.

Identification and diagnosis

We have worked hard to develop a reliable laboratory that can identify the subspecies associated with our clinical work. Most clinicians do not have such a facility, but the World Health Organisation has set up a network of reference laboratories, and a taxonomic study can be arranged through one of these. Identification is laborious, particularly with *L viannia braziliensis*, which is difficult to culture and requires sophisticated technology such as isoenzyme analysis, monoclonal antibody reactions, and evaluation of kinetoplastic

Núcleo de Medicina Tropical, Universidade de Brasilia-CP 153121, 70.010 Brasilia DF, Brazil
P D Marsden, MD, professor

Br Med J 1990;300:1716-7

Choloepus didactylus

ARDEA LONDON, KEITH AND LIZ LAIDLIER

DNA. Leishmanias, like trypanosomes, have kinetoplasts (independent structures containing DNA in the protozoal cell), which can conveniently be separated and studied.

With experience a clinician can diagnose cutaneous leishmaniasis fairly confidently by clinical examination alone. Patients have a skin granuloma caused by leishmanial amastigotes that are restricted to the skin. Only in special circumstances such as diffuse anergic leishmaniasis and post-kala-azar dermal leishmaniasis do the amastigotes metastasise to distant locations. This tropism by leishmania and the different behaviour

of the visceral and cutaneous species still await clarification. Turning again to our experience in Tres Bracos Bahia most of our patients with *L. viannia braziliensis* infections present with an ulcer (rapid necrosis is a feature of the histology). This ulcer often has an infiltrated erythematous rolled edge typical of the leishmanial ulcer. The border is sterilised and aspirated, and smears and cultures are examined for leishmania with Giemsa. We also obtain a punch biopsy specimen from this region for histological examination, and tissue is ground up for inoculation into hamsters. The hamster, which was introduced by Saul Adler of Jerusalem, has been a great help in unravelling the mysteries of *L. viannia braziliensis* because of its susceptibility to leishmanial infections. Immunological tests are used in conjunction with these methods to strengthen the clinical diagnosis. The Montenegro or leishmanin test is the most reliable skin test in diseases caused by parasites; and 0.1 ml of a concentrate of killed promastigotes is inoculated into the skin of the volar surface of the forearm. The result is regarded as positive if, after 48 hours, a papule with a diameter greater than 5 mm is present, a criterion similar to that used in the Mantoux test and indicative of delayed hypersensitivity. Enzyme linked immunosorbent assay (ELISA) is also useful, although leishmanias cross react with *Trypanosoma cruzi*. Fortunately, Chagas' disease is not present in Tres Bracos. We also use serological tests as one of the criteria to evaluate cure as titres usually fall to negative values within a year after the skin ulcer has scarred. A few patients have persistent titres on indirect fluorescence antibody testing, suggesting persistence of the parasite, and we are currently investigating this problem.

ANY QUESTIONS

A woman in her 40s has bilateral deafness resulting from an attack of rubella when a child. Might she benefit from an electronic cochlear implant?

Potential candidates for a cochlear implant must have a hearing loss greater than 90 dB at any audiometric frequency. They should be unable to gain any recognition of speech when using a powerful hearing aid without lip reading. There must be no active infection in the ear or upper respiratory tract, and the patient must be fit enough to undergo a four hour operation. For an intracochlear implant the cochlear duct should have been proved patent by radiological investigation. If there is any suspicion of central damage (such as infarction of the auditory cortex) computed tomography of the brain should be performed to exclude patients who have brain disease.¹

Patients should be advised what a cochlear implant could achieve for them and should also understand that they may not be able to understand any speech at all through the device. Postoperative rehabilitation requires long and repeated visits to the audiology department, so patients must be highly motivated and committed to the venture.²

When hearing is lost before the age of 6 recognisable speech is usually lost as well. If the hearing is lost between the ages of 6 and 12 there are gross errors in speech, but if hearing is lost after 12 speech is usually easily intelligible. People who have become deaf after learning to speak tend to have the best results from a cochlear implant. Adults who are deaf prelingually and those who are congenitally deaf have a high rate of failure after receiving implants. The functional development of the central auditory pathways and auditory cortex is unknown in these patients. Furthermore, they have grave difficulties in understanding sound and therefore gain only limited information from an implant even after extensive rehabilitation.

World wide, most implants have been provided for adults who are postlingually deaf and hence little is known about their usefulness in people whose deafness is prelingually acquired and congenital in origin.—M HAWTHORNE, consultant otolaryngologist, Middlesbrough

1 Gibson WPR. Cochlear implants. In: Kerr AG, Booth JB, eds. *Scott Brown's otolaryngology*. Vol 5. London: Butterworths, 1987:602-18.

2 Fraser JG. Selection of patients. In: Gray RF, ed. *Cochlear implants*. London: Croom Helm, 1985:124-39.

"Cunning alewives put the ashes of deadmen's bones in their ale to make it intoxicating" (Aubrey's "Brief Lives"). Is there any pharmacological basis for this belief?

Despite searching through numerous books of folklore I have been unable to find an answer. Bones and bone ash, both animal and human, have been used since the beginning of time to ward off evil spirits but it is difficult to see how they could have intoxicating properties. Many things have been added to improve the quality of beer. Calcium carbonate in various forms was used as a neutraliser; finings such as isinglass, fishy matter (sole skins), and hartshorn were popular, and it is possible that boiled bones could have produced a glutinous substance for this purpose. Stimulating matters are also described and included liquorice, grains of paradise, and guinea pepper; small animals such as mice, weasles, rats, and newts (some say the origin of "pissed as a newt") were added to improve the flavour and strength of beer and cider. A historical and technical account of this fascinating and complex subject can be found in J Patton's *Additives, Adulterants and Contaminants in Beer*.—ALEX PATON, honorary consultant physician, Oxford

Is there any evidence that the use of thiazides in young women for water retention predisposes to diabetes in later life? How do they compare with spironolactone?

Thiazide diuretics can impair diabetic control in patients with established diabetes and make latent diabetes manifest. There is also evidence that long term use in non-diabetic subjects can increase the fasting blood glucose concentration. This has been studied mainly in patients with hypertension who are being treated with diuretics over a long time. The data are somewhat conflicting, but the effect seems to be small in patients who start with normal glucose tolerance.

I know of no studies of the development of diabetes in later life in women who received diuretics when younger. But I think that any effect on blood glucose at the time would be reversed when the diuretic is stopped, and it is unlikely that the risk of developing diabetes would be increased in women with pre-existing normal glucose tolerance. Spironolactone does not impair glucose tolerance.—LINDA BEELEY, director, Drug and Therapeutics Unit, Birmingham

Dukes MNG. *Diuretics. Meyler's side effects of drugs*. 11th ed. Amsterdam: Elsevier, 1988:419-20.