Spontaneous sleep for 30 minutes during standard electroencephalography was attended by frequent snores; no apnoeic spells occurred.

After 10 weeks of thyroid supplement, the facial appearance resembled that in a photograph taken 18 months earlier and the tongue had shrunk to average dimensions. Snoring continued for a further 1 month, ceasing abruptly 10 days after thyroxine had been increased from 50 to 75 μg daily. Neurological symptoms and signs disappeared *pari passu*,[3] none being present 4 months post-treatment. Radiography showed that the post-nasal space now had a typical configuration.

Snoring signifies incomplete obstruction of the upper airway.[1] Factors predisposing to a critical closing pressure during sleep include altered ventilatory control, a small oropharyngeal lumen and an increase in pharyngeal compliance.[4] Each of these circumstances attends the hypothyroid state.

While the tongue and pharynx are both sites where mucinous oedema is apt to occur,[3,5] genioglossus enlargement has particular significance as this muscle is the key element opposing physiological pharyngeal closure. Its contractile properties, moreover, are deranged as well.[5] Decreased sensitivity of respiratory neurones both to hypoxia and to hypercapnoea has been amply demonstrated in hypothyroidism.[7–9] Hypotheses include a reset chemostat secondary to chronic alveolar hypoventilation[8] and central effects of reduced body temperature and metabolic rate.[6,9]

Apnoea is the counterpart to snoring when upper airway obstruction becomes complete. Episodes of obstructive sleep apnoea (OSA) are common in untreated hypothyroidism and, furthermore, moderate with thyroid supplement.[10] Analogous mechananisms may be invoked when seeking to explain cessation of snoring in the present case.

A reduction in body weight can be readily dismissed, since improvement of OSA has been observed in its absence.[6,10] Besides, our patient was not obese. Concomitant resolution of myxoedema[5] has obvious attractions. However, in at least 2 patients with OSA whose apnoeic episodes significantly decreased when euthyroid, the upper airway and neck pre-treatment were reputedly normal.[6,11] Furthermore, in the present case snoring persisted beyond the time physical stigmata of myxoedema had disappeared. Normalizing of respiratory control[6,10] may therefore be specially relevant. The manner in which thyroid hormone influences neural regulatory drive is unclear. Interestingly, elimination of OSA in a patient with hypothyroidism was effected not only by thyroxine but also by medroxyprogesterone which augments inspiratory effort independently of changes in chemical stimuli, metabolic rate or body temperature.[6]

Mechanisms aside, snoring is a predictable complication of hypothyroidism and thyroid function should be routinely evaluated in all habitual snorers.

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References

1. Robin, I.G. Snoring. *Proc R Soc Med* 1968, **61:** 575–582.
2. Norton, P.G. & Dunn, E.V. Snoring as a risk factor for disease: an epidemiological survey. *Br Med J* 1985, **ii:** 630–632.
3. Ingbar, S.H. The thyroid gland. In: Wilson, J.D., Foster, D.W. (eds) *Williams Textbook of Endocrinology*, 7th edition. W.B. Saunders, Philadelphia, 1985, pp. 682–815.
4. Stradling, J.R. & Phillipson, E.A. Breathing disorders during sleep. *Q J Med* 1986, **58:** 3–18.
5. Orr, W.C., Males, J.L. & Imes, N.K. Myxedema and obstructive sleep apnea. *Am J Med* 1981, **70:** 1061–1066.
6. Skatrud, J., Iber, C., Ewart, R., Thomas, G., Rasmussen, H. & Schultze, B. Disordered breathing during sleep in hypothyroidism. *Am Rev Respir Dis* 1981, **124:** 325–329.
7. Wilson, W.R. & Bedell, G.N. The pulmonary abnormalities in myxedema. *J Clin Invest* 1960, **39:** 42–55.
8. Nordqvist, P., Dhuner, K.-G., Stenberg, K. & Orndahl, G. Myxoedema coma and CO_2-retention. *Acta Med Scand* 1960, **166:** 189–194.
9. Zwillich, C.W., Pierson, D.J., Hofeldt, F.D., Lufkin, E.G. & Weil, J.V. Ventilatory control in myxedema and hypothyroidism. *N Engl J Med* 1975, **292:** 662–665.
10. Rajagopal, K.R., Abbrecht, P.H., Derderian, S.S. *et al.* Obstructive sleep apnea in hypothyrodism. *Ann Int Med* 1984, **101:** 491–494.
11. McNamara, M.E., Southwick, S.M. & Fogel, B.S. Sleep apnea and hypothyroidism presenting as depression in two patients. *J Clin Psychiatry* 1987, **48:** 164–165.

The need for new therapeutic approaches in visceral leishmaniasis during HIV infection

Sir,

Recently, several reports have emphasized the difficulty in diagnosing visceral leishmaniasis during human immunodeficiency virus (HIV) infection because of atypical clinical pictures.[1–3] In this letter, we would like to underline the therapeutic difficulties which are also frequently encountered in this condition.

A 27 year old drug addict positive for HIV-1 antibodies since 1986 and at the generalized lymphadenopathy stage presented weight loss of 5 kg and voluminous hepatosplenomegaly in February 1989. There was no fever, the differential blood cell count was normal and the lymphocyte T4 count was 230/mm^3. Leishmaniasis was diagnosed after hepatic needle biopsy and this was confirmed by an examination of the myelogram while an immunofluorescence serotest was negative. *Leishmania donovani infantum* was isolated and found to be sensitive to the usual drugs *in vitro*. The patient was given two successive courses of treatment with *N*-methylglucamine (60 mg/kg/day for 15 days). This was completely ineffective. *N*-methylglucamine (20 mg/kg/day) was then administered for 20 days in combination with intramuscular injections of gamma-interferon (Laboratoires Roussel-UCLAF, France) 2×10^6 U/m^2/day. The hepatosplenomegaly regressed by half and leishmania were no longer isolated from the myelogram.

The patient was then lost to follow-up due to renewed drug addiction and further imprisonment. In October 1989 he was again admitted to hospital with an irregular fever with peaks at 39°C caused by relapsing leishmaniasis. Serological tests for leishmaniasis were negative

by immunofluorescence though both these sera and the previous sera were found to be positive by Western blot (C. Mary, unpublished data). The *N*-methylglucamine and interferon-gamma combination was reinstituted and this restored a normal body temperature, improved the general status, partially relieved the hepato-splenic tumour syndrome and normalized the myelogram.

More than 50 cases of visceral leishmaniasis have been reported during HIV infection and this parasite seems to have developed an opportunist behaviour in this situation.[4] The use of antimonials and pentamidine often gives disappointing results in HIV-infected patients whereas there are only a few alternative, less effective therapies.[5]

Interferon-gamma was found to be capable of potentiating the anti-leishmanial effect of glucamine both *in vitro* and *in vivo*.[6] This combination induced a partial remission on two occasions in our kala-azar patient in whom a more conventional therapeutic regimen was ineffective. Clinical studies are under way to specify the optimum protocol.[7]

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References

1. Berenguer, J., Moreno, S., Dercenado, E. *et al.* Visceral leishmaniasis in patients infected with the human immunodeficiency virus (HIV). *Ann Intern Med* 1989, **111:** 129–132.
2. Condom, M.J., Clotet, B., Sirera, G. *et al.* Asymptomatic leishmaniasis in the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 1989, **11:** 767–768.
3. Smith, D., Gazzard, B., Lindley, R.P. *et al.* Visceral leishmaniasis (Kala Azar) in a patient with AIDS. *AIDS* 1989, **3:** 41–43.
4. Lafeuillade, A., Gastaut, J.A., Dhiver, C. *et al.* Leishmaniose viscérale et infection par le virus de l'immunodéficience humaine. Place nosologique et problèmes thérapeutiques à propos de trois nouvelles observations avec revue de la littérature. *Sem Hôp Paris* 1990, **66:** 145–149.
5. Dellamonica, P., Bernard, E., Le Fichoux, Y. *et al.* Allopurinol for treatment of visceral leishmaniasis in patients with AIDS. *J Infect Dis* 1989, **160:** 904–905.
6. Murray, H.W., Berman, J.D. & Wright, S.D. Immunochemotherapy for intracellular *Leishmania donovani* infection: gamma-interferon plus pentavalent antimony. *J Infect Dis* 1988, **157:** 973–978.
7. Badaro, R., Falcoff, E. & Badaro, F.S. *et al.* Treatment of visceral leishmaniasis with pentavalent antimony and interferon gamma. *N Engl J Med* 1990, **322:** 16–21.

Pneumococcal bacteraemia: a late complication following endoscopic variceal sclerotherapy

Sir,

A low rate of blood culture positivity after elective endoscopic injection sclerotherapy (EIS) has been reported when blood cultures are obtained 5 and 10 minutes after this procedure.[1] We here report an episode of pneumococcal bacteraemia in a cirrhotic patient occurring 10 hours after an emergency EIS procedure. A similar delay in the appearance of bacteraemia due to other microorganisms may be responsible for an underestimated incidence of bacteraemia following EIS in previous investigations.[2]

A 68 year old male alcohol-associated cirrhotic patient was admitted because of massive haematemesis. Physical examination revealed jaundice and ascites. Endoscopy revealed the presence of oesophageal variceal bleeding and an EIS procedure using an Olympus Q-10 fibroscope was performed. A total of 15 ml of 5% ethanolamine oleate was injected and bleeding was controlled. On the seventh hospital day, haematemesis recurred and a further EIS procedure was performed. Ten hours after sclerotherapy, the patient developed high fever (39°C) and chills. A chest film was clear. Ascitic fluid and urine cultures were negative. Three sets of blood cultures yielded growth of *Streptococcus pneumoniae*. Oropharyngeal exudate was unfortunately not obtained. Cefamandole 1 g every 6 hours, during 7 days, was administered. The symptoms improved after treatment and no overt gastrointestinal bleeding occurred again after sclerotherapy until discharge 2 weeks later.

Studies that have evaluated the incidence of bacteraemia following EIS have shown a marked variability of results, ranging from 5% to 50%.[1-4] On the other hand, although oropharynx or contaminated endoscopes have been implicated as the source of the bacteraemia, most of the microorganisms isolated (*Corynebacterium spp., Staphylococcus epidermis, Bacillus spp.*,[1] corresponded to skin flora and they were not simultaneously isolated from pharyngeal or from endoscopy surveillance cultures. These data are controversial and the role of prophylactic use of antibiotic in this setting is therefore not well defined. Our findings point to the oropharynx as the most probable source of bacteraemia, since there was no evidence of coexisting lung or peritoneal pneumococcal infection in this case.

To the best of our knowledge, delayed pneumococcal bacteraemia following EIS has not been previously reported. The development of bacteraemia later than it is currently recognized, as occurred here, could explain the low rate of bacteraemia found when blood cultures are obtained immediately after an EIS.[1]

Bacteraemia due to *S. pneumoniae* following EIS may be a life-threatening event in the cirrhotic patient, and it could be favoured by the abnormal splenic function present in these patients. Late pneumococcal bacteraemia must be suspected in this setting. Pneumococcal vaccine administration and/or antibiotic prophylaxis in these cases deserves further evaluation.