

adults with upper airway obstruction have been emphasised elsewhere.²

It is also surprising that both patients seemed to have most difficulty on expiration. Narrowing of the supraglottic air space usually leads to restricted inspiration and eventually inspiratory stridor.

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1 Mayo-Smith MF, Hirsch PJ, Wodzinski SF, Schiffam FJ. Acute epiglottitis in adults. *N Engl J Med* 1986;314:1133-9.

2 Gerrish SP, Jones AS, Watson DM, Wight RG. Adult epiglottitis. *Br Med J* 1987;295:1183-4.

AUTHOR'S REPLY.—Messrs Thompson and McClymont are correct in suggesting that urgent assessment regarding intubation is essential in acute upper airway obstruction in adults, and in this infectious diseases hospital the physicians are probably more aware of the implications of delay in diagnosis than many. The two cases described were both seen by consultant physicians who were perceptive enough to realise that the syndrome was unusual and did not fit the pattern of adult epiglottitis. The initial delay was caused by failure of junior staff to recognise the potential importance of the symptoms.

In the case reports there was insufficient space to discuss the odd observation that both patients felt that expiration was more difficult than inspiration, which goes against everything we know about acute upper airway obstruction. It will be interesting to see if further experience with similar cases will clarify the paradox.

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Temporal artery biopsy

I agree with Dr Miles C Allison (15 October, p 933) that to miss the diagnosis of giant cell arteritis may be disastrous. To delay steroid treatment, however, while awaiting the results of a biopsy or to fear that such treatment may obscure the microscopic diagnosis after seven days is equally dangerous and not valid. We reported 30 years ago on two patients with histologically proved giant cell arteritis in whom the early use of steroids did not alter the diagnostic value of an artery biopsy. Indeed, in one of the cases a biopsy specimen taken five weeks after steroids were started still showed intimal thickening and scarring and fragmentation of the internal elastic lamina, characteristic hallmarks of an arteritic process, although the original inflammatory and giant cell reaction had regressed.

The potential catastrophic consequences of giant cell arteritis, such as blindness, emphasise an immediate need to start steroid treatment when there is strong clinical suspicion. Clinicians must be reassured that prompt treatment will not alter the diagnostic yield from a delayed biopsy.

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1 Harrison RJ, Harrison CV, Kopelman H. Giant-cell arteritis with aneurysms. Effect of hormone therapy. *Br Med J* 1955;iii:1593-601.

AUTHOR'S REPLY.—The final sentence of my editorial emphasises that patients with typical clinical features of giant cell arteritis should be treated promptly with corticosteroids without waiting for confirmation from the results of temporal artery biopsy. Emergency biopsy is not usually

feasible so the patient should receive corticosteroids while arrangements are being made for the procedure to be done. There is insufficient information relating the diagnostic yield of biopsy of the temporal artery to the duration of preceding corticosteroid treatment because patients responding to treatment are not normally subjected to a repeat biopsy. In our retrospective study of 132 patients undergoing temporal artery biopsy the incidence of a positive result was 60% if the procedure was performed during the first week of treatment and 20% if biopsy was delayed beyond one week.¹ Only two of the 20 biopsy specimens in the second group manifested the change of healed arteritis discussed by Dr Kopelman. Many of the biopsies had originally been reported as showing changes consistent with treated arteritis. On review, however, most of these specimens displayed changes that were more in keeping with arteriosclerosis than the features of healed arteritis described by Lie *et al.*²

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1 Allison MC, Gallagher PJ. Temporal artery biopsy and corticosteroid treatment. *Ann Rheum Dis* 1984;43:416-7.

2 Lie JT, Brown AL, Carter ET. Spectrum of aging changes in temporal arteries. Its significance in interpretation of biopsy of the temporal artery. *Archives of Pathology* 1970;90:278-85.

Living under pylons

The Channel 4 television programme to which Dr Neville Goodman objects (5 November, p 1197) was first broadcast in 1984 and is now out of date.

The State of New York Public Services Commission has repeated its instruction to the New York Power Authority to bar residential development within 175 feet (53.3 m) of the centre of a 765 kV line.¹ Other states have either imposed restrictions on residential building beneath or close to high voltage power lines or are drafting legislation to do so.² In the Soviet Union "sanitary protective zones" based on electrical field measurements have been established to protect the public from power line fields.³ For example, domestic buildings may be located in the protective zones of 330-550 kV lines only if steps are taken to decrease the fields to recommended safe levels—that is, 0.5 kV/m inside houses and 1.0 kV/m—around houses.

The International Radiation Protection Association is revising downwards its limits of acceptable exposure to both electrical and magnetic fields at power frequencies.⁴ Such measures may be unpalatable to the electric industry and other interests but the reasoning behind them should not be likened to a mediaeval witch hunt. Quite recently one of your own columnists was of the same opinion.⁵

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1 Anonymous. New York Public Services Commission supports 1.6 kV/m and 100 mG limits at right of way. *Microwave News* 1988 Mar-Apr:5.

2 Anonymous. Florida power lines siting rules. States seek power line rules in face of federal inaction. *Microwave News* 1988 May-June:3-5.

3 Shandala MG, Dumanskiy Yu D, Bezdol'naya IS. The biological effects of power frequency electric fields in the environment. In: Marino AA. *Modern bioelectricity*. New York: Marcel Dekker Inc, 1988:927-63.

4 Anonymous. Power line talk. *Microwave News* 1988 July-Aug:8.

5 Dixon B. Scientifically speaking. *Br Med J* 1988;296:940.

When the programme *The Good, the Bad and the Indefensible* was first broadcast by Central Television many months ago I wrote to that company and telephoned the Central Electricity Generating Board without eliciting any reply. Belonging as I

do to the generation of people who worked in x ray departments before the war, I pointed out that we had been exposed to far higher intensities of low frequency electromagnetic fields than anyone living under pylons. I received no response.

The important feature of the "hazard" is the number of kilovolts per metre, but strangely the programme did not state whether the kilovolts were measured in peak or root mean square values. The latter are 30% less than the former. Even more important, power transmission is always in three phase so anyone not actually between the wires would be subjected to the vector sum of the three phases, a much lower figure than the figure for each wire separately.

In the days before the war before "shock-proofing" came into use, high voltage alternate current was transmitted from the transformer to the unprotected x ray tubes by aluminium tubes, supported usually from the ceiling by Bakelite insulators 30 or 40 cm long and by "rheophores," reels of wire similar to fishing reels that linked the tubes to the ends of the x ray tube. The distance between the live conductors and the radiographer or radiologist varied from a metre or less when screening to three or four metres during radiography. The operators were thus exposed to as much as 60 kV/m and at least 15 kV/m.

If all the radiologists and radiographers of my generation were subjected to intensities far higher than those produced in houses under power lines and if there really were harmful effects surely we would have shown the ill effects in such a striking manner that it would be impossible for it not to have been realised. I repeat my suggestion that the records of the Society of Radiographers and the British Institute of Radiology, both of which existed in that era, should be analysed together with the technical points raised above. Surely if we have shown no excess mortality after years of exposure people can safely sleep under power lines.

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Unusual presentation of pseudomonas infection

Dr Michael J Weinbren and colleagues described three unusual presentations of *Pseudomonas aeruginosa* infection in leukaemic patients (22 October, p 1034). We would like to describe two similar cases we have treated recently.

Case 1—Ten days after starting induction chemotherapy with mitozantrone and cytarabine for acute myeloid leukaemia a neutropenic 74 year old man complained of discomfort in the right nostril. He was afebrile and there was no evidence of local inflammation. Over the next 12 hours he became feverish and developed severe cellulitis and gross oedema of the face accompanied by dyspnoea. He was promptly started on empirical treatment with ceftazidime and benzylpenicillin but became shocked and died shortly afterwards. Blood cultures subsequently grew *P aeruginosa*, and necropsy showed in addition an unsuspected pulmonary embolus.

Case 2—Eighteen days after starting induction chemotherapy with mitozantrone and cytarabine a 66 year old woman with acute myeloid leukaemia developed cellulitis affecting the right side of her face associated with haemorrhagic ulceration of the corresponding buccal mucosa. Azlocillin and netilmicin were given initially, and when a fully sensitive strain of *P aeruginosa* was isolated from both blood cultures and an oral swab ceftazidime was added. The facial lesion became necrotic, however, and the cellulitis continued to spread down the neck and across the upper chest wall. Granulocyte infusions were given on three occasions. Fortunately the infection began to