intubation (with considerable difficulty) and ventilation for 48 hours. Airway pressures at the onset of ventilation were low (29 kPa). Recovery was otherwise uneventful. Bronchoscopy 14 days after admission showed minor swelling of the arytenoid cartilages, but the trachea and main bronchi were normal. Cytomegalovirus was isolated from upper respiratory tract secretions obtained on admission.

This case shows that inflammation confined to the region of the arytenoid cartilages can produce life threatening upper airway obstruction in the presence of a normal epiglottis, suggesting that supraglottitis might be a more accurate name for the condition. The previous intubation might have contributed to this illness, but the acute onset with fever and lack of features of previous airway trauma suggest that this was a primary infective condition, possibly due to cytomegalovirus.

Upper airway obstruction may be extremely difficult to diagnose in the presence of coexisting lower airway narrowing, as occurs in asthma and chronic obstructive airways disease. Upper airway obstruction should be considered as a cause for lack of improvement in severe exacerbations of asthma and chronic obstructive airways disease and when severe respiratory distress persists despite evidence to suggest diminishing bronchospasm. Indirect or direct fiberoptic examination of the larynx and upper airways should be performed.

M CHESTER
M A WOODHEAD
F J C MILLARD

Department of Thoracic Medicine,
St James's Hospital,
London SW12 8HW

D A JONATHAN

Department of Otolaryngology,
St George's Hospital,
London SW17 0QT

SIR,—Following the cases described by Dr S Gerrish and others I would like to report on a patient in whom microbiological investigations were of interest.

A 46 year old man presented in September 1987 with a 24 hour history of sore throat and progressive difficulty in breathing. He had returned 11 days previously from two months in Kenya. On examination he was feverish with inspiratory stridor. Indirect laryngoscopy confirmed acute epiglottitis and an emergency tracheostomy was performed. Treatment was begun with ampicillin 1 g intravenously four times a day and 100 mg hydrocortisone. A throat swab taken before treatment grew *Haemophilus influenzae* type B biotype I (Oxford Public Health Laboratory) resistant to ampicillin (β lactamase positive), chloramphenicol (chloramphenicol transacetylase positive), trimethoprim, and tetracycline but sensitive to cefuroxime, cefotaxime, and rifampicin (blood cultures were negative). The patient had remained feverish while receiving ampicillin but made an uneventful recovery after 10 days' treatment with intravenous cefotaxime 2 g three times a day.

Because ampicillin and chloramphenicol are extremely valuable for life threatening invasive *H influenzae* type b infection such as meningitis (usually in children) and epiglottitis the multiple antibiotic resistances of this probably imported strain were of concern. When there are children aged under 4 at risk rifampicin prophylaxis (20 mg/kg (600 mg maximum dose) a day for four days) is recommended for household contacts of patients with type b *H influenzae* infection¹; it eliminates carriage of *H influenzae* type b in over 95% of contacts. Antibiotics given to treat acute illness are usually less successful in eradicating *H influenzae* type b, so rifampicin may also be given to the index case after antibiotic treatment with other agents.¹

Our patient had no household contacts under 4 years of age, but because of the multiple resistances of his isolate rifampicin was advised in an attempt to eradicate carriage of the organism.

Blood cultures should be taken in epiglottitis, even though the yield of positive results may be as low as 23%.² Examination of the throat in children is traditionally not advised owing to the risk of provoking acute respiratory obstruction. Indirect laryngoscopic visualisation of the glottis is permissible in adults,² and practices probably vary regarding the taking of swabs for culture. It would be of interest to know whether laboratories overseas are encountering multiple antibiotic resistance in *H influenzae* type b and if secondary cases of invasive infection have occurred in household contacts of patients with adult epiglottitis.

I thank Mr C J Randall for permission to report this case.

LINDA V BOOTH

Public Health Laboratory,
Southampton General Hospital,
Southampton SO9 4XY

- 1 Immunisation Practices Advisory Committee. Update: Prevention of *Haemophilus influenzae* type B disease. *MMWR* 1986;35:170-80.
- 2 Mayo-Smith MF, Hirsch PJ, Wodzinski SF, Schiffman FJ. Acute epiglottitis in adults. *N Engl J Med* 1986;314:1133-9.

Psoriatic science

SIR,—Dr C M E Rowland Payne (7 November, p 1158) makes the assumption that keratinocytes cannot be defective in psoriasis because they proliferate at the same rapid rate in vitro as keratinocytes from normal subjects. This is not sufficient evidence, however, on which to base this conclusion; psoriatic and normal keratinocytes may respond differently to agents that enhance or inhibit cell growth.

Recent work carried out in our laboratory suggests that psoriatic keratinocytes do indeed differ from normal keratinocytes in their response to γ interferon, which has previously been shown to inhibit the growth of keratinocytes in a concentration dependent fashion.¹ Keratinocytes from psoriatic lesions, unlike those from normal skin, do not express HLA-DR antigens in the presence of γ interferon, nor is their growth inhibited. Proliferation of keratinocytes from unaffected psoriatic skin is likewise unaffected by γ interferon, although these cells can be induced to express HLA-DR antigens. This abnormal response to γ interferon, a cytokine produced by activated T lymphocytes and present in psoriatic lesions,² may contribute to the increased epidermal cell proliferation seen in this disease.

The example cited to illustrate that the primary defect in psoriasis is in the skin—that is, transplanting psoriatic skin to nude, athymic mice—is almost certainly inappropriate. We have recently transplanted psoriatic skin to nude mice and found that four or five weeks after transplantation the skin could no longer be classified as psoriatic as defined by histological criteria. Interestingly, T lymphocytes disappeared from the graft only two days after transplantation. Thus any defect of the skin is not sufficient by itself to maintain a psoriatic lesion; T lymphocytes and probably humoral factors are implicated in the disease process.^{3,4} Indeed, in the apparently normal, non-lesional skin of psoriatic subjects both epidermal cell proliferation and the number of T lymphocytes in the dermis are increased.⁵

Our findings suggest, therefore, that the pathogenesis of psoriasis may be explained by an altered response by keratinocytes to mediators produced by activated T lymphocytes, subsequent to their interaction with antigen presenting Langerhans

cells in the epidermis.³ Such T cell factors may alter fibroblast function, and this may in turn stimulate keratinocyte proliferation. Although it is probable that the primary defect in psoriasis is to be found in the skin, it cannot be claimed to reside in the dermal fibroblast on current evidence.

B S BAKER
A V POWLES
LIONEL FRY
J P MCFADDEN
L BRENT

Departments of Immunology and Dermatology,
St Mary's Hospital and Medical School,
London W2 1NY

H VALDIMARSSON

Department of Immunology,
University Hospital,
Reykjavik, Iceland

- 1 Nickoloff BJ, Basham TY, Merigan TC, Morhenn VB. Anti-proliferative effects of recombinant alpha and gamma interferons on cultured human keratinocytes. *Lab Invest* 1984;51:697-701.
- 2 Bjerke JR, Livden JK, Degre M, Matre R. Interferon in suction blister fluid from psoriatic lesions. *Br J Dermatol* 1983;108:295-9.
- 3 Valdimarsson H, Baker BS, Jonsdottir I, Fry L. Psoriasis: a disease of abnormal keratinocyte proliferation induced by T lymphocytes. *Immunol Today* 1986;7:256-9.
- 4 Baker BS, Griffiths CEM, Lambert S, et al. The effects of cyclosporin A on T lymphocyte and dendritic cell subpopulations in psoriasis. *Br J Dermatol* 1987;116:503-10.
- 5 Baker BS, Swain AF, Fry L, Valdimarsson H. Epidermal T lymphocytes and HLA-DR expression in psoriasis. *Br J Dermatol* 1984;110:555-62.

Compulsory treatment in the community for the mentally ill?

SIR,—Ms Clare Dyer (17 October, p 991) raises urgent issues for the future practice of psychiatry in Britain. In many of the United States such practices are already a reality.

In 26 states and the District of Columbia there are explicit laws governing compulsory outpatient treatment. Eligibility may be under the same condition of dangerousness as inpatient commitment (Michigan); even so, there may be no provision for enforcing treatment for non-compliers (Pennsylvania). Alternatively, a lesser requirement may be set for outpatient treatment. In North Carolina, for example, the probability of dangerousness without continuing treatment is sufficient to warrant compulsory treatment in the community.

In a further 20 states unratified ad hoc arrangements provide alternative models. In Rhode Island an informal order binds the patient to outpatient follow up, failing which admission follows automatically. In Wisconsin a lawyer negotiates a contract, which the patients and treatment team sign. Massachusetts provides for a judge to find a patient "incompetent" to consent to treatment and to merit compulsory outpatient treatment until competence can be re-established. Indeed, the American Psychiatric Association recommends coercion outside hospital only for those patients deemed incompetent.

Practitioners working within these legal provisions describe enforcement as the central issue. When patients refuse treatment can they be forcibly medicated at home? Predominantly the course taken is to admit such patients to hospital and enforce medication there.

Evaluative research in this area is sadly meagre, and case reports¹ do not allow us to judge which model can best deliver treatment to these patients. These legal provisions have come to the fore now that patients are being taken out of institutions and attempt to balance the patient's rights to treatment and to privacy. They carry the danger of becoming overrestrictive responses to the needs of the seriously mentally ill where inadequate