sees urgent surgical problems deferred for what he regards as less important a problem (21 November, p 1348).

In my practice I have had three patients who were transsexual, one female to male and two male to female. All were profoundly unhappy misfits, and one of the men had been labelled psychopathic. I have lost contact with the female to male patient, but since gender reassignment both the male to female patients have become integrated useful members of society and all their previous psychological problems seem to have disappeared.

Their misery as transsexuals is quite as great as that of patients requiring vasovasostomies, although it might seem to us to be more bizarre.

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SIR,—I agree completely with Mr Grant Williams's view that "to pursue gender reassignment surgery in the current climate must be bottom of the list of medical importance." However, he weakens his argument considerably by mentioning the need to do vasovasostomies for the reversal of vasectomy. This operation must be next to the bottom of medical importance. Should we not remember the *Concise Oxford Dictionary*'s definition of medicine as "the art of restoring and preserving health" and restrict surgery to that aim while resources remain scarce?

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SIR,—The correspondence after my leading article (22 August, p 454) requires summary. Mr Grant Williams would place gender reassignment surgery at "the bottom of the list of medical importance" and possibly abandon it altogether in view of the current pressures on the health service. His view will certainly attract support, especially from those who do not know the strange suffering of transsexual people and the improvement in the quality of their lives that can follow a carefully supervised gender reassignment programme. Mr Williams points out that members of the division of surgery at Charing Cross Hospital oppose the continuation of gender reassignment operations, and this is understandable since this one hospital has undertaken the major proportion of this work for the whole of Britain. This unfair burden should be corrected, as I pointed out, by the establishment of regional services.

Mr Williams is unfair in his criticism of the outcome studies conducted by the psychiatry team at Charing Cross Hospital for it carried out the study in a reasonable way. It would not be possible to randomise patients to a surgical and a non-surgical group since the decision to refer for surgical reassignment is a late event in a long period of contact, which includes other treatment procedures. This is not to say that outcome studies cannot be improved; they can, and they must, if psychiatrists are to continue to persuade their surgical colleagues that such a complex intervention is justified. The idea of independent assessment which I put forward should be considered, and we have in fact just completed such an assessment in our small series in Leeds.

Mr Williams is correct in saying that surgical reassignment does not always improve the quality of life and that suicide may occur. When this happens it is probably because staff have allowed reassignment to occur at too urgent a pace and the patient has not adapted to the new gender role before surgery is undertaken. A major task of the psychiatrist is to resist importunate pressure from unsuitable applicants. This is also a point in favour of continuing reassignment on the NHS. Those who have the money to "buy" their reassignment may dictate the pace, often to their own detriment.

I hope that gender reassignment can continue by carefully trained teams. The team at Charing Cross Hospital, and those whose decisions have enabled them to continue their work, should deserve the respect due to their commitment and they should not be pilloried or accused of carrying out trivial procedures at the public expense.

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**This correspondence is now closed.—ED, *BMJ*.

Child abuse and osteogenesis imperfecta

SIR,—We welcome Dr L S Taitz's timely reminder of the fact that osteogenesis imperfecta causes unexplained fractures in early childhood and can be mistaken for non-accidental injury (31 October, p 1083).

We hold a register of clinical details of 874 patients with osteogenesis imperfecta, of whom 773 live in the United Kingdom. All but 51 of these have been classified according to the Sillence scheme.[1] Types I and IV are mild or moderately severe cases, dominantly inherited, or arising as a new mutation. People with types IA and IB have classical blue sclerae throughout life, whereas those with types IVA and IVB have normal sclerae in adult life but often pale blue sclerae in early childhood.[2] Those with types IB and IVB have overt dentinogenesis imperfecta. Type II is very severe and causes stillbirth or early neonatal death; this group is identified by us in only the very few patients who survive the neonatal period. Patients with type III disease usually have fractures at birth and progressive deformity thereafter.

The table shows the figures for the 773 patients in the United Kingdom in whom the diagnosis is not now in doubt. In more than 10% of cases the parents had to contend with accusations of child abuse at the time of the first few fractures, and in 13 cases formal case conferences or care proceedings were arranged. In the worst case a child was in care for three and a half years before the diagnosis of osteogenesis imperfecta was accepted.

Can such inappropriate and damaging care be prevented? In known cases of osteogenesis imperfecta parents should be provided with a suitable letter to show to casualty staff. The real difficulty arises in patients with type IVA osteogenesis imperfecta with normal or near normal sclerae, normal teeth, and usually normal radiographic appearances at the time of the first fracture.[2,3] In 30% of cases (55% of patients born after 1970) parents had to face allegations of non-accidental injury.

In the type IVA disease a family history may be helpful, and apparently minor signs, particularly joint flexibility, may be important. New dominant mutation is, however, well recognised.[4] Wormian bones, if present, are helpful, but it is not true that their absence excludes osteogenesis imperfecta. Many of the patients reported on by Cremin and others had type III disease[5] and that series included no type IV patients at all (P Beighton, personal communication). Our experience of type IVA osteogenesis imperfecta indicates that excessive numbers of wormian bones are seldom found.[2] Osteopenia is seen in less than half of the patients at the time of the first fracture[2] and even in adults with osteogenesis imperfecta is uncommon in bones not previously fractured.[6]

Details of 773 United Kingdom patients with osteogenesis imperfecta

Sillence type	Total	Parents accused of non-accidental injury	Case conference or care proceedings
Type IA	346	40	2
Type IB	89	3	1
Type II	2	0	0
Type III	120	8	0
Type IVA	98	29	7
Type IVB	67	10	1
Unclassified	51	3	2

Our results indicate that type IVA osteogenesis imperfecta does occur without an obvious family history and without wormian bones or osteopenia. The number of such patients is indeed small, but in the United Kingdom as a whole such cases occur regularly; using either our figures or those of Dr Taitz we have probably failed to recognise some cases. Tragedies such as those of the 13 families who inappropriately faced care proceedings may, in part, be avoided by careful history taking, including a detailed search for all the features of osteogenesis imperfecta and for all the risk factors for non-accidental injury.

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Adult epiglottitis

SIR,—Dr S Gerrish and others (7 November, p 1183) reported on four adults with life threatening epiglottitis who presented with typical features of severe upper airway obstruction. We recently admitted a young woman with severe upper airway obstruction due to supraglottitis which was not initially apparent because of coincidental acute severe asthma.

One month before admission this 30 year old woman with a two year history of asthma had been electively ventilated for 72 hours for a severe attack of asthma from which she made an uneventful recovery. She presented with a four day history of sore throat and increasing wheeze. On examination she was feverish (39°C) and in respiratory distress and had widespread bronchospasm but no stridor. She was treated with nebulised β agonists, aminophylline, and steroids. Over the next 24 hours, as her bronchospasm improved, she developed progressive stridor, worsening distress, and respiratory failure (fractional inspired oxygen 0·5; arterial oxygen pressure 9·9 kPa, arterial carbon dioxide pressure 6·7 kPa). Bronchoscopy showed greatly swollen arytenoid cartilages but a normal epiglottis. Radiographs of the neck showed narrowing of the airway by soft tissue swelling localised posteriorly at the level of the vocal cords. Antibiotics and a humidified helium-oxygen mixture produced an initial improvement, but six hours later she deteriorated, requiring elective

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 airflow 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 fibreoptic examination of the larynx and upper airways should be performed.

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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 cefotamine 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* infection[1]; 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.

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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.

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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 reports[1] 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