the patient would be left breathless and sweating to fall rapidly back into light sleep. In between attacks the patient's sleep was poor. He woke frequently: one third of the night was spent in wakefulness, and he had negligible deep sleep and only a little rapid eye movement sleep near the end of the night. The patient was aware of the attacks but unable to control them, and on occasions he had sustained minor injuries. The pattern was the same every night. The patient remained chronically sleep deprived with a consequent propensity to fall asleep during the day, when he was still vulnerable to attacks.

A sleep polygraph contained features of "epileptic K complexes" and "bursts of highly synchronised high amplitude sharp waves and spikes,"[3] but our patient, like others reported on, showed no evidence of epilepsy.[2,4]

This particular parasomnia seems to be uncommon, and someone unaware of it could confuse it with sleepwalking or night terrors, or even sleep apnoea, if the patient presents with daytime sleepiness.[5] Unlike other parasomnias, however, this one apparently responds to carbamazepine. Our impression is that it is far more relentless and debilitating than sleepwalking or night terrors.

SHARON BORROW M L TATTERSALL
DAVID HARTMAN MARGARET OAKEY
PHILIP SEDGWICK A H CRISP

Sleep Disorders Service,
Sleep Laboratory,
Atkinson Morley's Hospital,
London SW20 0NE

1 Driver HS, Shapiro CM. Parasomnias. *BMJ* 1993;**306**:921-4. (3 April.)
2 Lugaresi E, Cirignotta F. Hypnogenic paroxysmal dystonia: epileptic seizure or a new syndrome? *Sleep* 1981;**4**:129-38.
3 Peled R, Lavie P. Paroxysmal awakenings from sleep associated with excessive daytime somnolence. A form of nocturnal epilepsy. *Neurology* 1986;**36**:95-8.
4 Lugaresi E, Cirignotta F, Montagna P. Nocturnal paroxysmal dystonia. *J Neurosurg Psychiatry* 1986;**49**:375-80.
5 Maccario M, Lustman LI. Paroxysmal nocturnal dystonia presenting as excessive daytime somnolence. *Arch Neurol* 1990;**47**:291-4.

"Waking treatment" best for night terrors

EDITOR,—In their article on parasomnias Helen S Driver and Colin M Shapiro state that psychotherapy or hypnosis should be the first line of treatment for night terrors, particularly in children.[1] There is no evidence either that psychological factors contribute to night terrors or that psychotherapy is of any value. As night terrors are most common in the age group 2-6 it seems extraordinary to recommend hypnosis as the first line of treatment: hypnotising this age group is extremely difficult. In any event, again there is no evidence for the effectiveness of hypnosis in night terrors. Thus the two treatments recommended are expensive, irrational, impractical, and of no proved value.

What has become known as "the waking treatment" is based on waking the child immediately before the onset of the terror as manifested by signs of autonomic arousal.[2] Since this treatment was first described about 50 more children have been treated in the same way with a success rate of about 80%. Reports have also been received from many other countries of the effectiveness of the treatment in individual cases. This should be the treatment of choice, given its known effectiveness, lack of any toxicity, and economy.

BRYAN LASK

Department of Psychological Medicine,
Hospitals for Sick Children,
Great Ormond Street,
London WC1N 3JH

1 Driver HS, Shapiro CM. Parasomnias. *BMJ* 1993;**306**:921-4. (3 April.)
2 Lask B. A novel and non-toxic treatment for night terrors. *BMJ* 1988;**297**:592.

Simple treatment for night terrors

EDITOR,—Helen S Driver and Colin M Shapiro recommend psychotherapy or hypnosis as the first line of treatment for night terrors in children.[1] These are expensive and often unavailable interventions.

Nightmares with a recurrent theme are often mentioned in child and adolescent psychiatric clinics, although usually not as the primary problem. I ask the child, with parental help as appropriate, to write out or draw as a comic strip, depending on age and ability, an account of the dream. He or she is then instructed to change the ending in a way that will make the dream safe. This has been effective when the dream has been triggered by, or based on, horror films and when it is part of post-traumatic stress disorder—for example, after sexual assault. This approach uses elements of cognitive-behavioural therapy. Palace and Johnston described a single case study using a similar technique, coupled with systematic desensitisation.[3] This is based on a theoretical model of dreams that postulates three components: periodic "visual bursts" witnessed passively and random in content; the emotional state or tone at the time of the visual burst; and cognitive integration and synthesis into a "plot."[4]

I recommend that the above approach to treating night terrors or recurrent nightmares in children and adolescents should be tried in primary care before drug treatment or more sophisticated and expensive methods are considered.

SEAN MASKEY

Child and Adolescent Mental Health Service,
Guildford,
Surrey GU2 5AZ

1 Driver HS, Shapiro CM. Parasomnias. *BMJ* 1993;**306**:921-4. (3 April.)
2 Palace EM, Johnston C. Treatment of recurrent nightmares by the dream reorganisation approach. *Behavioural Therapy and Experimental Psychiatry* 1989;**20**:219-26.
3 Seligman ME, Yellen A. What is a dream? *Behav Res Ther* 1987;**25**:1-24.

Nasendoscopy before surgery for snoring

EDITOR,—N J Douglas has written a succinct summary of the obstructive sleep apnoea syndrome and snoring.[1] We agree that surgical success in the treatment of obstructive sleep apnoea depends on careful selection of patients but disagree with the statement that there is "no easy way of finding out the level of obstruction during sleep."

The Royal National Throat, Nose and Ear Hospital has used sleep nasendoscopy in over 200 patients as a means of improving selection for surgery.[2] Sleep is induced with a mild sedative, and the upper airway is directly visualised with a flexible nasendoscope. Those patients who have predominantly palatal obstruction or vibration are offered palatal surgery. Those who have appreciable non-palatal airway obstruction are excluded from surgery and are assessed for alternative forms of treatment, including continuous positive airway pressure. This technique uses equipment and skills that are already available in otolaryngology departments and can be performed as an outpatient procedure. We recommend that this technique is used in conjunction with the history and findings on general examination and findings of sleep studies for assessing these patients as part of a multidisciplinary approach to managing snoring and sleep apnoea.

K T V REDDY
D R INGRAMS
M B PRINGLE
C B CROFT

Royal National Throat, Nose and Ear Hospital,
London WC1X 8DA

1 Douglas NJ. The sleep apnoea/hypopnoea syndrome and snoring. *BMJ* 1993;**306**:1057-60. (17 April.)
2 Croft CB, Pringle M. Sleep nasendoscopy: a technique of assessment in snoring and obstructive sleep apnoea. *Clin Otolaryngol* 1991;**16**:504-9.

Liver damage warning with insomnia remedy

EDITOR,—Chris Idzikowski and Colin M Shapiro state that "valerian has been found to improve the quality of sleep subjectively."[1] They fail to point out, however, that use of this herb has been linked to several cases of quite severe liver damage.[2] Despite this a growing number of manufacturers of "natural" health products are promoting valerian as a safe remedy for insomnia and anxiety. Surprisingly, I have yet to see a commercial preparation give any warning of possible hepatotoxicity or state that it should be used with caution by anyone with pre-existing liver disease. My personal experiences also indicate that pharmacists and retailers of health supplements are ignorant of any such connection.

The disturbing safety record of unusual herbal remedies has recently been the subject of several reports.[3,4] Practitioners who encourage the medicinal use of herbs (or those who see patients with mysterious abnormalities in liver function) must bear these adverse reactions in mind. Equally, patients who treat themselves with herbs like valerian ought to be protected by the presence of clear safety warnings on the packaging when genuine concerns about safety have been expressed. This is a matter on which the Department of Health needs to act.

CHARLES SHEPHERD

Friars Cottage Surgery,
Chalford Hill,
Gloucestershire GL6 8EH

1 Idzikowski C, Shapiro CM. Non-psychotropic drugs and sleep. *BMJ* 1993;**306**:1118-20. (24 April.)
2 MacGregor FB, Abernethy VE, Dahabra S, Cobden I, Hayes PC. Hepatotoxicity of herbal medicines. *BMJ* 1989; **299**:1156-7.
3 Perharic-Walton L, Murray V. Toxicity of Chinese herbal remedies. *Lancet* 1992;**340**:674.
4 Vanherweghem J-L, Depierreux M, Tielemans C, Abramowicz D, Dratwa M, Jadoul M, *et al.* Rapidly progressive interstitial renal fibrosis in young women: association with slimming regime including Chinese herbs. *Lancet* 1993;**341**:387-91.

Steroids cause sleep disturbance

EDITOR,—Chris Idzikowski and Colin M Shapiro's article on non-psychotropic drugs and sleep restated the problem of sleepless patients who are taking steroids, and we endorse their conclusion that awareness of this potential side effect should lead to more "appropriate prescribing."[1]

The indications for steroid therapy in advanced cancer are numerous,[2] and because rest and sleep are very important to this group of patients and their carers we have monitored steroid associated sleep disturbance closely. Most of our patients receiving high dose steroids (8 mg-16 mg dexamethasone daily) described changes to their sleeping habits in answer to specific inquiries. Many described insomnia and others described vivid, frightening, or morbid dreams (not mentioned in the article). The bad dreams seem to resolve as the steroid dosage is decreased. Steroid related insomnia may be relieved by changing the dosage schedule and confining the administration of steroids to the early part of the day. Traditionally, dexamethasone has been given at regular intervals because of its short plasma half life (3·6 (SD 0·9) hours). However, the more clinically relevant biological half life of dexamethasone is 36 to 54 hours, making it suitable for once daily administration.[3] Further relief of insomnia may be achieved by the regular prescription of night sedation for patients taking steroids and the night time administration of other drugs which have sedative side effects, such as antiemetics and antidepressants.

Recently we treated a 42 year old man with bowel obstruction at multiple sites due to advanced colonic carcinoma. A course of high dose steroids given eight months previously for radiotherapy induced emesis had resulted in a rapid evolution of a hypomanic state with complete insomnia. While treating him with steroids for his obstruction[4] we infused 12 mg of dexamethasone during the day and substituted a low dose of midazolam infusion at night. On this regimen his insomnia or hypomania did not recur and his bowel obstruction resolved on day 12. When he was converted to oral medication, diazepam 10 mg was substituted for midazolam at night and he continued to sleep well.

Sleep disturbance is not one of the best known side effects of steroids, but insomnia has medical and social sequelae that require recognition and action. Appropriate prescribing even in the most difficult cases may allow the patient enormous benefit from steroids and a good night's sleep.

ROSE TURNER
ELLI ELSON

Palliative Care Unit,
Royal Marsden Hospital,
Sutton,
Surrey SM2 5PT

1 Idzikowski C, Shapiro CM. ABC of sleep sisorders, non-psychotropic drugs and sleep. *BMJ* 1993;**306**:1118-20.
2 Hanks GW, Trueman T, Twycross RG. Corticosteroids in terminal cancer. *Postgrad Med J* 1983;**59**:702-6.
3 Dexamethasone. In: Dollery C, ed. *Therapeutic drugs*. Edinburgh: Churchill Livingstone, 1991:44-50.
4 Baines M, Oliver DJ, Carter RL. Medical management of intestinal obstruction in patients with advanced malignant disease. *Lancet* 1985;ii:990-3.

Buserelin nasal spray expires five weeks after bottle opened

EDITOR,—Intranasal buserelin is commonly used for pituitary desensitisation in preparation for induction of ovulation with gonadotrophins in programmes of in vitro fertilisation and gamete intrafallopian transfer. Common practice is to prescribe more buserelin than is needed in one cycle. It is not widely appreciated by clinicians (and patients) that when a bottle of nasal spray is opened a five week expiry date comes into effect. This is not stated on the patient information sheet—a fact that has been confirmed in writing by Hoechst to me.

Using intranasal buserelin after the five weeks runs the risk of it being ineffective and may result in the patient responding suboptimally.

DICKINSON B COWAN

Portland Hospital Fertility Unit,
London W1N 6AH

Chickenpox in pregnancy

Incidence underestimated

EDITOR,—Gwendolyn L Gilbert may have underestimated the incidence of chickenpox in pregnancy.[1] The incidence she quotes in those aged over 20—"about 2%"—comes from a 1981 paper. Since 1988 chickenpox has been notifiable in Scotland, and in this health board some 9·7% of notified cases have been in adults aged over 24 and 11·8% among females aged 15-34.[2]

Chapter 27 of the "green book" issued to all general practitioners is a good guide to dealing with the problem.[3] As most adults are immune, only pregnant women without a definite history of chickenpox need antibody testing to see if they require human varicella-zoster immunoglobulin. Even among those with a negative history of chickenpox some two thirds turn out to have antibody, which is fortunate as supplies of immunoglobulin are limited.[3] The green book gives the distribution points for the immunoglobulin except for Scotland (where it is obtained through the blood transfusion service).

There may be a case for routine antenatal screening for varicella antibodies, as is done for rubella. This is especially true if, as we have speculated,[2] relatively more people are being infected in their adult years and because there is the prospect of a vaccine being introduced.[4]

D S G SLOAN

Department of Public Health,
Argyll and Clyde Health Board,
Paisley PA2 7BL

1 Gilbert GL. Chickenpox during pregnancy. *BMJ* 1993;**306**:1079-80. (24 April.)
2 Sloan DSG, Burlison A. Shift in age in chickenpox. *Lancet* 1992;**340**:974.
3 Department of Health. *Immunisation against infectious disease*. London: HMSO, 1992.
4 Controversy about chickenpox [editorial]. *Lancet* 1992;**340**:639-40.

Pneumonitis more severe

EDITOR,—Gwendolyn L Gilbert states that "most people in developed countries . . . develop chickenpox during childhood,"[1] but recent epidemiological data have indicated an upward shift in the age distribution of chickenpox in the United Kingdom[2] and United States.[3] The threat of chickenpox pneumonitis in pregnant women is estimated to occur in 1-5/10 000 pregnancies.[4] When varicella pneumonitis does occur in pregnancy and remains untreated the mortality may be as high as 40% or more.

Although I agree that there are no data to indicate that varicella is more common in pregnant women than non-pregnant adults, Gilbert understates the importance of other risk factors in determining the severity of chickenpox in pregnancy. There is good evidence to suggest that pneumonitis in the third trimester is more severe and correlated with higher mortality, presumably as a result of advancing immunosuppression during the later stages of pregnancy, and that tobacco smoking is also a crucial risk factor for pneumonitis.[5] Clinicians should bear these facts in mind when assessing a pregnant patient with chickenpox. I believe that the presence of the above associated risk factors should merit either referral to an infection unit for observation or close supervision by the general practitioner. Only early treatment with acyclovir will prevent progression of pneumonitis. No advese effects have been associated with use of acyclovir in late pregnancy.

Finally, susceptible pregnant women who have had appreciable exposure to chickenpox should be given zoster immune globulin. The Department of Health's publication *Immunisation Against Infectious Disease* indicates that varicella-zoster immune globulin may attenuate chickenpox in pregnancy when given up to 10 days after exposure as opposed to "up to four days after contact," as stated in Gilbert's editorial. Although zoster globulin is ideally given as soon as possible after contact, my practice is to recommend it for up to seven days after appreciable exposure.

DILIP NATHWANI

King's Cross Hospital,
Dundee DD3 8EA

1 Gilbert GL. Chickenpox during pregnancy. *BMJ* 1993;**306**:1079-80. (24 April.)
2 Miller E, Vurdien J, Farrington P. Shift in age of chickenpox. *Lancet* 1993;**341**:308-9.
3 Gray GC, Palinkas LA, Kelley PW. Increasing incidence of varicella hospitalisations in the United States army and navy personnel: are today's teenagers becoming more susceptible? Should recruits be vaccinated? *Pediatrics* 1990;**86**:867-73.
4 Stagno S, Whitley RJ. Herpes virus infection of pregnancy. 11. Herpes simplex and varicella zoster virus infections. *N Engl J Med* 1985;**313**:1327-30.
5 Ellis ME, Neal KR, Webb AK. Is smoking a risk factor for pneumonia in adults with chickenpox? *BMJ* 1987;**294**:1002.

Acyclovir for uncomplicated chickenpox?

EDITOR,—Gwendolyn L Gilbert's editorial on chickenpox during pregnancy[1] coincided with a maternal death attributable to varicella infection in our unit. We have studied the advice in detail and would ask for clarification on future management policy.

The patient presented at 36 weeks' gestation complaining of lower back pain, which was severe enough to warrant admission. On examination she had a characteristic chickenpox rash, having recently been exposed to two known cases. There was no clinical evidence of any of the systemic complications associated with varicella infection. Two days after admission she suffered a substantial antepartum haemorrhage, which was associated with fetal distress necessitating delivery by emergency caesarean section. At intubation tracheal vesicles were noted, and four hours after delivery the patient became dyspnoeic with an oxygen saturation of 88%. She was admitted to the intensive therapy unit, ventilated, and given intravenous acyclovir and broad spectrum antibiotics. Despite active treatment of her respiratory and circulatory failure her general condition deteriorated and she died after 14 days. Postmortem examination confirmed the cause of death to be bronchopneumonia secondary to viral pneumonitis complicated by viral myocarditis.

Acyclovir for the treatment of varicella infection in pregnancy is usually reserved for patients with complications. Al-Nakib *et al* suggested that there is no benefit to be gained in treating uncomplicated varicella infection in adults with acyclovir.[2] None of the patients in their study, however, were pregnant; the suppression of cell mediated immunity associated with pregnancy may mean that these results are not directly applicable to pregnant patients. A recent review stated that varicella pneumonia is more common in pregnancy, particularly in the third trimester, and that the infection is more severe and more complicated if it occurs at this time.[3]

In view of these facts and our own recent experience, we would be interested to hear whether treatment with acyclovir should be started during pregnancy in clinically uncomplicated cases of varicella infection, particularly in the third trimester.

CHRISTOPHER H MANN
MICHAEL P WYLDES

Manor Hospital,
Walsall WS2 9PS

1 Gilbert GL. Chickenpox during pregnancy. *BMJ* 1993;**306**:1079-80. (24 April.)
2 Al-Nakib W, Al-Kandari S, El-Khalik DMA, El-Shirbiny AM. A randomised controlled study of intravenous acyclovir, Zovirax, against placebo in adults with chickenpox. *J Infect* 1983;**6**(suppl):49-56.
3 Rodrigues J, Niederman MS. Pneumonia complicating pregnancy. In: Niederman MS, ed. *Clinics in chest medicine*. Philadelphia: W B Saunders, 1992:679-91.

Surveillance of congenital anomalies

False economies

EDITOR,—Barbara Russell has drawn attention to the difficulties involved in using routine data for the investigation of suspected environmental teratogens.[1] She commends the multisource, population based system of congenital anomaly registration operated by the Greater Glasgow Health Board and implicitly questions the epidemiological validity of the recently established Scottish congenital anomaly register, which is based on centrally recorded routine data, mainly derived from hospital discharge notifications.

The anxieties expressed by Russell about such centrally recorded data are well founded. A syste-