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conditions like hyperprolactinaemia, partly because in developed countries there is less pressure on women to become pregnant, and partly because of increasing public knowledge about medical matters. Nevertheless, the incidence is still comparatively high among black people in Africa,^{10 11} and in developed countries immigrants may remain at risk.^{3-5 12} Patients are usually naive about medical matters,¹³ and may have either a strong desire for pregnancy or a fear of conceiving.¹⁴ The condition may be a form of hysterical conversion,³ or depression may be present.^{1 6 14} A "pregnancy" may help a woman cope with distress or loss,^{6 15} and an association with child stealing has been reported.¹²

What is the endocrine mechanism of pseudocyesis? In laboratory rats pseudopregnancy may be induced by various means, including genital stimulation,^{2 16} and is due to persistence of a corpus luteum in the absence of pregnancy; neurogenic suppression of prolactin inhibitory factor may occur, allowing prolactin to help maintain the corpus luteum.¹⁷ In human pseudocyesis, however, a corpus luteum is often absent^{2 17} and the basal plasma prolactin concentration may be normal^{18 19} or raised.^{5 17} Basal plasma concentrations of follicle stimulating hormone are normal,^{5 17-19} while the luteinising hormone value may be normal¹⁷⁻¹⁹ or raised.⁵ The pulsatile pattern of luteinising hormone and prolactin secretion is exaggerated,⁵ and administration of luteinising hormone releasing hormone and thyrotrophin releasing hormone produces exaggerated responses of luteinising hormone and prolactin respectively.¹⁷⁻¹⁹ In a recent study of five patients in Florida the gonadotrophin concentrations were within the normal range but luteinising hormone was consistently higher than follicle stimulating hormone, while prolactin and progesterone were mildly increased.¹³ Apart from the raised progesterone value this pattern is similar to that in polycystic ovary disease.

In some types of amenorrhoea—particularly hyperprolactinaemic amenorrhoea—there are increased concentrations of opioid peptides (endorphins). These inhibit pulsatile release of luteinising hormone,²⁰ and thus administration of the opioid antagonist naloxone to these women stimulates release of luteinising hormone. Since opioid peptides may influence behaviour as well as hormone concentrations it was suggested that their production might be increased in pseudocyesis; but when naloxone was given to women with pseudocyesis it failed to induce release of luteinising hormone or prolactin.¹³ After the patients were told their diagnosis, however, the naloxone response appeared to return to normal. This suggests that pseudocyesis is not associated with increased opioid activity, though possibly there may be a reduction in tonic opioid inhibition.

Treatment usually entails confronting the patient with the diagnosis.¹⁵ Normally when this is done hormone concentrations return to normal quickly, the abdominal distension begins to disappear,⁵ and there may even be a rapid drop in weight.²¹ Nevertheless, patients may resist the diagnosis,¹⁷ and once they realise the truth depressive illness may occur.⁷ Recurrence is common,^{2 4 14} and close cooperation between gynaecologist and psychiatrist is important.⁷ Psychotherapy^{2 14 15} and family therapy⁴ may be necessary, and appropriate follow up is essential.^{14 21}

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Phantom pregnancy

Pseudocyesis is a condition in which a non-pregnant—and non-psychotic—woman firmly believes herself to be pregnant and develops objective signs of pregnancy.¹ Most cases are said to occur between the ages of 20 and 39, though the age range in one series was 5-79,² and cases have recently been described in teenagers.³⁻⁵ The most common symptom is amenorrhoea or oligomenorrhoea, usually for nine months.⁶ There is also abdominal enlargement, but without effacement of the umbilicus. Breast changes, which occur in 80% of patients,² include tenderness and swelling, secretion of milk or colostrum, and areolar pigmentation. Patients often claim to feel fetal movements, usually earlier than in a genuine pregnancy.¹ There may be vomiting, morning nausea, aberrations of appetite, and weight gain, and a case of "toxaemia" has been reported.⁶ The diagnosis may be difficult,⁷ but nowadays should easily be made with the help of ultrasound.⁸ Occasional cases have been described in men,^{2 9} but these may be associated with psychosis or organic disease.⁹

Phantom pregnancy was first described by Hippocrates and has since affected all races and strata of society, including British royalty, American slaves, and Chinese coolies.² It seems to be becoming rarer,^{1 2} partly because increasing diagnostic accuracy means that it is no longer confused with

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Regular Review

Clinical management of benzodiazepine dependence

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The development of dependence after the long term use of benzodiazepines is now supported both by clinical evidence and by the results of double blind studies.¹⁻³ Withdrawal symptoms have been reported after treatment for as little as four to six weeks.⁴⁻⁵ The withdrawal symptoms observed are wide ranging, and, while they include some related to anxiety, they are clearly distinguishable from a simple re-emergence of pre-existing anxiety.⁶⁻⁸ Particularly frequently reported are instances of increased sensory perception such as hyperacusis, photophobia, paraesthesiae, hyperosmia, and hypersensitivity to touch and pain, but gastrointestinal disturbances, headaches, muscle spasms, vertigo, and sleep disturbances are also frequent.^{3,9,10} The proportion of long term users of benzodiazepines in whom withdrawal symptoms may be expected to emerge has been variably estimated to be between 15% and 44%.^{3,8} The symptoms typically emerge in the first week after stopping the drug but may develop after a reduction in dosage.^{7,10} Until recently the withdrawal syndrome was reported as lasting for up to three months,⁷ but we are now seeing more patients whose symptoms have persisted for more than six months—in some cases for a year or more.

Yet no one doubts that most patients currently taking benzodiazepines should stop them. One and a quarter million of the British population take benzodiazepines for more than a year,¹¹ although data supporting their continued effectiveness over such a period are sparse—to say the least.¹² A recent review concluded that benzodiazepines are no more effective than brief counselling by the general practitioner for the common minor affective disorders and that their prescription should be cut.¹³ There is, in addition, evidence suggesting possible psychological impairment and neuro-radiological changes associated with long term administration.^{14,15} Recent extensive publicity about tranquillisers has led to an increased consumer demand for medical guidance about withdrawal.¹⁶ Information on optimal withdrawal procedures is lacking—for little systematic research has been done on the treatment of benzodiazepine

dependence,⁶ and the studies that have been published have had methodological limitations.¹⁷ The guidelines set out below are based in part on a review of published work and also experience in our unit in withdrawing more than 60 patients over the past seven years.

Setting

In general withdrawal is best tackled in the outpatient setting.^{2,9} Patients having high doses or with a history of seizures or psychotic episodes during previous attempts at withdrawal are more safely treated as inpatients.

Rate of withdrawal

Stopping the drug abruptly is more likely to lead to severe withdrawal symptoms such as fits or confusional states^{18,19}—and to loss of patients from the withdrawal programme.³ No consensus exists, however, on the precise duration of the withdrawal process or the size of each reduction in dosage. Four weeks is probably the minimum period,^{1,2,9} and programmes as long as 16 weeks have been recommended.²⁰ Over the withdrawal period dosage should gradually be tapered off in steps ranging from 0.5 to 2.5 mg diazepam or its equivalent.^{3,4,7,12,21} The table sets out equivalent doses for benzodiazepines on the limited list.

Some patients referred for withdrawal of benzodiazepines may already be complaining of withdrawal symptoms.¹⁰ Published reports tend, however, to emphasise patients who had severe problems in withdrawing and so need appraisal by specialists. Some and possibly most patients will suffer less severe withdrawal symptoms and will cope well with a fairly rapid reduction in dosage over a few weeks.²²

In our view—and certainly for patients who have previously had problems withdrawing—the rate of reduction of dosage should not be fixed at the outset but should be “titrated” against the patient’s withdrawal symptoms.