The most potential harm, however, lies in the drug's selective serotoninergic toxicity and its dopaminergic actions,[4 5] and the suggestion that it might induce psychosis de novo deserves further research.

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Postoperative urinary retention

SIR,—We welcome Messrs J B Anderson and J B F Grant's report on the use of intermittent catheterisation in men with postoperative urinary retention.[1] Their description closely mirrors our experience of postoperative urinary retention in women who have fractured their leg. We have also found that intermittent catheterisation needs to be continued for weeks or months. Eleven women aged 70-86 required intermittent catheterisation for between nine and 120 days. Ten patients recovered continence, and only one required a permanent indwelling catheter.[2] Women commonly present with urinary incontinence, frequency, and voiding of small volumes as well as failure to void. We have carried out urodynamic studies in 13 women presenting in this way, 12 of whom had an acontractile bladder.

Intermittent catheterisation offers many advantages over the use of indwelling urethral or suprapubic catheters. The presence of a catheter bag may inhibit normal dressing and walking in the immediate postoperative period, preventing the patient from benefiting from early mobilisation.

We have observed that some women have a raised residual volume (>300 ml) before undergoing surgery. This raises the question of how common subclinical retention is among young and old people living at home. A series of more than 1500 urodynamic studies in older patients has indicated a trend towards acontractility in older women (J M Lee, personal communication). As Messrs Anderson and Grant point out, postoperative urinary retention is a common condition that affects patients' comfort and length of stay in hospital. Possibly by identifying patients at risk it could be predicted and treated earlier than at present.

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SIR,—We were impressed by Mr P H O'Reilly's statement that about four fifths of cases of postoperative urinary retention in men have an iatrogenic cause.[1] We have become increasingly concerned with the influence of opiates and opiate-like drugs in triggering acute and postoperative retention in elderly people.

Opiates have long been recognised as one of several classes of drugs that cause urinary retention.[2] Urodynamic investigation of patients receiving epidural morphine has shown that opiates induce inhibition of detrusor contraction and impaired bladder sensation, resulting in an increased maximum bladder capacity and overdistension.[3] These changes are rapidly reversed by therapeutic doses of intravenous naloxone. In addition naloxone increases urinary urgency[4] and reverses both urinary retention and paralytic ileus induced by opiates.[5 6] Animal studies have confirmed that reflex contraction of the urinary bladder can be induced by naloxone[7]: the parasympathetic reflex pathway to the urinary bladder is subject to tonic enkephalinergic control, which may be antagonised by naloxone.

Catheterisation, particularly in elderly people, is associated with an increased prevalence of urinary tract infection, increased morbidity, and a threefold increase in mortality in hospital.[8] We suggest that the therapeutic use of naloxone would safely reverse some cases of acute retention associated with opiates, thereby avoiding the risks of urinary catheterisation.

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Thyroid function in fetuses with chromosomal abnormalities

SIR,—Mr J G Thorpe-Beeston and colleagues begin their short report by stating that the incidence of thyroid disorder in children with Down's syndrome is reported to be 6%.[1] The study from which this figure has been quoted actually found that the incidence of thyroid disorder in children was 10% (6% congenital and 4% acquired) if thyroid disorder was defined by abnormal concentrations of thyroxine and thyroid stimulating hormone and 37% if it was defined by an abnormal concentration of only one of the hormones.[2]

Mr Thorpe-Beeston and colleagues concentrate on only congenital hypothyroidism and thus would have been better served by quoting a study of 1130 infants that reported an incidence of congenital hypothyroidism of 0·7% (detected by neonatal screening).[3] It is this figure of 0·7% that is "28 times higher than that expected in the general population" and not the 6% referred to by the authors.

Furthermore, the authors state that "Evidence suggests that early diagnosis and effective treatment may not only improve the physical wellbeing of such infants but also have an appreciable impact on intellectual function." Unfortunately, their reference refers only to a prevalence study,[4] and at no point was a therapeutic trial carried out showing any evidence of the benefits of treatment as the authors imply. Evidence does exist showing the effectiveness of treatment but only for definite hypothyroidism.[5] For thyroid disorder on the borderline of normal a recent study failed to show any efficacy of short term treatment with thyroid hormone.[6]

The authors also comment on prompt postnatal treatment in preventing adverse sequelae. They fail to point out, however, that congenital hypothyroidism may be transient and thus resolve without any medical intervention; this was the case in 27% of the cases in one study.[3] Prompt treatment may in fact mean inappropriate and unnecessary treatment, possibly for life.

Finally, the authors fail to discuss the hypothesis that thyroid disorder may predispose to chromosomal abnormalities. Evidence exists showing that a thyroid disorder in a woman may predispose her to have a fetus with Down's syndrome.[7]

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AUTHORS' REPLY,—We are grateful to Dr Prasher and Professor Corbett for clarifying some of the statements that we made in our short report. Because of the limitations placed on short reports we could not fully expand on the quoted references. We considered it appropriate to reference the most recent work. Although the stated prevalence of thyroid disorders in Down's syndrome varies from 0·7% to 37%,[1 2] it is accepted to be much higher than that observed in the general population.

The aetiology of this thyroid dysfunction remains uncertain. Postnatally, autoimmune thyroid disease is considered to be the most likely cause of the hypothyroidism, with thyroid autoantibodies being reported in up to 30% of children with Down's syndrome.[3] This has not, however, proved to be the case in our fetuses with chromosomal abnormalities. Thus in a prospective study of blood samples from five fetuses with trisomy 21 and high thyroid stimulating hormone concentrations, at 16-32 weeks' gestation (mean 24 weeks) there were no detectable microsomal or thyroglobulin antibodies on enzyme linked immunosorbent assays (ELISA).

An alternative explanation, that maternal thyroid disorder predisposes to fetal chromosomal abnormalities, is also unlikely as in our study the serum concentrations of thyroid stimulating hormone and free and total thyroxine and triiodothyronine in women with fetuses with chromosomal abnormalities were not significantly different from those in women with fetuses that were chromosomally normal.

In a study of 151 patients with Down's syndrome Pueshel and Pezzullo found that the intellectual function of those with abnormal concentrations of both thyroid stimulating hormone and thyroxine was significantly lower than that of those with either normal thyroid function or an increased concentration of thyroid stimulating hormone alone.[4] They postulated that early detection of thyroid dysfunction and prompt treatment should