Outcome of elective prostatectomy

SIR,—The paper by Mr David E Neal and colleagues on the predictive role of urodynamics in assessing the outcome of elective prostatectomy¹ continues a fascinating debate. It seems correct that the basic parameters of micturition—namely, pressure and flow—should form the basis of a diagnosis of obstructed micturition, but there are several factors that may have prevented Mr Neal and colleagues from reaching this conclusion.

Firstly, we know that the symptoms of patients with obstructed micturition are unreliable, and there have been few good correlations with urodynamic findings.^{2,3} We also know from several drug studies that there is a large placebo effect in these patients: in a recent study 60% of patients declared themselves much improved after one month receiving placebo.⁴ An operation is probably a far better placebo than any drug. Hence the use of only analysis of symptoms as the final outcome when there was no control group is less than satisfactory.

Secondly, as Mr Neal and colleagues pointed out, theirs was a selected series—that is, those patients with a maximum urinary flow rate > 14 ml/s were excluded. My series, which they quoted, was truly unselected,⁵ and the results showed good flow to predict lack of improvement or a poor result. I believe that the authors are correct in saying that excluding patients with high preoperative flow may have masked the beneficial predictive effect of low flows. I would be interested to see what the preoperative voiding pressures and changes in flow and pressure were after surgery in patients with flows > 14 ml/s. If they had high preoperative voiding pressures then they had obstruction and should have benefited from surgery. If their preoperative voiding pressures were normal they did not have outflow obstruction, and their improvement must have been either a placebo effect or some hitherto unreported effect of prostatectomy.

If Mr Neal and colleagues are advocating prostatectomy for treating symptoms in patients with no outflow obstruction then this is an expensive and dangerous placebo treatment. Their warning that patients with low flow due to detrusor underactivity is well stated and supports the work of Schafer *et al.*⁶ Further work should be carried out to increase the predictive value of urodynamics, and advanced flow curve analysis may be one way forward.

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AUTHORS' REPLY.—We agree completely with Mr Abrams that debate and further research are necessary to define the hard ground for recommending prostatectomy. We found certain preoperative urodynamic measurements to be associated with outcome; thus men with stable detrusors and high voiding pressure had greater success rates (88%) for prostatectomy than men with low voiding pressure (60%). Although urodynamic data and outcome were associated, they

were not sufficiently precise to predict individual outcome accurately. Failure to account for this point explains some confusion in the published work on urodynamics.

One concern was that the men were not representative as some with high preoperative flows who might have had a poor outcome were excluded. Mr Abrams studied 53 men selected on clinical grounds: their preoperative flow rate (8 ml/s) was similar to that of our "selected" men ($n=217$; 9 ml/s); moreover, the flow rate of his poor outcome group ($n=14$; 11 ml/s) suggests that our entrance criteria should have included most of them. We operated on 16 men with flow rates of ≥ 15 ml/s; their voiding pressures mirrored those of the remainder, and all but one of them did well. Mr Abrams's and other studies do not confirm his opinion that preoperative flow rate measurements are a strong predictor of individual outcome.^{2,3}

Secondly, he thought that we should have used urodynamic parameters to define the need for and the outcome of surgery. This is inappropriate because, as he agrees, preoperative symptoms correlate poorly with urodynamic data. Not only did we rigorously assess outcome at 11 months postoperatively by careful questioning by two independent observers, symptom scoring, and completion of a linear analogue scale, but 207 men underwent urodynamic or flow rate studies. We reported clear associations between outcome and postoperative urodynamic findings. The suggestion that men with normal voiding pressures are incapable of benefiting from prostatectomy because they do not happen to conform with rigid concepts of outflow obstruction is surely not wholly serious in view of the known difficulties physicians have in defining outflow obstruction.⁴

Mr Abrams's fear that we advocate prostatectomy as a panacea for any urinary tract symptom is not derived from our paper: indeed we have shown that a quarter of men selected on clinical grounds and with low flow rates do not have a good outcome. We simply put to the test our idea that urodynamic studies might enable us to advise individual patients whether they should or should not undergo prostatectomy. This hypothesis was not supported by our study.

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Myalgic encephalomyelitis by proxy

SIR,—The letter by Drs F Harris and L S Taitz describing the damaging effects of the diagnoses of myalgic encephalomyelitis in children¹ prompts us to report a case that we believe shows myalgic encephalomyelitis by proxy. (The name of the general practitioner has not been cited as a co-author to protect patient confidentiality.)

A 12 year old boy presented with his mother to the general practitioner with complaints of recurrent nasal congestion, runny nose, sore throat, muscle aches, headaches, and lethargy. Clinical examination revealed no abnormalities apart from mild obesity. At the insistence of his mother, who was convinced her child was suffering

from myalgic encephalomyelitis (ME), the child was referred to a general medical clinic to reassure her that he had no actual illness.

When the mother was questioned on the child's symptoms the criteria for myalgic encephalomyelitis or the postviral fatigue syndrome² were sufficiently fulfilled to be diagnostic, but when the child was questioned the symptoms were not sufficiently diagnostic. Because of the overbearing nature of the mother it was necessary to interview the child separately to obtain an unbiased history. There were no supporting clinical or laboratory features of the syndrome or other illness.

The mother had presented to the same practice in March 1988 with headaches, painful muscles and joints, periorbital oedema, and swelling of fingers and feet. These symptoms persisted and were investigated. In April she diagnosed herself as having myalgic encephalomyelitis after watching a television programme on the subject. She joined the ME Association and became an active member. One month later a diagnosis of hypothyroidism was confirmed biochemically, the thyroid stimulating hormone being > 50 IU/l. She was referred to an endocrinology clinic and was treated with thyroxine. Her muscle aches persisted but other symptoms abated. Her symptoms and investigations were not diagnostic of the postviral fatigue syndrome, but she clung to the belief that she had myalgic encephalomyelitis despite being told the opposite. In May 1989 she was once again told that her symptoms were not typical of the condition. In the same month she brought her child to the practice for the first time with the symptoms she attributed to myalgic encephalomyelitis.

When interviewed in the general medical clinic the mother seemed crestfallen when admitting that her other doctors did not think she had myalgic encephalomyelitis. She frequently prompted her son on points of history, and he reluctantly agreed with her on these points.

Meadow described Münchausen syndrome by proxy as a syndrome in which anyone, but usually a mother, fabricates symptoms in another person.³ We believe that our case is an example of myalgic encephalomyelitis by proxy, possibly a variant of the Münchausen syndrome by proxy. We support the views of Drs Harris and Taitz on the part that the ME Association could play in preventing the referral of children to hospital clinics. When doctors are confronted with parents insisting on the diagnosis of myalgic encephalomyelitis in their children myalgic encephalomyelitis by proxy should be considered.

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- 1 Harris F, Taitz LS. Damaging diagnoses of myalgic encephalitis in children. *Br Med J* 1989;299:790. (23 September.)
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SIR,—Drs Frank Harris and L S Taitz's letter criticising the role of the ME Society (no such society exists) in the diagnosis of myalgic encephalitis (encephalomyelitis)¹ deserves further comment.

Firstly, there are two registered charities connected with myalgic encephalomyelitis (ME)—namely, the ME Association (president Dr Melvin Ramsay) and the ME Action Campaign (president Clare Francis). As there is no ME Society it is difficult to be certain who their criticism is aimed at.

The diagnosis and management of children with myalgic encephalomyelitis is clearly a sensitive and controversial topic. The policy of the ME Association, however, is quite clear—non-medical staff do not offer diagnoses, suggest treatment, or refer patients to specific doctors. If a general practitioner requests help in any of these

areas (as some do) then a qualified medical adviser will try to help. In the case of children this may consist of giving out the name of a sympathetic consultant paediatrician if the parents (or general practitioner) have been told the child cannot possibly have myalgic encephalomyelitis because "it doesn't exist in children."

I agree that the diagnosis of myalgic encephalomyelitis in children must not be made without rigorous clinical appraisal and know of one case to illustrate this point. The child was initially diagnosed as having myalgic encephalomyelitis; this was subsequently changed to myasthenia gravis (along with treatment) before the correct diagnosis of Lyme disease was made.

Paediatricians are no different from other specialists in their views on myalgic encephalomyelitis—some accept that it exists, whereas others are still quite hostile to the idea that it can affect children. Unfortunately, in purely practical terms, the incorrect management advice of forced return to full time schooling, physical exercise, and all "normal" activities results in rapid deterioration of the clinical condition.

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1 Harris F, Taitz LS. Damaging diagnoses of myalgic encephalitis in children. *Br Med J* 1989;299:790. (23 September.)

Children of mothers with bulimia nervosa

SIR,—We would like to add to Drs Alan Stein and Christopher Fairburn's interesting study of the children of mothers with bulimia nervosa.¹ In treating bulimic women of normal weight we found that the conflict between the demands of childrearing and the bulimic disorder creates a great deal of tension in the mother-child relationship. All of the five patients (table) whom we are currently treating were poorly prepared for their pregnancies, all of which were unplanned. The patients reported powerful guilt feelings if they had been binge eating or purging during the pregnancy. Only one patient had an uncomplicated pregnancy. Three of the women were single parents and had to manage the child during the bingeing episodes. This was usually done by confining the child to its room. Interruptions were usually poorly tolerated, and in case 2 the mother would physically punish the child when she interrupted her. Although we asked mothers about their attitudes to their children's shape and weight, we were unable to detect any undue concern and the children seemed to be developing normally. Any concern that was expressed related to the mothers' fear that their behaviour would lead the children to develop eating disorders later in life. The mothers experienced considerable difficulty feeding their children because of their resulting proximity to food and the possibility that this

would start a binge. Our clinical impression is that underweight mothers with anorexia nervosa are more likely to display undue concern about their children's shape and to severely underfeed the child.

In three of our patients the key factor in the decision to seek help was concern about the welfare of the child and the realisation that the bulimia was seriously interfering with the mother-child relationship. In case 1 the patient was encouraged to seek help by her social worker, who was concerned that the child was being neglected during the mother's binges.

Bulimia is a common disorder among young women.² Bulimic behaviour during pregnancy may have a detrimental effect on the fetus³; but the results of recent studies of treatment are encouraging. For these reasons we would advocate including a brief inquiry about binge eating, self induced vomiting, and laxative abuse in the assessment of all young pregnant women.

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Oestrogen deficiency and oestradiol implants

SIR,—We, as others,^{1,2} have long been aware that climacteric symptoms after an oestradiol implant may recur while the oestradiol concentration is still considerably raised. However, we dispute the conclusion of Dr Kevin Gangar and colleagues that this response is a form of tachyphylaxis—a "diminished response to later increments in a sequence of applications of a physiologically active substance" (*Webster's Medical Dictionary*). The phenomenon is analogous, rather, to that of poorly controlled diabetics who develop symptoms of hypoglycaemia when their blood glucose concentration is lower than usual, though it is still higher than in well controlled diabetics. As an implant induces persistent unphysiological, virtually pharmacological, concentrations of oestradiol it is not surprising that as oestradiol starts to fall symptoms reappear. Flushes characteristically develop with oestrogen withdrawal rather than just with low levels of oestradiol. Thus women with gonadal dysgenesis, whose oestrogen concentrations have always been low, do not flush unless they are treated with oestrogen that is then discontinued. And oestradiol concentrations are no different in menopausal women who flush than in those who do not.⁴

Although we agree that implants should not be renewed when oestradiol concentrations are still

greatly raised, we are concerned that Dr Gangar and colleagues expect women to tolerate severe symptoms while oestradiol slowly falls to physiological levels. This is unnecessary. By substituting an alternative preparation such as vaginal oestriol¹ symptoms can be controlled until a more physiological oestradiol concentration is attained. A decision can then be taken regarding future treatment.

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Difficult asthma

SIR,—Professor Peter J Barnes and Dr K Fan Chung remind us that patients whose asthma is precipitated by aspirin should also avoid other non-steroidal anti-inflammatory drugs as these similarly inhibit cyclo-oxygenase.¹ They state, however, that "for analgesia paracetamol and opiates are usually safe."

It is generally believed that opiates, which inhibit respiratory drive, are particularly hazardous in the presence of the increased airways resistance found in asthma.² In addition, opiates may release histamine, to which the airways of asthmatic patients are hyperreactive, leading to bronchoconstriction and further increase in resistance. Few physicians would prescribe opiates for patients known to be asthmatic. It would seem essential, therefore, that the authors provide the evidence on which they base their unexpected recommendation.

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AUTHORS' REPLY.—As we stated in our article, opiates are usually safe to give in patients with controlled asthma. They must be avoided during acute exacerbations as they reduce central respiratory drive. When directly injected into the skin opiates release histamine from mast cells by a mechanism which does not include an opioid receptor but do not degranulate mast cells of airways. Many years ago opiates were recommended for severe asthma, although no trial had been performed. There has recently been a revival of interest in using opiates in treating asthma as they are effective in inhibiting neurogenic inflammatory responses (due to the release of peptides from sensory nerves) in airways,^{1,2} which may contribute to the inflammatory reaction of asthmatic airways. In addition, morphine sulphate has been shown to inhibit vagally mediated bronchoconstriction in mildly asthmatic subjects³; in this study morphine did not cause bronchoconstriction. Newly developed opioids such as the pentapeptide BW443C may be useful; it does not

Details of mothers with bulimia nervosa and their children

Case No	Age (years)	Duration of bulimia nervosa (years)	History of anorexia nervosa	Age (years) and sex of child	Problem
1	24	5	Yes	2, M	Baby born at 38 weeks weighing 2170 g; mother unable to breast feed and unable to cope; drug and alcohol abuse; child taken into care voluntarily
2	33	8	Yes	2, F	Forceps delivery at 43 weeks; child has learning difficulties; mother has difficulty feeding child; no food in house; feeding child precipitates binges; mother locks child away during binges
3	22	2	No	1, F	Gained 29 kg during pregnancy; does not cook at home; feeding precipitates binges; feels guilty about effects on child
4	35	7	Yes	4, M	Amenorrhoea for seven years before pregnancy; did not know she was pregnant until 7 months; delivered at 33 weeks, baby weighed 2130 g; "left overs" precipitate binges; very guilty about effects on child
5	23	6	No	4, F	No food in house; does not cook for child; very conscious of child's weight; child sent to own room during binges