PREDICTION OF RESPONSE TO ECT

DEAR SIR,

The recent analysis of prediction of response to real and simulated treatment in the Northwick Park ECT trial (*Journal* 1984, **144,** 227–37) made fascinating reading. Its most notable finding was that the real ECT was significantly superior to simulated ECT only for the 22 deluded depressives included in the trial. The authors conclude that delusional depression may be a specific entity which is relatively resistant to tricyclic antidepressants but responsive to ECT.

However, the hypothesis that ECT is a relatively specific treatment for the entity of delusional depression fails to take account of the reports of its effectiveness in the treatment of mania (McCabe, 1976), schizophrenia (Taylor & Fleminger, 1980) and schizoaffective disorder (Ries *et al,* 1981). The possible common factor in these three conditions and in delusional depression is the presence of psychosis. The effectiveness of ECT in all these illnesses can thus be explained on the basis of ECT's having a specific, powerful anti-psychotic action.

As far I am aware this hypothesis has not previously been advanced. Much attention has been given to determining the mode of action of ECT's supposed anti-depressant effects, and in comparing its efficacy to that of anti-depressant drugs. It may be that future trials should incorporate comparisons of ECT with neuroleptic medication in suitable psychotic patients.

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BLADDER RUPTURE DURING ECT

DEAR SIR,

A 74 year old male with recurrent unipolar depression developed abdominal pain after the tenth well-modified treatment in a series of ECT. He became confused and was unwilling to eat or drink over the next 24 hours, and he did not pass urine. Abdominal distension with signs of generalised peritonitis developed. Blood urea was 30 mmol/l. At laparotomy, three litres of clear fluid were sucked out of the peritoneal cavity, a 6 cm longitudinal laceration in the dome of the bladder was sutured and a peritoneal drain inserted. A urethral catheter was passed without difficulty. Recovery was uneventful. Bladder histology was normal.

The patient had been investigated three years previously for difficulty in passing urine, and at that time although no obstructive lesion was found at cystoscopy, 2.5l of residual urine was measured. An in dwelling urinary catheter had been left for one month after which micturition returned to normal. Over about two months before the bladder rupture, the patient had been noted to have urinary dribbling and incontinence. He had not received tricyclic antidepressants during the year before bladder rupture.

In the absence of urological endoscopic instrumentation, closed intraperitoneal rupture of the bladder is generally associated with blunt injury to a distended bladder (usually after a high alcohol intake), but the abdominal trauma need not be severe. Obstructive uropathy, due to prostatism or urethral stricture is commonly associated. In this case, the anticholinergic effects of tricyclic antidepressants cannot be implicated.

Rupture of the bladder occurring in association with ECT is not recorded although ruptured spleen (Ernst, 1980) and perforated colonic diverticulum (Pippard & Ellam, 1981) have been reported.

Although this is an isolated case, it suggests that if there is any evidence of prostatism, urinary difficulty or retention, particular care should be taken to empty the bladder before ECT.

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ELECTROSTIMULATION VS ORAL METHADONE IN OPIATE WITHDRAWAL

I was interested to read Gossop *et al's* paper