independent of the diuretic and naturetic response. This vasodilator action is probably mediated by local prosta-
glandins, kinin and kallikrein activity. Pharmacologi-
cally, bumetanide is 40-fold more potent than frusemide
on a weight for weight basis. The K⁺ losses are less with
bumetanide.

The role of bumetanide is acute pulmonary odema and
congestive heart failure is well established. Bailey has
described the use of bumetanide in 10 patients 2 months
after their renal transplants and no serious side effects or
biochemical abnormalities occurred. As far as we are
aware, this is the only documented report of the use of
bumetanide in renal transplant patients. We recommend
its use at an early stage in the cadaveric renal transplants
to obtain diuresis.

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References

2. Sigurd, B., Hesse, B., Valentin, N. & Bollerup, A.C. Investiga-
tions with intravenous bumetanide. Postgrad Med J 1975, 51:
27–34.
4. Bailey, R.R. Open comparisons of diuretic effects of piretanide
and bumetanide in patients with stable renal transplants. N Z

Pseudo-obstruction secondary to anticholinergic
drugs in Parkinson’s disease

Sir,

We would like to report a case of pseudo-obstruction in a
patient with Parkinson’s disease who was taking lofeprami-
ne and oxybutinin. Neither drug has been reported to
cause this complication, and thus this case illustrates the
difficulty in treating patients with Parkinson’s disease
with anti-cholinergic drugs.

A 60 year old man with a 12 year history of Parkinson’s
disease, presented with urinary frequency, incomplete
voiding, and increasing depression. The main features of
his Parkinson’s disease were rigidity, marked dyskynes-
as, severe ‘on–off’ fluctuations and constipation.
Drugs on admission were cocaerelopa, co-beneldopa,
subcutaneous apomorphine as required, domperidone,
lactulose, frusemide, digoxin, nifedipine and aspirin.

Videocystometrography showed an unstable bladder
for which he was treated with oxybutinin 5 mg twice a day
and 10 mg at night.

Lofepramine was started 3 days later, at a dose of
70 mg at night, for depression. Three days after this, the
patient went into acute urinary retention and needed
catheterization. The oxybutinin and lofepramine were
stopped. Twelve hours later he was found to be hypoten-
sive and oliguric. On examination the abdomen was
distended and tender, bowel sounds were absent and the
rectum was empty. Abdominal radiographs showed

dilated large and small bowel, with air throughout the
bowel. A single contrast barium enema showed free flow
up to the caecum. Amylase, sodium and potassium were
normal.

A diagnosis of pseudo-obstruction was made and


treatment was conservative consisting of intravenous
fluids and a nasogastric tube. After 5 days he had made
a full recovery. All previous medication except the lofepra-
mine and oxybutinin were reintroduced without recur-
rence of the pseudo-obstruction.

The temporal relationship between the administration
of lofepramine and oxybutinin and the bowel obstruction
strongly suggests that one or both drugs played a
pathogenic role. It is of interest that lofepramine is widely
thought of as having markedly less anti-cholinergic side
effects than other tricyclic antidepressants. Nevertheless,
all anti-cholinergic drugs produce smooth muscle relaxa-
tion which leads to bowel stasis and therefore may
predispose to pseudo-obstruction. This is a relatively rare
complication and is usually an idiosyncratic reaction.

Mecagol and dilatation of the small bowel have been
described in patients with Parkinson’s disease not taking
anticholinergic medication. Contributory factors in park-
insonian patients include disorder gut motility with
generalized hypokinesia, delayed gastric emptying aggra-

vated by levodopa, autonomic dysfunction and aggra-

vation of constipation by anismus. Furthermore, Kupsky et al. have shown the presence of Lewy inclusion bodies
in myenteric plexus ganglion cells in the oesophagus and
colon in a patient with megaloclon and Parkinson’s
disease. This may reflect primary involvement of the
toenteric nervous system by degenerative processes in
Parkinson’s disease. Thus it can be seen that disturbances
of alimentary function are a prominent feature in patients
with Parkinson’s disease and may make these patients
particularly vulnerable to the effects of anti-cholinergic

drugs on the bowel.

This case therefore illustrates the difficulty in treating
patients with Parkinson’s disease with anti-cholinergic
drugs. Particular care is advised, when prescribing them
to patients with Parkinson’s disease.

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References