had encouraging results with supratrigonal vesicostomy in the management of a carefully selected group of these hypersensitive bladders.

The longest follow-up is now nearly five years, but the immediate clinical result in most of them has been so striking that it seems probable that this relatively simple operative procedure offers a very fair chance of symptomatic relief, at least for a reasonable period. If this is so, it would certainly have a clear advantage over both long-term steroid therapy and repetitive bladder distension, and the details of our series will be presented to the British Urological Association meeting in July.

Secondly, we would like to express some degree of disagreement with the statement in your leader that the results of cystoplasty for patients with interstitial cystitis are disappointing. In our experience it has offered the great majority of patients a reasonably normal restoration of their bladder function.

With regard to cystoplasties in general, it is important to appreciate that bowel peristalsis is not an adequate replacement of the vesical detrusor. Selective relaxation of the sphincter mechanisms is not infrequently required to overcome the relative outflow obstruction that results from this iatrogenic voiding imbalance, and the complications of residual urine, infection, stone formation, and pyelonephritis are usually avoidable by a proper attention to postoperative supervision and outflow rebalancing. In our series, cystoplasty failure rate is about 10%; mostly patients in whom it proves impossible to establish a balance between voiding efficiency and continence. The great majority of the remaining 90% void at two to three hourly intervals with reasonably normal urinary control and bladder sensation.

When a cystoplasty is performed for interstitial cystitis it is particularly important to ensure that the hypertensive bladder is resected almost to the margins of the trigone, in order to reduce the chance of persistent bladder pain and hypersensitivity.—We are, etc.,

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SIR,—Your leading article on "Interstitial Cystitis" (11 March, p. 644) gives an excellent summary of its main features, but omits a method of treatment which I believe offers great promise. I refer to denervation of the bladder, a procedure which is of increasing interest to urologists.

Interstitial cystitis is rare; consequently I have myself had the opportunity of treating only one patient by denervation. This is a woman of 56 who presented three years ago with a classic Hunner's ulcer. Dilatation afforded increasingly less relief of her symptoms, and a course of prednisone had no effect. She reached a point where she agreed to urinary diversion, and as I thought that, theoretically at least, denervation should help her condition, I proposed this as an alternative, and pointed out to her the experimental nature of the procedure. Denervation was performed in August 1971. Within 24 hours of operation she declared herself symptomatically improved, and has

had no urinary symptoms of any sort since that time. At cystoscopy a fortnight ago—seven months postoperatively—her bladder capacity had increased from 160 ml pre-operatively to 500 ml. There is no residual urine. The ulcer has healed and the bladder looks normal.

The technique of denervation was briefly described by Turner-Warwick in another context.¹ My colleague Mr. W. Keith Yeates and I have used this operation now on a number of patients and we have developed a simple and satisfactory operative technique. Only one patient with interstitial cystitis has so far had a bladder denervation; the remainder have suffered from a variety of symptoms arising from hyperactivity of the detrusor presumably of neurological origin.

Our results so far have been excellent, and will be submitted for publication at a later date. The operation consists of circumferential supratrigonal division of the bladder with complete disruption between the two parts. Normal anatomy is restored by cysto-cystoplasty. This causes motor denervation of the supratrigonal detrusor. It is easy enough to formulate a hypothesis to explain the effect of this operation on Hunner's ulcer, but as yet there is no proof. So far as I am aware, supratrigonal motor denervation has not yet been described in the treatment of Hunner's ulcer.

Admittedly one patient is not conclusive evidence. In this instance, the result has been so impressive that I shall certainly consider denervation at an early stage in future patients suffering from interstitial cystitis.—I am, etc.,

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¹ Turner-Warwick, R. T., and Ashken, Handley M., *British Journal of Urology*, 1957, 39, 3.

Papillary Necrosis in a Transplanted Kidney

SIR,—Dr. R. P. S. Edmondson and others' report (26 February, p. 547) prompts us to record a similar case, in which the association between rejection and papillary necrosis was conspicuous.

A 26-year-old man, on our maintenance haemodialysis programme since 1969 for end-stage glomerulonephritis, received a kidney transplant on 22 October 1970. The donor was a man, aged 42, who died of brain damage. The recipient was not diabetic. Blood groups of donor and recipient were identical. HL-A compatibility studies disclosed two identities and two potential incompatibilities. There were no detectable preformed cytotoxic antibodies against the donor's lymphocytes in the recipient's serum. Total ischaemia time was 225 minutes (cooling with Collins's solution) and the surgical procedure was uneventful. Initial treatment consisted of prednisone 1/3 mg/kg and azathioprine 3 mg/kg. A brisk diuresis followed declamping (300 ml/min) but the urine output soon started to decline: 125 ml/hr at 24 hr, then 80 ml/hr at 48 hr. Pain and fever together with progressive anuria led us to increase the dosage of prednisone to 2 mg/kg without improvement. On the seventh day the patient passed a piece of soft tissue, without pain or haematuria. Histology disclosed the typical aspect of necrotic renal papilla. The following day the patient was anuric. Percutaneous

renal biopsy yielded a black piece of infarcted kidney tissue.

Nephrectomy was done. The renal vessels and the ureter were patent. The whole surface of the swollen kidney was mottled with black and reddish areas. Section disclosed scattered foci of necrosis in the cortex, predominating in the corticomedullary junctions. Most of the papillae were necrotic, and several had sloughed off.

Histology showed a necrotic cortex, invasion of the interstitium by erythrocytes and round cells, and tubular necrosis. Intrarenal vessels were mostly normal. The medulla was destroyed by interstitial haemorrhage. No fixation of antisera was detected on the glomeruli. Using immune fluorescent techniques, round cells fixed anti-IgM antiserum. Most of the peritubular capillaries fixed the antifibrinogen serum.

In our opinion, this case demonstrates that papillary necrosis can indeed be due to acute rejection.—We are, etc.,

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Suction Retractor

SIR,—Mixed parotid tumours may be difficult to enucleate, since conventional tissue-holding forceps tear out when any tension is exerted on them. Similar problems may also be encountered with branchial cysts or malignant nodes. It may not be possible to manipulate the tumour so that its under surface can be adequately visualized.

The suction retractors (Fig.) are made up in sets of three cup sizes. The cup is applied directly to the tumour, in place of tissue forceps. In practice the middle size of cup has been found adequate for most situations. The other end of the instrument is fitted into the sucker tubing. The amount of suction can be increased by covering the hole at the side of the adaptor fitting.

These retractors, known as "Burke's Trum-pets," may be obtained from Peacocks Surgical Medical Equipment Ltd., Newcastle upon Tyne.—I am, etc.,

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Islets of Munchausen

SIR,—In his article on spontaneous hypoglycaemia (12 February, p. 430) Professor Vincent Marks did not discuss self-induced hypoglycaemia—no doubt because this type is neither spontaneous nor common. A young man suffering—if this is the correct word—from this condition was admitted to the