High pressure chronic retention

N J R GEORGE, P H O'REILLY, R J BARNARD, N J BLACKLOCK

Abstract

In a prospective study of high pressure chronic retention, a newly defined disorder of the urinary tract, 21 patients were analysed for their mode of presentation and urodynamic, renographic, and operative findings.

High pressure chronic retention is characterised by late onset enuresis, a tense, palpable bladder, hypertension, and progressive renal impairment associated with bilateral hydronephrosis and hydroureter commonly leading to uraemia and death. Obstructive urological symptoms are typically absent in uncomplicated cases.

The study confirmed that patients with the disorder are commonly elderly and present, not necessarily to a urologist, with late onset enuresis or symptoms of cardiac decompensation. After transurethral resection the patients, if correctly managed, may be expected to make a satisfactory recovery.

Introduction

Chronic retention of urine (the painless retention of urine within the bladder after micturition) may occur in both the young and elderly. It may be associated with a large prostate or almost no prostatic tissue at all. The bladder wall may be massively hypertrophied or so thin as to be transparent. Uraemia may rapidly ensue or the patient may survive for years without reduction in creatinine clearance—despite bladder capacities of several litres. Symptoms of outflow tract obstruction may be either severely incapacitating or entirely absent. Mitchell¹ investigated this problem and recognised that there might be two broad types of chronic retention. His observation was complemented by others,² who reported an apparent association between upper tract dilatation and a "systolic" or high pressure inflow limb on cystometry in some cases.

We have followed up these initial clues by defining carefully the exact characteristics of what has emerged as a highly homogeneous group now described as having "high pressure chronic retention." These patients are so defined owing to the presence of a raised intrinsic detrusor pressure during the *filling phase* of micturition and thus are clearly distinguished from patients with "low pressure" chronic retention, whose intrinsic detrusor pressure during filling is "normal"—that is, equal to general intra-abdominal pressure.

Patients and methods

The study was entirely prospective. After notification of a suitable patient attending the outpatient clinic a full history was obtained and

examination performed by one of us. Serum electrolyte and creatinine concentrations were estimated and patients with a blood urea value exceeding 20 mmol/l (120 mg/100 ml) excluded from the study and admitted immediately for catheterisation. The remaining patients were investigated as outpatients, the tests being arranged in an orderly fashion over 10–14 days before admission. Urine frequency and volume charts were completed by patients at home before these investigations.

Urography was performed in routine manner, the patient micturating immediately beforehand. Tomography was used to detail the caliceal pattern when necessary. Abdominal compression was avoided and the bladder not drained during the examination. Baseline bladder pressure was measured by direct suprapubic puncture, after which medium fill water cystometry was performed as described.³ Particular attention was paid to constancy of measurement, and to allow the bladder to return to full state equilibrium no other investigations were undertaken within 48 hours.

Renographic studies were performed using ¹²³I-labelled Hippuran (sodium iodohippurate) and an Elscint Dymax LF gammacamera-computer system.⁴ When indicated repeat suprapubic inflow cystometry was performed at the time of renography. Reflux studies were performed by direct suprapubic injection of isotope into the bladder.

After admission to hospital creatinine clearance estimations were made before elective transurethral resection of the prostate. The surgeon's comments on the bladder and outflow tract were noted. Post-operative fluid balance, blood pressure (erect and supine), and serum and urine electrolyte concentrations were monitored, and when satisfactory equilibrium was reached the patient was discharged from the ward.

Results

CLINICAL FINDINGS

Four cardinal criteria dominated the clinical presentation. These were late onset enuresis, a tense, painless bladder, hypertension and bilateral hydronephrosis with hydroureter, commonly leading to progressive uraemia. Out of 21 patients, 18 had three or more of these criteria at the time of initial consultation. Only three patients were aged under 65 (range 25–81 years, mean 69).

The symptom of late onset enuresis was typically a small leakage (10–20 ml) occurring soon after falling asleep, the patient invariably being awakened immediately by the feeling of dampness. Such a leakage could also happen during an afternoon nap in a chair but never occurred when the patient was awake. Hence the word enuresis is taken literally as "unwanted leakage of urine." None of the patients had typical urge enuresis as recorded in younger persons, and no patient in the series had been enuretic as a child.

On examination all 21 patients were found to have a tense, painless bladder, though only a few had noticed the swelling for themselves—a fact made more remarkable by their generally slim build. The suprapubic swelling, of almost uterine consistency, was central and symmetrical in all but two patients, in whom a diverticulum was noted extending to the right. In relation to these first two criteria, four of the nine patients without outflow tract symptoms (see below) had been brought to their practitioner by their wife or a near relative because of either the extra laundry load or alarm at the need for new clothes to accommodate the patient's enlarging waistline.

Hypertension was found in 14 patients on presentation. In all except those with advanced renal failure the raised pressure fell to normal almost immediately after catheterisation. Heart failure, ankle oedema, and paroxysmal nocturnal dyspnoea were additional reasons for initial referral (the swollen bladder passing unnoticed), and these symptoms also settled rapidly (less than 24 hours after catheterisation).

Raised serum creatinine concentration was found in 17 patients but in only two advanced cases was this the reason for medical referral. Bilateral hydronephrosis and hydroureter, present in almost every case, preceded the uraemia by a variable time interval.

Twelve patients presented with typical symptoms of an obstructed outflow tract. In nine cases, however, complaints of hesitancy, poor stream, and increasing frequency were almost completely absent, the

Salford Royal Hospital, Salford M60 9EP

N J R GEORGE, FRCS, senior registrar and lecturer in urology

Stepping Hill Hospital, Stockport SK2 7JE

P H O'REILLY, MD, FRCS, consultant urological surgeon

University Hospital of South Manchester (Withington Hospital), Nell Lane, Manchester M20 8LR

R J BARNARD, FRCS, consultant urological surgeon

N J BLACKLOCK, FRCS, professor of urology

Correspondence to: Mr N J R George.

frequency and volume charts providing objective confirmation of satisfactory voided volumes and little frequency.

There were few other abnormal findings on examination. Size of the prostate was difficult to assess. Routine neurological examination showed nothing abnormal, and there were no cases of back injury or sciatica in the series.

URODYNAMIC OBSERVATIONS

The resting end-void subtracted bladder pressure (baseline filling phase pressure)—that is, the intrinsic pressure within the bladder at the end of micturition when the bladder is supposedly empty—was raised in all patients (12.5–35.0 cm H₂O, mean 24.5). Figure 1 shows the relation between intrinsic subtracted detrusor pressure and serum creatinine concentration in patients with sterile urine. No patient was aware of the residual urine associated with this abnormal pressure. Mean residual urine volume was 1480 ml (range 475–2100 ml). Subsequent inflow cystometry caused a rapid rise (systolic inflow limb) in subtracted detrusor pressure, which quickly reached the micturition threshold producing a desire to void. In no case did the bladder capacity on repeat inflow cystometry exceed 300 ml despite predrainage capacities as noted above recorded only a few minutes earlier.

FIG 1—Relation between end-void subtracted detrusor pressure and serum creatinine concentration in 14 patients with cell free sterile urine.

Conversion: SI to traditional units—Creatinine: 1 $\mu\text{mol/l} \approx 0.01 \text{ mg/100 ml}$.

UROGRAPHY

Preoperative urography showed bilateral hydronephrosis and hydro-ureter in all but the earliest cases (fig 2). Cortical thickness was characteristically well preserved until late in the disorder. Ureteric dilatation appeared to begin at the lower end and proceed proximally.

RENOGRAPHY

Gammacamera renography was possible before operation in 16 patients, all of whom showed an obstructive excretory pattern. Uptake of tracer was variable but generally good in all except patients with advanced renal failure (fig 3). Ureteric reflux, either passive or active during micturition, was not detected.

OPERATIVE FINDINGS

At cystourethroscopy the bladders were found to be grossly trabeculated (fig 4). Residual urine volumes matched urodynamic predictions, but those patients who had been catheterised before operation had noticeably shrunk bladders, making transurethral resection difficult. Although variable weights of tissue were resected, opinion was that the obstructing element was chiefly at the bladder neck, which was usually massively hypertrophied—that is, over 1 cm thick when felt through the rectum against the cystoscope. Despite some moderate resections the full clinical expression of high pressure chronic retention was noted even in patients requiring resection of as little as 10 g of the bladder neck.

POSTOPERATIVE OBSERVATIONS

Patients with high pressure chronic retention exhibited a typical salt and water losing state after operation. The degree of fluid imbalance generally correlated with the preoperative reduction in renal function.

FIG 2—Urographic findings. Top: Renal tomogram. Bottom: Typical ureteric appearances of tortuosity and hold up at ureterovesical junction.

The typical (and expected) symptoms and signs of such fluid loss were postural hypotension, lethargy, and rise in blood urea concentration. Most patients required substantial intravenous fluid and electrolyte replacement to counter these losses, and few could be discharged before the eighth postoperative day.

Discussion

Using clinical criteria, Mitchell^{1, 5} described two broad groups of patients with chronic retention of urine characterised by the presence or otherwise of a raised intrinsic pressure within the bladder. He noted that patients with high pressure, though frequently without symptoms, had noticeably trabeculated bladders, often associated with upper tract dilatation. The symptom of "late onset" enuresis, as defined, may be mistaken for overflow incontinence (near-continuous urinary loss occurring by day and night) if discriminating inquiry is not made. Such leakage occurring soon after falling asleep was found in this series to be highly specific for high pressure chronic retention.

FIG 3—Iodine-123 gammacamera renogram in patient with normal serum creatinine concentration. Frusemide 0.5 mg/kg given intravenously at 15 minutes (arrow). Results expressed as percentage uptake of isotope.

FIG 4—Typical endoscopic appearance. (Picture shows area 1 cm above right ureteric orifice.)

Studies of serial urograms suggest that the first sign of high pressure chronic retention is a gradual asymptomatic enlargement of the bladder. Later slight dilatation of the lower end of the ureter is followed by dilatation of the renal pelvis. Finally, as the intrinsic detrusor pressure continues to rise full hydro-ureter and hydronephrosis develop, leading eventually in most cases to progressive renal impairment. Normal creatinine concentrations may be maintained despite massive dilatation of the upper tract, so that a raised concentration indicates an advanced stage of the disorder. All of our patients were men and commonly elderly. We do not know whether the condition occurs in women or whether it cannot progress owing to the relative inability of the female sphincter mechanism to withstand high intravesical pressures.

In our series symptoms of bladder outflow tract obstruction were commonly associated with the pathological diagnosis of either carcinoma of the prostate or supra-added infection of the residual urine. All five patients later found to have carcinoma of the prostate presented with typically obstructive symptoms,

not necessarily of sudden onset. Of 13 patients with histologically benign prostatic tissue, those with supra-added infection (three cases) presented in such a manner to a urologist. Eight out of 10 patients with cell free sterile urine did not complain of obstructive symptoms, even under direct questioning. Symptoms were precipitated in one of the remainder by aggressive diuretic treatment for cardiac decompensation. This important point, supported by objective frequency and volume charts, is emphasised by the fact that four of these asymptomatic patients were referred either to physicians complaining of thirst, ankle oedema, or hypertension or to general surgeons with an abdominal mass.

The essential urodynamic observation was a raised intravesical pressure throughout the filling phase of the micturition cycle. It seems reasonable to presume that the upper tract characteristics were related to this raised pressure. Counterbalancing this observation was the equally important and perhaps interrelated fact that the higher centres were quite unable to sense the presence of the overfull bladder. We cannot comment on the neurological implications, but evidently (fig 1) there is a relation between end-void intrinsic detrusor pressure and the serum creatinine concentration in patients with sterile urine. Early cases have a low intrinsic pressure but as the pressure rises through an apparently critical 20-30 cm H₂O barrier uraemia rapidly supervenes.

Our findings suggest that most cases of high pressure chronic retention progress at a variable rate to uraemic death. The data indicate that the commonly advanced hypothesis that the thick walled bladder of high pressure retention will in time "give way" and lead to the thin walled bladder of low pressure chronic retention (normal urea and creatinine values) is most unlikely to be true. "High" and "low" pressure disorders seem at present to be discrete entities.

The relation of high pressure chronic retention to obstruction of the outflow tract is a matter for debate. Patients with severe infravesical obstruction (urethral stricture or valves) may undoubtedly present with symptoms and signs very similar to those exhibited by patients with high pressure retention. It is also true, however, that the many patients who present to urologists with unequivocal and urodynamically proved outflow tract obstruction related to prostatic hypertrophy do not have the cardinal characteristics of high pressure retention. These patients generally present for surgery because of increasingly severe outflow tract symptoms long before residual urine volumes approach those of high pressure retention, and upper tract dilatation is very rare in such cases.¹ In our study admissions for high pressure retention probably constituted less than 5% of urological admissions for "typical" obstructive outflow tract problems.

The critical factors governing the development of this type of bladder response remain unknown; we cannot state with certainty that high pressure chronic retention is a consequence of or directly related to infravesical obstruction.

Initially patients with this syndrome are likely to present to their general practitioners. The absence of typical obstructive symptoms in many cases (see above) will mean that the grossly enlarged bladder may pass unnoticed and the patient will be referred to a physician for a second opinion regarding hypertension, heart failure, confusion (due to uraemia), or incontinence. The presence of any of the cardinal criteria, and "late onset" enuresis in particular, should alert the clinician to the true nature of the abnormality.

On admission urgent blood urea, creatinine, and electrolyte estimations should be requested and catheterisation avoided until the results are obtained. A suprapubic or urethral catheter is not only unnecessary if the blood urea concentration is under 20 mmol/l (120 mg/100 ml) but may cause positive harm by introducing infection into a dilated and obstructed urinary tract. This risk must be accepted in patients with concentrations exceeding 20 mmol/l in order to prevent further renal damage.

It is commonly taught that decompression of the bladder should be performed slowly in such cases of chronic retention to minimise haemorrhage or tubular necrosis. Further studies on these patients have shown us that, owing to the physical

characteristics of the high pressure bladder, removing even a small (<100 ml) volume of urine cannot prevent a rapid drop in intravesical pressure with consequent brisk onset of the diuretic phase, which may precipitate some bleeding from either lower or upper urinary tract. We find it impossible to regulate the decompression process with enough precision in the ward and prefer instead to concentrate on the problem of the often mild but occasionally severe salt losing state that sets in after the obstruction has been removed.

Operation is generally by transurethral resection. Bimanual examination under anaesthesia may be very misleading in these patients, as the prostatic capsule may be extremely muscular (greater than 1 cm thick), deceiving the operator and making enucleation difficult if an open procedure were to be chosen.

Postoperatively careful monitoring of weight, blood pressure (erect and supine), and electrolyte values is required. It must be emphasised that diuresis induced dehydration may ensue with great rapidity in these patients, and a rising urea or creatinine concentration after operation is as likely to be due to this cause as to the readily blamed "residual obstruction." Depending on the nephron damage, such losses may continue for many days post-

operatively, though not necessarily to such a severe degree. Before discharge an estimation of creatinine clearance will allow nephron recovery to be followed with precision. Follow up of our patients at one year showed that initial clinical progress was being well maintained.

References

- Mitchell JP. *Principles of transurethral resection and haemostasis*. Ch 18. Bristol: John Wright, 1972.
- Abrams PH, Dunn M, George NJR. Urodynamic findings in chronic retention and their relevance to results of surgery. *Br Med J* 1978;iii: 1258-60.
- George NJR, Slade N. Hesitancy and poor stream in younger men without outflow tract obstruction—the anxious bladder. *Br J Urol* 1979;51: 506-10.
- O'Reilly PH, Shields RA, Testa HJ. *Nuclear medicine in urology and nephrology*. London-Boston: Butterworths, 1979.
- Mitchell JP. MS thesis. University of London, 1955.

(Accepted 31 March 1983)

SHORT REPORTS

Measurement of respiratory rate in the newborn

Infants who appear normal at birth are transferred to postnatal wards where twice daily measurements of temperature are usually the only regular nursing observations. During the past 10 years more infants have been admitted to postnatal wards instead of special care baby units to avoid separation from their mothers. We found that there was no satisfactory method of observing these babies when four apparently normal infants were found dead in their cots in one postnatal ward nursery between October and December 1976. All these deaths occurred in the early hours of the morning and the nursing staff were terrified of starting the night shift for fear of a further death. No cause of death was found despite extensive investigation of the circumstances and postmortem examinations. At the time Lancefield group B streptococci were being recognised as a cause of rapidly progressive disease in our unit.¹ As it is the most common pathogen in the newborn and usually causes a raised respiratory rate² we decided to assess the value of regular measurements of respiratory rate to detect occult disease.

Patients, methods, and results

From January 1977 the respiratory rates of all infants in the postnatal wards have been recorded hourly for the first 12 hours and two hourly for the subsequent 24 hours. Initially the respiratory rate was counted by observation, which was not reliable, or by the nurse placing her hand on the baby's chest, which disturbed the infant. A new method of detecting respiration was therefore developed,³ in which an air filled capsule was taped to the abdominal wall so that expansion of the abdomen compressed it and generated a pneumatic signal. This signal was carried along a flexible tube to a small (12×5×2 cm) hand held box in which it was converted to an electrical and then audible and visible signal. The sensor capsules were very simple and cheap, so that one could be attached to each baby shortly after birth, allowing the end of the tube to protrude from the cot. Each ward was supplied with a box, which could be carried round by a nurse and connected to each baby in turn for counting the respiratory rate. Analysis of the respiratory rates in 54 infants admitted consecutively to one postnatal ward showed that the highest rate (mean 48±SD 6/min) was at the age of 3 hours. Infants with a respiratory rate above 60/min persisting for more than one hour were transferred to the special care unit.

During February 1981 to February 1982, 2789 infants were admitted to the postnatal wards, and 29 of them were transferred to the special care unit with a raised respiratory rate (table). Four had evidence of colonisation or infection with group B streptococci and one had heart failure due to hypoplastic left heart syndrome. Nine had a raised respiratory rate for only a few hours and were given no treatment, but the remaining 19 had a raised respiratory rate for a considerable period and received antibiotics.

Comment

Use of the new respiratory rate detector has shown that about 1% of apparently normal infants in the postnatal wards have a raised respiratory rate and that in most of them it persists for a long period. Most of the 19 infants without reported evidence of group B streptococcal infection probably had delay in lung fluid absorption. No investigation is helpful in confirming this diagnosis and the respiratory rate falls to normal spontaneously. Despite the negative reports some of the 19 infants may have had group B streptococcal infection, as routine laboratory methods do not identify all cultures of this organism.

Group B streptococcal infection has a mortality rate between 20 and 50%¹ and early treatment is the most important factor in prognosis.⁴ It has been suggested that the mortality from this infection can be reduced only by giving antibiotics prophylactically to all infants in a unit.⁵ We consider that it is preferable to select a group of infants for

Details of infants with raised respiratory rates who were transferred from postnatal wards to special care unit

Case No	Birth weight (g)	Weeks of gestation	Age (hours) when raised respiratory rate noted	Duration (hours) of raised respiratory rate	Group B streptococci cultured from*:		Treatment
					Mother	Infant	
1	2680	39	3	6	0	0	Nil
2	3500	40	1½	5	0	0	Nil
3	2800	39	3	35	0	Neg	Nil
4	2820	38	2½	10	0	0	Nil
5	3760	40	2	72	0	Neg	Antibiotic
6	3100	40	8	6	Neg	0	Nil
7	2980	40	6	120	0	Neg	Antibiotic
8	3480	41	29	15	Pos	Pos	Antibiotic
9	3260	41	26	19	Neg	Neg	Nil
10	2980	41	32	3	0	Neg	Nil
11	3700	41	12½	136	0	Neg	Antibiotic
12	3820	40	4	23	0	Pos	Antibiotic
13	3240	37	3½	8	Neg	0	Nil
14	3150	38	9	55	0	Neg	Antibiotic
15	4110	39	28	35	0	Pos	Antibiotic
16	3280	40	5	27	0	Neg	Antibiotic
17	3140	40	8	72	0	Neg	Antibiotic
18	3880	41	5	48	0	Pos	Antibiotic
19	2700	39	23	29	0	Neg	Antibiotic
20	3420	40	14	30	Neg	Neg	Antibiotic
21	3660	41	5½	72	0	Neg	Antibiotic
22	3010	39	12	50	0	Neg	Antibiotic
23	3860	40	24	168	0	Neg	Antibiotic
24	3530	41	22½	96	Neg	Neg	Antibiotic
25	3410	40	24	†	Neg	Neg	Antibiotic
26	2480	36	2	17	Neg	Neg	Antibiotic
27	3480	39	8½	17	Neg	Neg	Antibiotic
28	2950	38	4	7	0	Neg	Nil
29	2800	34	2	66	0	Neg	Antibiotic

* 0 = No investigation.

† Hypoplastic left heart syndrome.