rather than two underfunded and often ailing quangos we need a powerful office in central government with ramifications throughout the NHS and other bodies.

In the mean time, what can we expect from Dr Catford in Wales? The answer is not much in five years. It took 10 years to produce a fall in mortality from heart disease in North Karelia, and it will probably take as long in Wales. Professor Catford should not be thrown out if he has not produced the goods in five years. Let us hope, indeed, that in five years' time government, professional, and public commitment backed up by ample funds will see us well into what's been called the "second public health revolution."

1 Anonymous. Policies on prevention. *Br Med J* 1984;**288**:1182.
2 Anonymous. New thoughts for the Health Education Council. *Br Med J* 1984;**285**:1761-2.
3 Bailey BH, Player DA. New thoughts for the Health Education Council. *Br Med J* 1983;**286**:226.
4 McCron R. New thoughts for the Health Education Council. *Br Med J* 1983;**286**:645-6.

Regular Review

Management of chronic urinary retention

J P MITCHELL

In chronic urinary retention the residual urine remaining in the bladder after micturition has reached a volume equal to or greater than the normal bladder capacity. In acute retention the amount of urine withdrawn on catheterisation of the patient will be around 500 to 600 ml, but in chronic retention it will be 800 ml or more. Patients with chronic retention most commonly present with a bladder content of something between 1000 and 1500 ml but volumes above four litres have been described.[1] The patient can still pass urine, but this large residue is left in the bladder after micturition.

Chronic urinary retention may be so insidious in onset that it is completely symptomless in the early stages. The patient may have no sense of incomplete emptying, and his volume of micturition does not produce any frequency. His bladder becomes insensitive so that he has no indication that it is already distended beyond its normal capacity. The patient may develop symptoms only when the first episodes of retention with overflow produce enuresis. In fact any form of incontinence in a man should alert the clinician to the possibility of chronic retention with overflow. Alternatively, patients with chronic retention may present with renal failure due to back pressure on the urinary tract. The patient may be losing weight and his appetite may diminish at the same time as he may become conscious of a slight increase in girth. The third possible presentation for chronic retention is when acute retention supervenes—the patient in chronic retention who has been passing urine suddenly finds that he is unable to do so. This state of acute on chronic retention differs from true acute retention in three respects. Firstly, the amount of pain is disproportionate to the size of the bladder—the patient does not appear to be in the intense discomfort that his distended bladder would be expected to produce. Secondly, the volume of urine in the bladder is greater than would be expected in straightforward acute retention. Thirdly, the inability to pass urine may last considerably longer than in simple acute retention. Patients with acute on chronic retention may even give a history of inability to pass urine for two days.

The distinction between straightforward acute retention and acute on chronic retention is important from the point of view not only of diagnosis but also of the prognosis and subsequent management. Chronic retention takes two forms, which are quite distinct in their clinical presentation. The distinguishing feature is the intravesical pressure.[2] In high pressure chronic retention the pressure in the bladder is usually above 30 cm H_2O and the usual cause is obstruction to the urinary outflow or incoordination of the detrusor and sphincter mechanism. In low pressure retention the intravesical tension is less than 20 cm H_2O and the cause is usually failure of the detrusor muscle. High pressure retention may exert back pressure on the upper urinary tract, but the low pressure bladder, in total contrast, by its complete failure of the detrusor will protect the upper urinary tract from any back pressure.

Why some patients develop low and others high pressure retention remains one of the mysteries of urology, and why one patient with obstruction should develop acute retention and another chronic retention is also ill understood. As for the low tension bladder, the only similar condition in any other system in the body is Hirschsprung's disease of the colon, but no depopulation of neuromuscular synapses has been found to explain the failure of the detrusor muscle. The neurogenic bladder seen in spina bifida, in patients presenting with transverse myelitis, or in acute transection of the cord in spinal injury invariably produces the features of chronic retention, usually of the high pressure type. Incontinence from failure of the sphincter mechanism is an uncommon neurological disorder, the usual form of incontinence seen in patients with neurogenic bladders being the occasional overflow incontinence, most frequently noticed in those with spina bifida. Although the results of urodynamic investigations can distinguish between mechanical obstruction of the lower urinary tract and the incoordination of a dyssynergia of the sphincter mechanism, these studies have failed to throw any light on the reason why some patients should develop a high pressure chronic retention.

The complications of chronic retention are principally those of urinary stagnation. The risk of infection is increased and it may become rampant shortly after it is established, with the consequent probability of spread to the upper urinary tract. The second risk is of back pressure on ureters and kidneys. Necessarily the chance of damage to the upper urinary tract is considerably greater in high pressure chronic retention.

Establishing the diagnosis in high pressure chronic retention is not difficult. On abdominal examination a tensely distended mass will be found in the midline suprapubically. Low pressure chronic retention may be a more elusive diagnosis, in that the outline of the bladder will be only vaguely palpable because of the flaccid distension. A dull area to percussion will be evident in the suprapubic region, but at best the clinician will only be suspicious that the bladder is not emptying completely. The first investigations should be determination of the serum concentrations of urea and creatinine to exclude any renal failure, which might vitiate the results of intravenous pyelography. Provided the blood urea concentration is below 30 mmol/l, an intravenous pyelogram should show the state of the upper urinary tract and confirm the distension of the bladder.

Catheterisation of these patients often induces an episode of haematuria and in some instances this may be heavy. Bleeding which occurs within an hour or two of emptying the bladder will almost certainly be caused by the sudden hyperaemia which develops in the bladder mucosa from the large veins, which become grossly distended as a result of the release of pressure.[3] If the distended bladder wall is viewed by endoscopy during emptying and before the release of any urine a sudden engorgement of vessels with numerous petechial haemorrhages will be seen, provided the urine is clear and not too concentrated. A second type of haematuria that may occur 24 to 48 hours after catheterisation is due to acute urinary tract infection ascending to produce an acute pyelonephritis. It was to reduce the risk of haematuria that urologists considered that the bladder should be decompressed slowly in all patients with chronic urinary retention. In theory slow decompression should relieve the sudden engorgement of the bladder mucosa and the development of petechial haemorrhages. In practice, however, slow decompression is extremely difficult to achieve: the first few ml of urine withdrawn from a bladder which is totally inelastic will reduce the pressure at a considerable rate.[4] (An analogy may be made with a simple urine drainage bag which fills to capacity with the pressure no more than the height of the fluid within the bag. If further fluid is forced in under pressure only a small amount will raise the pressure within the bag dramatically. Similarly, withdrawal of this very small amount will relieve the pressure in the bag immediately.) The traditional slow decompression of the bladder by gradually releasing a gate clip on the drainage tube, or by removing small quantities of urine at regular intervals, does not achieve its aim of gradual reduction in intravesical pressure. Only one method can be considered and that is an inverted U tube (ventilated at the apex to avoid siphon action) in the drainage system, suspended at a height corresponding to the bladder pressure.[3,5] In theory this system appears simple to manage, with the tube being lowered gradually 2 or 3 cm at a time until the bladder is empty, but in practice it may not be easy to control. The plug of cotton wool in the ventilating outlet of the U tube may become sodden with any movement of the patient which increases his abdominal pressure, when immediately the U tube creates a siphon draining the bladder.

Nevertheless, textbooks of urology still advocate slow decompression, not only to prevent vesical haemorrhage but also to reduce the risk of tubular necrosis of the kidney.[6,7] Yet George and his coworkers have shown from isotope renography that there is a dramatic change in isotope washout from the upper urinary tract as the bladder pressure diminishes, and there seems no justification for delaying this improvement by slow decompression.[4] Furthermore, slow decompression carries with it one additional disadvantage—the increased risk of urinary infection, which may be rampant from the moment of catheterisation, particularly if stagnant urine continues to remain at high pressure. The postobstructive diuretic state which develops immediately the urinary tract is decompressed may be controlled by intravenous fluids with a careful watch on the biochemical homoeostasis.

The conclusion must be, therefore, that slow decompression is rarely indicated for patients with high pressure chronic retention and never for those with low pressure chronic retention. In the occasional patient for whom the clinician decides on slow decompression it must be carefully controlled by carefully managed manometric pressure release. In most patients with high pressure chronic retention preliminary drainage should be carried out under careful bacteriological control as well as chemical balance. Surgery to the obstruction can then be performed when the maximum improvement has been achieved in the upper urinary tract and before any infection develops. In patients with low pressure chronic retention no preliminary drainage of the bladder will improve the detrusor action, and definitive surgery to the outflow obstruction (or if no obstruction exists to reduce outflow resistance) should be the first urethral intervention.

If correctly managed, high pressure chronic retention has a good prognosis after transurethral resection.[8] In low pressure chronic retention, however, the prognosis is far less satisfactory as recovery of the detrusor is unlikely, and the patient should be warned accordingly. A regimen of micturition by the clock—discouraging the patient to wait until he has the desire to pass urine—is the only hope of achieving complete or almost complete emptying of the bladder.

J P MITCHELL

Honorary Professor of Urology,
Bristol BS9 1AW

[1] Young TW, Mitchell JP. Distension of the bladder leading to vascular compression and massive oedema. *Br J Urol* 1968;40:248-50.
[2] Mitchell JP. Estimation of intravesical tension in the retained bladder. The diagnosis and treatment of congenital bladder neck obstruction. Appendix 1. London: University of London, 1955. (Thesis.)
[3] Mitchell JP. Preoperative preparation of the patient. In: *Endoscopic operative urology*. Bristol: Wright, 1981:259-90.
[4] George NJR, O'Reilly PH, Barnard RJ, Blacklock NJ. The practical management of patients with dilated upper tracts and chronic retention of urine. *Br J Urol* 1984;56:9-12.
[5] Mitchell JP. Closed drainage of the bladder. In: *Urology for nurses*. Bristol: Wright, 1980:271-85.
[6] Blandy JP. Benign enlargement of the prostate gland. In: Blandy JP, ed. *Urology*. Oxford: Blackwell, 1976:889-90.
[7] Philip PF. The prostate and seminal vessels. In: *Bailey and Love short practice of surgery*. London: Lewis, 1977:1239-50.
[8] George NJR, O'Reilly PH, Barnard RJ, Blacklock NJ. High pressure chronic retention. *Br Med J* 1983;286:1780-3.