UNIVERSITY OF ALBERTA

The Effect of Transurethral Resection of the Prostate on Detrusor Instability in Elderly Men with Benign Prostatic Hyperplasia

BY

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A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science.

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled THE EFFECT OF TRANSURETHRAL RESECTION OF THE PROSTATE ON DETRUSOR INSTABILITY IN ELDERLY MEN WITH BENIGN PROSTATIC HYPERPLASIA submitted by Elizabeth Ann Gormley in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE in Experimental Surgery.

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DEDICATION

To my parents,
Michael and Helen Gormley,
for their unfailing support and encouragement.

ABSTRACT

Detrusor instability is a common finding in men with evidence of outflow obstruction. Detrusor instability reverses in approximately two-thirds of younger men post-transurethral resection of the prostate (TURP). Detrusor instability is a common finding in the elderly without outflow obstruction and its incidence increases with increased age. Detrusor instability is the cause of urge incontinence. In elderly men detrusor instability may be due to obstruction or it may be related to age. To determine if TURP has an effect on detrusor instability and urge incontinence in elderly men, elderly males with urge incontinence or frequency and urgency and symptomatic benign prostatic hypertrophy were studied before and after TURP.

Men, 70 years of age or older, who were clinically felt to require a TURP, and who had established urge incontinence or the symptoms of frequency and urgency, were studied. Investigations consisted of; a history and physical including cognitive testing; a 24 h quantitative monitoring of fluid intake, incontinence, voiding and residual urine; and videourodynamic testing. The amount of obstruction was assessed by urodynamics. The patient then underwent TURP and 6 weeks postoperatively the investigations were repeated. The following were used to determine the response to TURP: symptomatology, changes in bladder emptying, incontinence, flow rate, obstruction and reversal of detrusor instability. Various preoperative parameters were examined retrospectively to determine if they were predictive of a good or poor response to TURP.

Twelve men with a mean age of 80 years of age were studied. Seven patients were cognitively impaired. Preoperatively 11 of the 12 patients were incontinent. Postoperatively all patients had symptomatic improvement. Eight of 11 incontinent patients had an improvement in continence. The improved patients were more

obstructed preoperatively than the unimproved patients. Detrusor instability reversed in only one patient post-TURP. This is in contrast to other studies that have shown that in younger men relief of obstruction will completely abolish detrusor instability in 62% of patients. This difference is statistically significant ($p < 0.001$).

In the geriatric population detrusor instability may be a result of age changes and not secondary to obstruction. Detrusor instability is likely to persist postoperatively in the elderly geriatric patient. Preoperative urodynamic assessment of obstruction in the incontinent male with BPH may be useful since incontinence responds well to TURP if there is marked obstruction.

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CHAPTER 1

INTRODUCTION

Benign prostatic hyperplasia with urethral obstruction is commonly accompanied by detrusor instability in the older man. Symptoms of detrusor instability may include frequency, urgency, and urge incontinence. Detrusor instability has been shown to reverse with relief of obstruction in younger men (mean age 67). Patients in whom it does not reverse can remain symptomatic despite relief of obstruction. Detrusor instability is also seen in unobstructed patients and its incidence increases with increased age. It is the most common cause of incontinence in the institutionalized elderly.

The purpose of this study was to determine if bladder instability in the older man with an obstructing prostate is reversible when the obstruction is relieved or whether the bladder instability is secondary to an irreversible age-related change. Patients were investigated with 24 hour (h) monitoring of incontinence and voiding and conventional urodynamics before and after transurethral resection of the prostate.

CHAPTER 2

LITERATURE REVIEW

A) BENIGN PROSTATIC HYPERPLASIA (BPH)

i) Incidence of BPH

Benign prostatic hyperplasia (BPH) is common in elderly men. The prevalence of pathologically evident BPH at autopsy increases every year after the fourth decade.[1] The incidence rate is variable depending on the criteria used. Autopsy studies have shown that 37 to 73% of men in their seventh decade have BPH.[2,3] Lytton felt that a more meaningful measure of the significance of this entity would be the need for operative relief of obstruction. In 1968 he stated that "the probability of a 40 year old man requiring an operation for benign prostatic obstruction, if he lives to be 80 years of age, is about 10%.[4]" A normative aging study done in 1985 showed that this had increased to a 29% probability.[5]

ii) Symptomatology of BPH

The size of a prostate does not relate to the degree of outflow obstruction. Severe obstruction may be present without obvious prostatic enlargement and conversely large prostates may not create outflow obstruction.[6]

The symptoms of BPH are variable and some patients may be asymptomatic.[5] Prostatic size does not correlate with symptomatology. Patients with large prostates may have no symptoms of obstruction and no evidence of significant obstruction. Conversely patients with small prostates can have significant obstructive symptoms.[6] Symptoms are divided into obstructive or irritative. Obstructive symptoms consist of decreased stream, hesitancy, interrupted stream, dribbling, straining, and incomplete emptying. Irritative symptoms are frequency, nocturia, and urgency. Symptoms may be extremely variable over time. Birkhoff and others found that after 2 years more than half of the patients were unchanged or actually improved based on a subjective symptom score.[7] Symptoms are unreliable in assessment of obstruction when compared to urodynamics.[8-12] McLoughlin *et al.*[9] studied 108 patients with symptoms of bladder outflow obstruction who were on hospital waiting lists for prostatic surgery and found that 28% of them were unobstructed on urodynamic criteria. The only symptoms that have reliably been associated with outflow obstruction are hesitancy and poor stream.[12] Some of these same studies have shown that irritative symptoms have no statistically significant correlation to urodynamic obstruction. Andersen showed that irritative symptoms were highly correlated to the presence of detrusor instability.[8,12] Irritative symptoms and poor stream may result not only from detrusor instability without obstruction but also from detrusor instability with obstruction, urine infection or outflow obstruction bordering on retention.[6]

iii) Treatment of BPH

Transurethral resection of the prostate (TURP) is the most common operation performed to relieve prostatic obstruction. It is also one of the most frequently performed of all operations, ranking from number 10 to number 2 in frequency.[13] In Alberta an average of 3069 prostatectomies are performed annually. Over 50% of these are performed on men who are 70 years or older.[14] In most Western countries 95% of the prostatectomies performed are transurethral.[13]

As transurethral resection has gradually replaced open prostatectomy it has become a safer operation. A cooperative study of 13 institutions completed in 1989, showed that the mortality rate for transurethral resection has been reduced from 2.5% in 1962 to 0.2% in 1989. Although the average age of patients undergoing prostatectomy remained the same, significant morbidity was reduced with the overall morbidity remaining constant.[15]

B) DETRUSOR INSTABILITY

i) Definition

Normally as the bladder fills, intravesical pressure rises very slowly despite large increases in volume. Eventually the rise in intravesical pressure produces the sensation of distension and a voluntarily induced bladder contraction is initiated. Detrusor contractions may also occur involuntarily. According to the International Continence Committee involuntary detrusor contractions during the filling phase on cystometry, which may be spontaneous or provoked and which the patient cannot completely suppress are characteristic of overactive detrusor function. Detrusor overactivity is further defined using the terms the "unstable detrusor" and "detrusor hyperreflexia".[16]

An unstable detrusor is one that is shown objectively to contract, spontaneously or on provocation, during the filling phase while the patient is attempting to inhibit micturition.[16] The patient with detrusor instability may be completely unaware of the involuntary contraction or he may perceive the contraction as an urge to void, suprapubic pain or dysuria. He may or may not be able to suppress its onset or abort it once it starts.[17] An uninhibited detrusor contraction may result in urge incontinence. Uninhibited detrusor contractions in prostatic hyperplasia were first described by Leppanen in 1962 although he used another term.[18]

The term "detrusor hyperreflexia" is defined as overactivity due to disturbance of the nervous control mechanisms. It should only be used when there is objective evidence of neurological disease.[16] The terms detrusor hyperreflexia and detrusor instability are occasionally used interchangeably in the literature and some patients may be incorrectly diagnosed as having detrusor instability if they have an overt or undiagnosed neurologic disease.[8]

ii) Etiology and Pathogenesis

Detrusor instability has only one recognized etiological factor, outflow obstruction in males. Once obstruction has been eliminated as a cause then the instability is said to be idiopathic.[19] The presence or severity of detrusor instability in obstructed males does not necessarily relate directly to the severity of outflow obstruction.[20]

Experimental evidence suggests that there are substantial differences between idiopathic and obstructive instability.[19] Speakman,[21] Sibley,[22] and Brading[23] have shown in obstructive instability in animals that the detrusor develops post-junctional supersensitivity. Using pharmacological and microanatomical studies they have shown that post-junctional supersensitivity is characterized by a reduced sensitivity of the nerve supply to electrical stimulation but a greater sensitivity to stimulation with acetylcholine. The muscle fibers when electrically stimulated *in-vitro* have increased sensitivity which is due to a partial parasympathetic denervation. When obstruction is relieved reinnervation can occur and the detrusor returns to normal behavior.

Pharmacological and histochemical studies of biopsies from patients with idiopathic detrusor instability have shown that the detrusor has a greater degree of spontaneous contractile activity and that it is more sensitive to electrical stimulation and to stimulation with acetylcholine. When the muscle itself is electrically stimulated it responds normally.[19,24]

There are other similarities between idiopathic and obstructive instability besides increased sensitivity to acetylcholine. Cholinergic receptor density is reduced and the alpha-adrenergic receptor density is increased. A reduction of cholinergic receptors is compatible with a reduced innervation but the actual significance of this observation is unclear.[19,25]

Turner-Warwick[20] and others[8,26,27] have stated that detrusor instability frequently develops in response to simple mechanical bladder outflow obstruction and is thereby reversible when the obstruction is relieved. Andersen[8] discusses two main hypotheses on the etiology of detrusor instability in bladder outlet obstruction. The first was proposed by Bors and Comarr[28] who stated that the compensatory detrusor hypertrophy in infravesical obstruction results in a lowered threshold of the stretch reflex in the bladder wall, thereby producing uninhibited detrusor contractions. Detrusor hypertrophy with an elevated detrusor pressure is a common response to outflow obstruction but it is also seen in the neuropathic bladder without any evidence of outflow obstruction.[20] The second hypothesis was put forward by Andersen and Bradley[29] who suggested that glandular enlargement may cause anatomical distortion and compression of nerve endings in the lower trigone and the posterior urethra. These areas are well supplied with sensory nerve fibers and distortion and compression could cause increased sensory stimuli. Relief of obstruction by prostatectomy would lead to interruption of the reflex arc and reversibility of instability.[8]

Abrams[26] attributed bladder instability in obstructed patients to raised micturition pressures. This theory also explains the reversibility of instability since micturition pressures fall when the obstruction is relieved.

Since the incidence of detrusor instability increases with age in both sexes, regardless of whether obstruction is present, it has been suggested that idiopathic detrusor instability occurs due to an age determined functional neurological change.[30] The etiology of detrusor instability in elderly patients with BPH is therefore probably double.

iii) Incidence

Detrusor instability is the most common abnormal urodynamic finding. In patients with symptoms or signs of bladder outlet obstruction it is found in more than 50% of patients.[8,12,20,27,29,31] Its incidence also increases with increased age.[8]

In Andersen's[8] study population, who are described as being elderly, 53% of patients with prostatism had detrusor instability. The incidence of detrusor instability was the same in healthy elderly males. There was no statistically significant difference in age between patients with detrusor hyperreflexia and normal cystometry.

Abrams[27] found an increasing incidence of detrusor instability with age in both obstructed and unobstructed males when he studied 190 men aged 47 to 85 years. Instability was noted in 118 patients or 62%.

Cystometry in females has shown that there is a baseline rate of instability associated with age alone. Jones and Schoenberg[30] studied 45 women, aged 60 to 68 years, who were hospitalized with nonurological complaints and did not have evidence of neurological disease. Eleven percent had detrusor instability with no evidence of outlet obstruction.

Abrams[32] studied 2,124 females and noted that the incidence of detrusor instability increased from 27% in women under 65 years of age to 38% in women over 65 years old. Outlet obstruction occurred in only 3.7% of the total group and the incidence of obstruction did not increase with age.

iv) Reversibility

As noted earlier detrusor instability in the presence of bladder outlet obstruction has been found to be reversible when the obstruction is relieved. However, if detrusor instability is due in part to age-related neurological changes there may be persistence of detrusor instability following relief of outlet obstruction. In elderly men with obstruction the question arises whether their obstruction alone is responsible for their detrusor instability or if it is the result of increased age.

Andersen[31] in 1976 reported a series of patients who were investigated pre-prostatectomy and post-prostatectomy with cystometry. Forty-five percent of the patients had detrusor instability. Six months postoperatively 62% of these patients had regained normal bladder function. The mean age of the patients with persistent instability was 77 years whereas the mean age of patients with reversible instability was 71 years. According to Andersen, it suggested that younger patients more easily regain the physiological balance between input and inhibition than do older patients.

A similar study was performed by Abrams[26] who investigated 318 patients with symptoms suggestive of outflow obstruction. Thirty-four of 55 patients (62%) who preoperatively had detrusor instability had stable bladders 4 months postoperatively. The mean age in those whose instability disappeared and for those in whom it persisted was identical, 66.7 years. Abrams notes however, that the patients in his series were younger than those in Andersen's series. The final incidence of detrusor instability in this series was 23% which is lower than that found by Abrams in a group of 22 "normal" asymptomatic men aged 50-75 years in which the incidence of instability was 50%.

A joint study between Abrams, Farrar, Turner-Warwick, and others[27] confirmed that instability decreased from 60% to 25% postoperatively. Again, no significant age difference between those whose instability reversed and those whose did not was seen. The age range in this series was from 47-85 years.

In 1982, Andersen[8] reported on another series of patients in whom 69% of the patients had reversal of preoperative detrusor instability to normal detrusor reflex function 6 months postoperatively. Although the mean age of patients with reversible detrusor instability was lower than that of patients with persistent instability the difference was not statistically significant.

Persistent detrusor instability post-prostatectomy can be responsible for frequency, urgency and urge incontinence. One must however verify that obstruction has in fact been relieved.[20] The majority of patients with persistent symptoms have some residual outflow obstruction. The "natural incidence" of detrusor instability may also result in the persistence of symptoms.[6]

In clinical studies that have examined reversibility of instability, postoperative cystometrograms have been performed from 1-12 months postoperatively. The timing of the postoperative cystometrograms is controversial.

Abrams,[32] in his review of detrusor instability and obstruction, suggests that most postoperative changes will have occurred by 3 months. Turner-Warwick[6] however believes that patients may take a year or more to revert to stability. During this period detrusor instability related to age or detrusor hyperreflexia develops or progresses. Andersen[31] showed that the most common cause of incontinence following prostatectomy is persistence of preoperative detrusor instability.

C) URODYNAMIC CHARACTERISTICS OF NORMAL MICTURITION

Urodynamic studies in normal males have generally been performed in patients less than 50 years old. Andersen[8] reviewed 8 studies and found that normal micturition as judged by urinary flow rates and intravesical pressure varies within wide limits. Common characteristics seen in younger men include a maximum flow rate of greater than 15 ml/sec and maximum micturition pressure less than 100 cm H_2O. Neither maximum flow rate or intravesical pressure are reliable urodynamic parameters when used alone. Maximum flow rate is dependent on the voided volume and intravesical pressure is affected by changes in abdominal pressure.

Andersen[8] studied 17 elderly males, with a mean age of 67 years, who all claimed to have normal voiding patterns. Their maximum flow rates ranged from 9 to 15 ml/sec. Detrusor instability was found in 53% of these patients. In a review of 5 other studies done on normal men with a mean age greater than 50 years maximum flow rates were less than 15 ml/sec and maximum micturition pressures were consistently greater than 100 cm H_2O. The mean voided volume was also lower. Andersen noted that these changes in micturition parameters with age are not an "all or none" phenomenon and therefore a spectrum of normality exists.

D) MICTURITION IN BLADDER OUTLET OBSTRUCTION

A clear cut urodynamic definition of infravesical obstruction has not been established. A number of different parameters can be measured with urodynamics for each voiding event but none of these can singly define infravesical obstruction.

When urodynamic values from obstructed males are compared to values from unobstructed males the specificity of a single urodynamic parameter is questionable.[8]

Parameters that have been studied, in an attempt to define obstruction include:

Q,	flow rate
Qmax,	maximum flow rate
Pves,	intravesical pressure
Pdet,	detrusor pressure = Pves - Pabd
Pves,	pre, premicturition pressure
Pves,max,	intravesical pressure at maximum flow rate
Pves,cont,	vesical contraction pressure = Pves,max - Pves,pre
Pdet,max,	detrusor pressure at maximum flow rate
Popen,	opening pressure
R,	urethral resistance factor = Pves,max/Qmax

The definition of these parameters and their role in the assessment of defining obstruction will be discussed individually. The definitions of these parameters are taken from the International Continence Society Committee on Standardization of Terminology 1988.[16]

E) URINARY FLOW RATE MEASUREMENT

i) Voided Volume

The voided volume is the total volume expelled via the urethra.[16] The voided volume varies from one void to another and varies between individuals. It is not used to define obstruction but it is used in examining other values, such as flow rate, that are volume dependent.

ii) Flow Rate

Flow rate is defined as the volume of fluid expelled via the urethra per unit time. The maximum flow rate is the maximum measured value of the flow rate.[16] Maximum urinary flow rates vary with age and voided volume. A reduction in maximum flow rate has been a relatively consistent finding in patients with prostatic bladder outlet obstruction but because of the variations seen in healthy elderly males it cannot be used alone to define obstruction.[8] The Liverpool nomogram is a set of maximum and average urine flow rates in normal male and female populations and it takes into account that there is an age related deterioration in male urinary flow rates.[33]

Abrams and Griffiths[34] analyzed the maximum flow rate alone compared to plotting of maximum flow rate versus detrusor pressure. They found that all cases who had a maximum flow rate less than or equal to 6 ml/sec had obstruction and all cases with a maximum flow rate greater than or equal to 20 ml/sec were unobstructed. Andersen[8] has drawn the following conclusion from their work: "With maximum flow rate less than or equal to 10 ml/sec the majority of patients will have infravesical obstruction. Patients with maximum flow rates between 10 ml/sec and 15 ml/sec may be obstructed or not, and with maximum flow rate greater than 15 ml/sec the majority of patients will have unobstructed micturition." If the above values for maximum flow rates are used in screening 60% of patients will be accurately classified.

The average flow rate is voided volume divided by flow time. The calculation of average flow rate is only meaningful if flow is continuous and without terminal dribbling.[16]

iii) Volume Dependence of Flow

The validity of the flow rate depends upon the passage of a reasonable volume of urine. There is a positive relationship between the initial bladder volume, voided volume and the maximum flow rate. Maximum flow rate increases with an increase in voided volume up to a voided volume of approximately 500 ml. A volume of at least 100 ml is required for accurate assessment.[17] In the patient with severe bladder instability they may have such frequent contractions that they never fill their bladder to a sufficient capacity.[6] To overcome this problem and to allow comparisons to be made between individuals who void different volumes nomograms have been designed that relate voided volume to maximum and mean flow.[17]

Patients also show improvement in voiding between the first and subsequent voids when multiple measurements of uroflow are taken so that multiple assessments of uroflow are recommended.[17]

iv) How to Judge a Flow Curve

Most flowmeters can electronically generate a volume-versus-time curve. The shape of the curve is useful in assessing whether the flow rate is maintained or whether it is variable. A normal flow curve has a rapid rise to maximum flow that takes approximately one third of the total voiding time, a period of fairly stable flow and a decrease in flow that is more prolonged that the initial increase (Figure 1). Flow curves are affected by movement, interruption of flow and by a short voiding time interval.[17] The flow curve cannot be relied on exclusively to diagnose obstruction because like other urodynamic measurements it is only one of the parameters necessary for evaluating voiding efficiency.[20]

v) Other Causes of Decreased Flow

Another problem with uroflowmetry is that a poor urinary flow rate could be caused by either obstruction or by a poor detrusor contraction. Blaivas[17] defines outlet obstruction as "a poor urinary flow rate in the presence of an "adequate" detrusor contraction." The measurement of detrusor contraction will be dealt with in a later section.

F) BLADDER PRESSURE MEASUREMENTS

Cystometry measures bladder pressure as a function of filling volume. The following are noted: resting bladder pressure, proprioception, volume at which first desire to void occurs, cystometric bladder capacity, vesical compliance, ability to initiate and suppress detrusor contraction and detrusor hyperactivity or hypoactivity. Intravesical pressure is measured through a catheter in the bladder. A second catheter, inserted in the rectum, estimates the abdominal pressure. If the abdominal pressure is subtracted from the intravesical pressure the detrusor pressure is obtained. The detrusor pressure is the real measure of the detrusor contraction. On a multichannel study each of these measurements is displayed as a separate curve.

In the ICS definitions the following parameters are applicable to measurements of each of the pressure curves: intravesical, abdominal and detrusor pressure. "The premicturition pressure is the pressure recorded at the onset of measured flow. The opening pressure is the pressure recorded at the onset of measured flow. The maximum pressure is the maximum value of the measured pressure. The pressure at maximum flow is the pressure recorded at maximum measured flow rate. The contraction pressure at maximum flow is the difference between pressure at maximum flow and premicturition pressure.[16]"

The opening time is the time from the initial rise in detrusor pressure to onset of flow.[8] The values of premicturition pressures in men with BPH was shown by Castro and Griffiths[11] not to differ from pressures reported in younger "normal" males. In the same study they confirmed Claridge and Shuttleworth's finding that opening pressures are higher in patients with BPH than in younger "normal" males.

Smith[35] in 1968 found that the intravesical pressure at maximum flow rate (Pves, max) was the most accurate single parameter in assessment of urethral obstruction. He found that a value greater than 100 cm H_2O, in the absence of abdominal straining, to be evidence of obstruction. Since Andersen[8] found that most normal elderly men's intravesical pressure at maximum flow rate was more than 100 cm H_2O the usefulness of this measurement is doubtful. Castro and Griffiths[11] found that intravesical pressure at maximum flow rate was very variable and therefore unreliable in the diagnosis of obstruction. The intravesical pressure varies independently of the recorded flow rate and vice versa.[36,37]

G) PRESSURE-FLOW RELATIONSHIPS

Outlet obstruction was defined previously as a poor urinary flow rate in the presence of an adequate detrusor contraction.[17] Uroflowmetry can be used to screen patients for outflow obstruction but it can not distinguish low flow secondary to obstruction from low flow secondary to poor detrusor contractility. Given a patient with a poor urinary flow rate the purpose of synchronous pressure-flow studies is to distinguish obstruction from poor contractility. Simply put a patient who voids with a high pressure and a low flow is obstructed whereas the patient who voids with a low pressure and a low flow has poor detrusor contractility. Unfortunately the majority of patients with low flow rates can not be so easily categorized as there remains a diagnostic "grey zone" between obstruction and poor contractility.[33]

i) Assessment of Obstruction

a) Mathematical Resistance Formula

Attempts have been made to define outlet obstruction mathematically. Maximum urinary flow rate and infravesical pressure can be combined into a single urethral resistance factor. The formula that is commonly used is $R = (p_{ves,max})/Q_{max}$. Formulas to calculate this however are faulty in that they all assume the urethra is a rigid tube.[8,38] Abrams and Griffiths,[34] did show that the calculated urethral resistance could separate clearly obstructed from unobstructed cases, but not cases in the borderline area.

Bruskewitz *et al.*[39] studied 46 patients with urodynamic testing pre-TURP and repeated their studies at 3 and 12 months. The surgeon performing the TURP was blinded to the results of the urodynamic testing. Although a correlation was found between urethral resistance and symptomatology and uroflowmetry, urethral resistance was not useful in predicting which patients would benefit from TURP.

b) Detrusor Pressure

Although intravesical pressure has been used in the calculation of urethral resistance, detrusor pressure (intravesical pressure - abdominal pressure) may better represent true bladder activity. Gleason[36] first stated that detrusor pressure was the more clinically useful voiding parameter as it is independent of extraneous influences and varied independently of the recorded intravesical pressures. In a state of no flow the detrusor pressure would be representative of the strength of the detrusor contraction. However, if voiding occurs through a normal, relaxed urethra during the contraction a high detrusor pressure can not be generated. The strong contraction will instead yield a high flow rate.[40,41] A high detrusor pressure (eg. >50 cm H_2O) is representative of a strong contraction but a low detrusor pressure does not necessarily imply that the contraction is poor. It may imply that there is a good contraction in the presence of normal urethral resistance.[40]

c) URA/URR

The function of the bladder has been assessed using detrusor pressure at peak flow. Detrusor pressure alone though is not a good test of the function of the bladder because it depends on the properties of the urethra. To obtain separate information about the bladder and urethra during voiding Griffiths simultaneously measured flow rate and detrusor pressure and plotted the pairs of measured values throughout the course of micturition. A curve is obtained which shows the detrusor pressure necessary to generate a given flow rate. The curve graphically demonstrates the urethral resistance to flow and has been called the urethral resistance relation. Distinct urethral resistance relations are seen in obstructed and unobstructed urethras. The urethral resistance relation can be used in the assessment of urethral obstruction. As with other methods of assessing obstruction some patients' urethral resistance relation curves will fall into a grey zone.[42]

Obstruction can also be assessed using the URA, which is the group-specific resistance factor. It is a numerical value obtained from the urethral resistance relation. Although it is intended as a research tool to measure changes in urethral resistance it has been suggested that values above a critical upper limit of 29 cm H_2O represents outlet obstruction.[43]

d) Abrams and Griffiths Nomogram

Abrams and Griffiths[34] developed a nomogram of maximum flow rate versus detrusor pressure at maximum flow. The nomogram is divided into 3 areas, obstructed, equivocal and unobstructed (Figure 2). Interpretation of pressure-flow data using the nomogram showed a significantly better subjective outcome of surgery in patients classified as "obstructed" than those "unobstructed".[33]

ii) Assessment of Contraction Strength

There have been numerous measures of detrusor contraction strength or contractility based on pressure/flow studies of voiding.[17,40] To assess the detrusor contraction during flow a number of mathematical equations have been used. The Hill equation, which was developed for striated muscle, can approximate the behavior of smooth muscle. The equation relates the force generated by an actively contracting muscle, the load, to its velocity of shortening.[40]

The BOR or bladder output relation equation is volume dependent. This equation is suited to a hollow organ like the bladder since the pressure generated, the volume of the lumen, and the rate of flow of liquid out of the lumen are all measured.[40]

Griffiths[40,41] has suggested that instead of using the Hill equation which relates force to velocity or the BOR which relates pressure to flow that instead a hybrid relation be used which shows how pressure depends on velocity of shortening for a given contraction. The advantage of this equation is that as long as the contraction is sustained, it does not change much as the bladder empties.

Schafer[44] has analyzed pressure/flow in a different way. He calculates a generalized contractile power. The maximum value of this for a given void represents the greatest contraction strength attained. The relative contractile power which is the percentage of the maximum value is then plotted against time. The disadvantage with this calculation is that no allowance is made for the volume dependence of the flow rate and the flow rate is used in the calculation of the contractile power.

Griffiths[40] has also used a power factor WF ("Watts factor") but it is based on a pressure/velocity analysis which eliminates the difficulties in using volume dependent flow. The power factor, WF, is detrusor pressure multiplied by shortening velocity. WF is the mechanical power generated per unit area of the detrusor surface. Although the calculation of WF is complicated the interpretation is simple. If there is no flow WF is equal to the detrusor pressure. If there is flow, WF is equal to the detrusor pressure plus a contribution from the velocity of shortening. By plotting WF against bladder volume the course of a contraction during an entire void can be examined.

Detrusor contraction strength or power, like other urodynamic measurements, cannot be used singly to define obstruction. They may be useful clinically in the assessment of a patient with BPH to demonstrate that the patient's poor flow rate is secondary to a poor contraction and not urethral obstruction. In patients with BPH and a poor flow rate, a weak detrusor contraction, not urethral obstruction, will lead to a residual urine in 25 to 30% of patients.[34,38] Although a transurethral resection of the prostate will relieve their obstruction, their residual urine will persist if they have a poor detrusor contraction. Measurement of the detrusor contraction preoperatively could be used to predict the response to transurethral resection of the prostate.[45] Contraction strength can also be used as a research tool to analyze changes in bladder performance post-treatment.[40]

H) URETHRAL PRESSURE MEASUREMENTS

The measurement of the urethral pressure at consecutive points along the urethra forms a urethral pressure profile. The maximal urethral pressure is the maximum pressure of the measured profile. The maximum difference between the urethral pressure and the intravesical pressure is the maximum urethral closure pressure.[16] The maximum urethral closure pressure has not been useful in the diagnosis of bladder outlet obstruction since obstruction is not necessarily associated with a high urethral closure pressure.[17,46] The urethral pressure profile can be used to assess the functional urethral length and the length of the prostate. If prostatic length is defined as extending "from the most proximal point when prostatic urethral pressure exceeds bladder pressure to the point when urethral pressure exceeds prostatic pressure" it can be used to estimate adenoma weight.[47] The maximum urethral pressure usually occurs at the external sphincter. In the male an elevation occurs in the presphincteric area and this has been termed the prostatic plateau. The height of the plateau is measured above the level of the bladder pressure. The prostatic peak is any proximal pressure peak. Abrams[48] found that prostatic length, prostatic plateau and the proximal pressure peak all correlated with obstruction as judged by pressure-flow studies. The prostatic plateau was most significantly associated with obstruction.

I) VOIDING CYSTO-URETHROGRAPHY

A voiding cysto-urethrogram is performed simultaneously with measurement of bladder, urethra, and rectal pressures. These pressure curves, together with the urinary flow curve and the sphincter EMG recording, are superimposed on the fluoroscopic image of the cysto-urethrogram. These are all video recorded. Blaivas *et al.*[17] have said that this "provides the most artifact-free, precise display of normal and abnormal physiology." Voiding cysto-urethrography has a role in the identification of the actual site of an obstruction. It may however be difficult to determine whether a proximal posterior urethral obstruction is primarily due to the bladder neck or prostate unless the prostatic cavity is overtly distensible.[20]

J) RESIDUAL URINE MEASUREMENT

The patient's post-void residual urine may be directly measured at catheterization or it can be estimated radiologically. A post-void film of an IVP may be used but it is quite inaccurate. Alternatively ultrasound scans may be used. Various formulae have been used to calculated the bladder volume. Rageth and Langer[49] have shown that, by measuring the areas of the bladder in both longitudinal and transverse directions, the amount of residual urine volume can be accurately estimated. Their formula assumes that the shape of the bladder is similar to a rotation ellipsoid. They compared the calculated volume to the catheterized volume and the difference was less than 15% on average. A nomogram and a table for estimation of residual volume have been computed. The advantages of ultrasound are: it is noninvasive, has no risk of infection, no exposure to radiation, is reasonably accurate, and requires only a few minutes to perform.

Griffiths *et al.*[50] validated this method by comparison with catheterized urine volume in 22 patients; on average the calculated urine volume was within 5 ml of the measured volume, with a standard deviation of 38%.

Historically the presence of residual urine has been used as an indicator for prostatectomy. The use of this has been questioned for a number of reasons. Patients with a severe degree of outflow obstruction may present with no residual urine whereas patients with minimal obstruction can have a large residual urine. Bruskewitz *et al.*[39] also questioned the role of this parameter in the diagnosis of obstruction because of the great intra-individual variation found.

Residual urine seen in patients with urethral obstruction does not occur as a result of obstruction but has been attributed to an abnormality of bladder function.[6,8,20,34] Detrusor muscle fatigue has been suggested as a cause for the development of a residual urine.[8] Detrusor decompensation with low detrusor pressure voiding may occur and lead to the development of a residual.[8,20] Turner-Warwick[6] concludes that since a sizable residual urine only indicates a failure to empty and not obstruction that these patients should undergo urodynamic evaluation. Residual urine following prostatectomy may represent a relative outflow obstruction from residual tissue at the bladder neck or prostate or from a stricture. Alternatively the residual urine may be the result of the pre-existing abnormal bladder function.[20]

K) POST-PROSTATECTOMY

i) Urodynamic Changes

Most post-prostatectomy urodynamic studies have been performed on patients who have persistent symptoms or a new symptom such as incontinence.[20,26,27,51] Meyhoff *et al.*[52] studied 11 patients preoperatively and 6 months postoperatively. The patients all had symptoms of obstruction, including urgency, frequency, and weak stream, and all required transurethral resection of the prostate. No patient in whom an operation failed or who had persistent symptoms was studied. Postoperatively the following urodynamic changes were seen: increased volume voided, increased peak flow rate, increased bladder capacity, no change was seen in the residual urine, and the incidence of detrusor instability decreased from 73% preoperatively to 36% postoperatively. Postoperatively there was a decrease in the amount of power required of the bladder per ml of urine expelled and there was a significant increase in the kinetic energy of the voided stream. The total work capacity of the bladder remained constant.

ii) Symptoms

The outcome of elective prostatectomy, either open or transurethral, is generally satisfactory.[27,53-56] Neal *et al.*[55] studied 217 men post-prostatectomy and found that symptoms were relieved in 79% of patients. Seventy-two percent of the patients, in addition to being symptomatically improved, were also improved on the basis of urodynamics. Patients with an unsatisfactory outcome included those who had preoperative urge incontinence, detrusor instability, low voiding pressures, low urethral resistance, and small prostate size. Men with irritative symptoms postoperatively were more likely to have had preoperative urge incontinence and detrusor instability. Patients with poor urinary flow postoperatively were more likely to have had low voiding pressures, low urethral resistance and small prostate size preoperatively.

A third cause of an unsatisfactory outcome post-prostatectomy is residual obstruction.[20,27] Since obstruction can cause detrusor instability, it is essential to insure that obstruction has in fact been relieved if detrusor instability persists after prostatectomy.[20]

Abrams[27] studied 152 men post-prostatectomy in terms of their symptoms and with urodynamics. Ten patients (7%) had unrelieved obstruction owing to residual adenoma or strictures. More than half of the patients had detrusor instability preoperatively and this converted to stability in between two-thirds and one-half of the patients postoperatively. When symptom scores and urodynamics were both examined, 14 of the patients were unimproved.

Studies that have looked only at patients with symptoms post-prostatectomy have confirmed that detrusor instability is an important factor in the evaluation of the unimproved patient post prostatectomy. In a review of 128 patients who complained of symptoms after prostatectomy, 11 had normal urodynamics. These patients were reassured and 7 of them then lost their symptoms completely. Forty-seven patients had residual obstruction. Forty-five patients complained of urge incontinence and 42 of these were found to have detrusor instability.[20]

Turner-Warwick[6] says that 3 out of 4 patients with proven outlet obstruction present with detrusor instability and that 2 of the 3 bladders will revert to stability when obstruction is relieved. Although these figures are somewhat different than those reported in other series, he uses them to illustrate that at least 1 patient will be disappointed if they all expect that surgery will relieve their symptoms of frequency, urgency and nocturia. Surgery can relieve the outflow obstruction and restore a normal voiding stream. The symptoms of instability may be somewhat improved even if the instability persists. The long term result, in terms of instability, of postponing an operation to relieve obstruction is not known.

Meyoff *et al.*[57] saw a high incidence of irritative symptoms at follow-up in patients who had either a transurethral resection or a transvesical prostatectomy. They concurred with Abrams[32] and others[58] that these symptoms appeared to be more resistant to treatment. They also noted that patients from the transurethral prostatectomy group had a higher incidence of urge incontinence than the transvesical prostatectomy group. All urge incontinent patients from the transurethral group had detrusor instability.[57]

Since the majority of symptoms that occur after a prostatectomy have a urodynamic basis an appropriate urodynamic evaluation is more likely to answer the problem than an endoscopic examination. Symptoms that persist for more than 6 months after the operation are likely to be associated with persistent detrusor instability and/or residual obstruction.[20]

iii) Incontinence

The risk of incontinence after transurethral resection of the prostate is variable. In the American Urology Association National Cooperative Study which evaluated 3,885 patients the incidence of significant incontinence was 0.4%.[15,56] Neal *et al.*[55] have shown that incontinence occurs in as many as 10% of patients. In their particular study however they examined a group of patients whose rate of urge incontinence was substantial preoperatively. Twenty-nine percent of patients had urge incontinence preoperatively which was significantly higher than the incidence postoperatively.

Incontinence following transurethral resection of the prostate occurs as a result of preexisting bladder instability or hyperreflexia, residual obstruction, or damage to the external sphincter.[31,56] In Turner-Warwick's studies,[6,20] the great majority of patients with incontinence post-prostatectomy have persistent detrusor instability. Normally the external sphincter mechanism of the male is capable of containing detrusor contractions unless the unstable contraction creates a grossly elevated intravesical pressure. In patients with urge incontinence post-TURP a degree of distal sphincter damage may contribute to their incontinence.

In the patient with both outflow obstruction and a neuropathy there has been concern that removal of the prostate may result in incontinence. Turner-Warwick[6] argues that "mechanical outflow obstruction, either natural or artificial, is an unsatisfactory form of urinary control for this type of bladder and furthermore it is unreasonable to expect a normal bladder, let alone a neuropathic bladder, to overcome a proven bladder outflow obstruction indefinitely."

Andersen[8] studied a group of patients with post-prostatectomy incontinence and found that uninhibited detrusor contractions were present in 71% of the patients. Fifteen of 34 patients had 2 or more lesions contributing to their incontinence. The most common combination of lesions was detrusor instability with sphincter damage. This same combination of causes had been reported in 1973 by Turner-Warwick *et al.*[20] It is important to evaluate the patient with post-prostatectomy incontinence for detrusor instability if contemplating an incontinence procedure.

L) PREDICTION OF POST-PROSTATECTOMY RESULTS

Cote *et al.*[59] used cystometrography/electromyography and urine flow rates preoperatively and postoperatively to attempt to find predictors of early and late postoperative symptoms. In their study they consider outlet obstruction to be a cause of detrusor hyperreflexia and therefore they do not differentiate between patients with detrusor hyperreflexia and detrusor instability. Of 17 patients who were hyperreflexic preoperatively, 14 were still hyperreflexic 4 weeks postoperatively and 7 remained hyperreflexic at 3 months. When patients were studied at 4 weeks all were improved on the basis of flow rates alone. Twenty-eight percent of the hyperreflexic group had an exacerbation of symptoms in the early postoperative period. All patients with significant symptoms at 3 months postoperatively had hyperreflexia preoperatively and postoperatively. They concluded that "Patients who have hyperreflexia before prostatectomy constitute a group from which it is likely that poor postoperative results may come . . . however, with the passage of time cystometric findings in most of these patients will revert to normal and symptoms will improve." This study confirmed the work of Andersen and others.[8]

Patients with a neurological disorder and prostatic obstruction have been shown to have a poorer prognosis in terms of postoperative continence.[8] Moisey and Rees[60] studied 22 patients with outflow obstruction and a past history of a cerebrovascular accident. Sixteen patients, 15 of whom had a good recovery from their cerebrovascular accident, were continent postoperatively. The remaining patients were incontinent and all had poor recovery and additional neurological problems from their previous cerebrovascular accident. The incidence of preoperative incontinence and or detrusor hyperreflexia was not studied in this series.

Turner-Warwick[6] found that the demonstration of detrusor hyperreflexia in the obstructed patient prior to prostatectomy was not predictive of postoperative incontinence. In complicated patients with outflow obstruction and a coincident neuropathy there is no way of determining if hyperreflexia is due to the obstruction and may reverse with surgery or if it is due to the neuropathy.

The lignocaine test has been used in an attempt to predict post-prostatectomy results in patients with infravesical obstruction and detrusor instability. Patients were given intravesical lignocaine to see if it could be used to differentiate between idiopathic detrusor instability and detrusor instability caused by infravesical obstruction. The lignocaine test was considered to be positive if detrusor instability disappeared for a short period of time. In the 3 patients who had a positive lignocaine test the detrusor instability had disappeared by the sixth postoperative month.[61]

CHAPTER 3

MATERIALS AND METHODS

A) INCLUSION/EXCLUSION CRITERIA

Patients were considered for inclusion in the study if they were male, 70 yrs or greater, had urinary incontinence or frequency and urgency of micturition, and if they had been recently seen by a urologist who was willing to carry out a transurethral resection of the prostate. To be eligible for the study patients had to have urodynamically proven urge incontinence and/or detrusor instability.

Patients were excluded if they were bedridden, had evidence of prostatic malignancy preoperatively, had a history of previous prostatic surgery, had evidence of urethral stricture, were catheter dependent, or had overt subpontine neuropathy. Dementia was not a reason for exclusion. Patients were excluded if they lacked detrusor instability during urodynamics.

Written informed consent was obtained from the patient. Consent was obtained from a relative or guardian if the patient was confused. The study was approved by the ethics review body of the General Hospital (Grey Nuns) of Edmonton.

B) METHODS OF REFERRAL

Patients formed a subgroup of an ongoing geriatric incontinence study being done at the General Hospital. Patients admitted to the geriatric assessment unit with incontinence or urinary frequency and urgency were referred for inclusion into the geriatric incontinence study. Patients were then placed into this subgroup if they had detrusor instability on videourodynamics and urodynamic evidence of obstruction and if a transurethral resection of the prostate was recommended. Other patients were referred to us by their urologist prior to transurethral resection of the prostate because they were thought to fulfill the inclusion criteria.

C) PROTOCOL

The protocol used was the same as that used for the geriatric incontinence study. The protocol consisted of: a history and physical examination; a period of investigation consisting of noninvasive 24 h monitoring of fluid intake, voids, incontinence, and post-void residual urine; videourodynamic studies during filling and voiding; cognitive testing; an intervention, which in this arm of the study was a transurethral resection of the prostate; a SPECT scan of the brain; and repeat 24 h monitoring and videourodynamic studies approximately 6 weeks post-intervention.

i) History and Physical

A history and physical examination, including a general urological and neurological examination, were performed. The rehabilitation medicine staff performed an assessment of the patient's degree of independence using an activities of daily living scale.[62] The patient's medications were recorded and any drug with a potential to affect the lower urinary tract was classified according to Schick.[63] Any drug that had been prescribed to alter bladder function was stopped for one week prior to the patient's urodynamic assessment.

ii) First Investigation Period

The patient was admitted during each investigation period to a nursing unit in the General Hospital with specially trained staff to carry out the monitoring. The patient was asked to drink and eat normally and to follow his usual voiding routine. The patient was then started on a 24 h observation period during which time the total fluid intake and urinary output was monitored and recorded. All of the patient's voids were into a toilet equipped with a flowmeter (Wiest 4150) which registered the amount, the flow curve, and the peak flow rate of each void. The entire series of curves for each patient was assessed and an overall classification was assigned. Flow curves were classified as being consistently normal, consistently abnormal or questionable.[64] A padded undergarment (diaper) was worn throughout the 24 h period and checked by the nursing staff every 2 h. The diapers were weighed on a scale (Mettler PJ 4000) prior to being applied. During the daytime the diapers were changed every 2 h and the removed diaper was weighed to determine the amount of fluid leaked. At night diapers were only changed and weighed if they were felt to be damp when checked every 2 h. Diapers were also weighed sooner if they were obviously wet. The International Continence Society suggests that a weight gain of less than 2 g in 1 h may be disregarded to account for the contribution of perspiration or error.[16] Since our patients wore the same pad for at least 2 h and as long as 8 h overnight, we chose to use a slightly different criterion. We considered measurable leakage to be a weight gain of 3 g or more in any pad together with a total 24 h weight gain of at least 10 g.

During the 24 h study, three post-void residual urines were measured by the nursing staff, using transabdominal ultrasound (Pie Medical 1100). These were done in the early afternoon, evening and following the first void in the early morning. A transverse and sagittal scan of the bladder was taken; the dimensions were then estimated by a built-in software package. Following the method of Rageth and Langer[49] the dimensions of the 2 scans were combined and the residual urine was calculated. In addition to estimating the average post-void residual, diurnal variation was also obtained.

Following completion of the 24 h observation period patients were taken to the urodynamics unit. A voiding and incontinence history was taken and cognitive testing done. The cognitive testing consisted of the Mini Mental State exam (MMSE)[65] and the Cambridge Cognitive Exam (CAMCOG).[66] Both of these yield numerical estimates of the severity of dementia or cognitive impairment and the sensitivity and specificity of the 2 tests for detecting organic mental impairment has been calculated. Maximum obtainable scores are 30 on the MMSE and 107 on the CAMCOG. We have deleted one question on the CAMCOG therefore the maximum obtainable score was 106. A score of 23 or less on the MMSE indicates significant cognitive impairment with 94% sensitivity and 85% specificity.[65] The CAMCOG consists of all of the items on the MMSE plus 43 additional items covering other aspects of cognitive function. A cut-off of 79 on 107 yields 94% sensitivity and 85% specificity for significant cognitive impairment.[66]

Patients also had their blood pressures measured and then underwent a videourodynamic examination. The purpose of the videourodynamics was to gain a complete picture of the function of the bladder and urethra during filling and voiding. If the patient had incontinence the intention was to provoke it during the exam, so as to identify the factors responsible. If there were voiding problems the intention was to identify the immediate causes such as poor contraction or urethral obstruction.

Prior to starting the videourodynamic examination the patient voided in private and the flow rate and voided volume were measured. With the patient supine he was catheterized with two urethral (8 and 5 Fr) catheters using sterile technique. The bladder was emptied through the 8 Fr catheter and the residual urine was determined. A catheter urine sample was taken for culture and sensitivity. The 8 Fr catheter was used during the urodynamics to fill the bladder and the intravesical pressure was measured through the 5 Fr catheter. A 10 Fr feeding tube was placed into the rectum to estimate the abdominal pressure. A pair of disposable silver sphincter electrodes mounted on a sponge (Dantec 13L81) were also passed into the rectum. The electrode was then connected to the amplifier input of a Dantec Cantata EMG machine. A saline-soaked grounding strap was attached to the patient's thigh. Electrical activity was measured during filling and voiding. The intravesical and abdominal pressure-measuring catheters were connected to external pressure transducers at the level of the symphysis pubis. They were flushed with normal saline and tested by having the patient cough. The pressures, flow rate, leakage, and fluoroscopic appearance of the bladder and urethra was recorded (Laborie UDS 500 urodynamic analyzer).

Bladder filling with room temperature contrast (Conray-30) was begun and the position of the catheters was checked with fluoroscopy. Filling continued at a rate of 70 ml/min, using a Dantec 21HO4 pump, until the patient reported pain or an extreme desire to void, if detrusor pressure began to rise, if voiding or substantial leakage occurred or when 1500 ml had been instilled. During filling intravesical and abdominal pressures were measured and the patient was repeatedly questioned regarding sensation of bladder filling and urge to void. Fluoroscopy was performed at intervals and if there were detrusor contractions or leakage. It was also used to examine the full bladder. The patient was asked to cough under fluoroscopy to attempt to provoke bladder contraction or leakage. The patient was then sat up and again asked to cough. He then voided under fluoroscopy. The bladder was then refilled in the sitting position and he again voided. Bladder filling and voiding were performed twice in each session to check reproducibility and to allow the patient to become accustomed to the examination.

Computer analysis of the all the measured variables was used to separate out the contributions of the bladder and the urethra during voiding. The strength (WF at Qmax) and time course of the bladder contraction and the resistance to flow offered by the urethra (URA) were calculated, allowing the severity of the prostatic obstruction to be quantified.

The bladder was emptied through the 8Fr catheter and the residual urine was measured. The 8Fr catheter was then removed. The patient was again moved to the supine position and a urethral pressure profile was measured while the 5Fr catheter was withdrawn by a mechanical puller.

iii) Intervention-Transurethral Resection of the Prostate

Following their first investigation period patients underwent TURP. The

TURP was done under general or spinal anaesthesia. Postoperatively patients had an indwelling catheter attached to continuous or intermittent irrigation. The irrigation was generally discontinued on the 1st postoperative day and the catheter removed the 2nd or 3rd postoperative day. Patients usually received oral antibiotics for 1 to 2 weeks postoperatively. Patients were restudied at the General Hospital, again for a 3 day period, approximately 6 weeks postoperatively.

iv) SPECT Scanning

In the time period between their two investigations all patients had a SPECT (single photon emission computed tomography) scan of the brain using 500 MBq of ^{99m}Tc labelled hexamethylpropyleneamine oxime (HMPAO). SPECT scanning reflects local distribution of blood flow to different regions of the brain. The spatial resolution of the scan is 10 mm, enabling regional differences in cerebral perfusion to be quantified. Quantitative results are expressed as % perfusion of 14 different brain regions. A qualitative report was prepared by a radiologist who was blinded to all other patient data.

v) Second Investigation Period

Approximately 6 weeks post-TURP, the patient was again admitted to the urodynamic nursing unit and the 24 h monitoring and videourodynamics were repeated.

D) ANALYSIS

The results from the first and second investigation periods were compared. The following results were examined preoperatively and postoperatively; the severity of incontinence, the voiding frequency, the residual urine, the degree of obstruction, and the presence and degree of bladder instability. Preoperative predictors of a good response to TURP were also sought. The following factors were evaluated; post-void residual urine, severity of obstruction, cystometric bladder capacity, maximum unstable contraction pressure, strength of detrusor voiding contraction, sensation of bladder filling, degree of cognitive impairment, blood pressure, and SPECT scan results. Statistical analysis was performed with the SPSS/PC+ package, using nonparametric statistics and 2-tailed significance levels.

As these patients form a subgroup of the ongoing geriatric incontinence study, their results are also subject to the same analysis as all patients in that study. The objective of the geriatric incontinence study is to identify any supraspinal dysfunction and the subsidiary factors contributing to incontinence, especially urge incontinence.

CHAPTER 4

RESULTS

A) GENERAL PREOPERATIVE CHARACTERISTICS

A total of 12 patients were studied. They ranged in age from 72 to 90 years with a mean of 80 years.

Their activities of daily living score ranged from 2 to 5. A score of 2 indicates complete independence except for incontinence (Figure 3). The majority of the patients were independent enough to be living either in their own home or in a nursing home.

The patients' mental status was variable: minimental scores ranged from 15 on 30 to 30 on 30. Five patients scored 23 or less indicating significant cognitive impairment. An additional 2 patients were considered cognitively impaired using the CAMCOG (Figure 4).

All patients had sterile urine cultures at the time that they underwent preoperative urodynamic testing.

B) STORAGE CHARACTERISTICS PREOPERATIVELY (TABLE 1)

Patients' daytime frequency ranged from 2-11 times and their nighttime frequency from 0-6. Eleven of the 12 patients were incontinent. They leaked 21 to 1056 g of urine in 24 h. Instability was noted preoperatively on videourodynamics in all 12 patients. Bladder capacity was measured during videourodynamics and ranged from 144 to 689 ml.

C) VOIDING CHARACTERISTICS PREOPERATIVELY (TABLE 2)

During the initial 24 h study mean flow rates ranged from 2 to 6 ml/sec. Maximum flow rates ranged from 4 to 25 ml/sec. When mean and maximum flow rates were plotted on the Liverpool nomogram[33] 11 of the 12 patients had flow rates which were at or below the 50th percentile for men over 50 years of age. Ten patients had abnormally-shaped flow curves and the remaining 2 patients had flow curves that were considered questionable. On ultrasound patients had a mean residual urine that ranged from 57 to 381 ml. Five patients had mean residuals greater than 200 ml. Morning residuals were generally higher and ranged from 50 to 541 ml.

The strength of the detrusor contraction (WF) at maximum flow was assessed during urodynamics. A value of 10 W/m^2 is considered to be normal in younger patients. A normal value in elderly patients has not been defined. In the geriatric urge incontinence study WF has been shown to be lower than 10 W/m^2 in the majority of patients who otherwise have normal voids. This implies that the elderly have some loss of strength of detrusor contraction. WF at maximum flow in this group of elderly patients pre-TURP ranged from 3.4 to 10.4 W/m^2.

D) ASSESSMENT OF OBSTRUCTION PREOPERATIVELY (TABLE 3)

Obstruction preoperatively was assessed using urine flow rates, and pressure/flow voiding studies using the Abrams and Griffiths nomogram and the URA value. The primary test of obstruction was the free flow rate. This was taken from the patients numerous free voiding flow curves obtained during 24 h monitoring. The pressure/flow voiding studies obtained during videourodynamics were used as confirmation of obstruction.

Based on Anderson's[8] analysis of obstruction by flow rates 6 patients were classified as being obstructed. Two patients had flow rates that were equivocal and 4 patients had flow rates that were unobstructed.

One patient was unable to void during preoperative videourodynamics and therefore his obstruction could not be assessed urodynamically. However his preoperative free flow curves were persistently abnormal with an obstructed appearance. His maximum flow was 4 ml/sec, the lowest recorded in this series.

When the detrusor pressure and maximum flow were plotted on the nomogram,[34] 7 patients were considered to be obstructed. Four patients' values fell into the equivocal zone (Figure 5).

If a URA value of greater than 27 is used to define obstruction on urodynamics,[43] 8 patients were defined as being obstructed and 3 were unobstructed. One patient who was near the line between the equivocal zone and the obstructed zone on the nomogram had a URA value that was indicative of obstruction.

The variable number of patients who were classified as being obstructed using flow rates, the nomogram, and URA illustrates the imperfections that exist in determining obstruction urodynamically. In total we classified 8 patients as being urodynamically obstructed.

E) TURP

Patients underwent a TURP under general or spinal anaesthesia. The amount of tissue resected ranged from 4.5 to 60 g. Three patients were diagnosed as having Ca of the prostate. There were no major complications in the perioperative period in any of the patients. The 24 h monitoring and videourodynamics were repeated at an average of 7 weeks postoperatively in 11 of the 12 patients. One patient dropped out of the study following his TURP. His family and caregivers at the nursing home where he lived claimed that he had no difficulty voiding and was completely continent post-TURP.

F) EFFECT OF TURP ON STORAGE CHARACTERISTICS (TABLE 4)

The patients all felt that they were improved postoperatively. Patients all felt that they voided better, and had less frequency day and night. Those who were incontinent preoperatively felt that they were less incontinent postoperatively.

Despite all patients thinking that they had less frequency the repeat 24 h study showed that only 1 patient had a marked reduction in the number of daytime voids from 11 to 6 whereas the other patients had minimal changes or an increase in frequency (Figure 6). The change in the number of nighttime voids was also minimal.

When the absolute change in incontinence is examined in the 10 incontinent patients tested postoperatively 7 patients improved, although some only had minimal improvement. One patient had no change in the amount he was were incontinent, and 2 patients were more incontinent postoperatively (Figure 7a, 7b). One patient was 109 g more incontinent post-TURP. On repeat videourodynamics his incontinence was urge and was not due to external sphincter damage. The patient who withdrew from the study was allegedly completely dry and therefore will be referred to as an improved patient.

Since the patients' amount of incontinence preoperatively was extremely variable the change in incontinence is better examined by the percent change (Figure 8a). Three patients had an improvement of more than 50%, 5 patients had an improvement of less than 50%, and 2 patients were more incontinent postoperatively. The patient not tested had a 100% improvement in continence according to his caregivers (Figure 8b).

Detrusor instability persisted in 10 of 11 patients tested post-TURP with only 1 patient having no instability post-TURP. Two additional patients had significant reductions in maximum unstable detrusor pressure (Figure 9a). Four patients actually had an increase in their maximum unstable detrusor pressure (Figure 9b).

The change in bladder capacity was variable post-TURP. Six patients had a reduction in capacity, 4 had an increase and 1 patient had no change.

G) EFFECT OF TURP ON VOIDING CHARACTERISTICS (TABLE 5)

The mean flow rate improved in 8 patients. The maximum flow rate improved in all but 2 patients (Figure 10). Ten of 11 patients improved in terms of their position on the Liverpool nomogram. Maximum flow rates postoperatively ranged from the 10th to above the 95 percentile and the mean flow rates ranged from the 5th to above the 95 percentile.

The patient who was considered obstructed based on flow rates alone had a dramatic change in both his maximum and mean flow rates post-TURP. His maximum flow rate increased from 4 ml/sec to 42 ml/sec and his mean flow rate increased from 2 ml/sec to 14 ml/sec.

Preoperatively his maximum and mean flow rates were at the 5th percentile. Postoperatively his maximum flow was at the 90th percentile and his mean flow was at the 50th percentile. His voiding curves also changed dramatically (Figure 11). These changes confirm that he was indeed obstructed.

On repeat 24 h study only 6 patients had a change in their voiding curves, 4 patients with preoperative abnormal curves had questionable curves postoperatively and 2 patients with preoperative questionable curves had normal curves postoperatively.

Results of the repeated 24 h monitoring showed that 8 of the 11 patients studied had a reduced mean residual urine postoperatively. All patients with a preoperative mean residual greater than 100 ml had a reduction in postoperative residual. Those patients whose residuals increased had relatively small residuals preoperatively, and relatively small increases postoperatively (Figure 12). There was also a similar decrease in the morning residual urine.

One patient had an increase in detrusor contraction strength (WF) postoperatively, 4 patients had negligible change and 5 patients had a decrease.

H) EFFECT OF TURP ON OBSTRUCTION (TABLE 6)

When free flow rates were used to determine the presence of obstruction post-TURP only 1 patient remained obstructed. Two patients had maximum free-flow rates postoperatively that were equivocal.

Interpretation of pressure/flow studies using Abrams and Griffiths nomogram showed that 5 previously obstructed patients were improved postoperatively. Two patients who had equivocal values on the nomogram had URA values that were obstructed.

On postoperative testing 3 of the 4 patients who were considered to be unobstructed had some improvement in terms of an improved flow and a lower detrusor pressure when voiding.

Of the 8 patients who had been obstructed preoperatively 1 patient did not undergo postoperative testing. Using free-flow rates and pressure/flow studies to assess obstruction 5 of the remaining 7 patients were improved postoperatively.

Two patients, 1 who was obstructed preoperatively and 1 who was unobstructed preoperatively voided with higher pressures and lower flow rates when tested postoperatively. When these values are plotted on the nomogram they fall within the obstructed zone (Figure 13). If the URA is calculated it is greater than 29 which also indicates obstruction. On fluoroscopy, both these patients had wide open prostatic cavities and no evidence of obstruction. Their high pressures and low flows are attributed to poor relaxation of the pelvic floor during voiding. This was confirmed with EMG tracing in one patient. EMG was not available for the other patient (Figure 14). Both of these patients had improvements in urinary flow rates.

I) ASSESSMENT OF IMPROVEMENT AND POSSIBLE PREDICTIVE FACTORS

Improvement could be considered using the following parameters: obstruction, incontinence, frequency, residual urine and instability. Improvement in all parameters was only seen in 2 patients. Two additional patients had improvement in 4 of the 5 parameters (Figure 15).

Although the purpose of the study was to examine the change in detrusor instability, the patient is more likely to notice a change in incontinence particularly if there is an improvement. As stated earlier, 8 of 11 incontinent patients had an improvement in continence. In an attempt to find a predictor of improvement post-TURP the data was examined by dividing the patients into those whose incontinence improved versus those who did not. No difference is seen between the

2 groups of patients in terms of preoperative symptoms. The following is noted when the mean for each group is calculated. Preoperatively the improved patients had less frequency and less nocturia, more incontinence, higher maximum detrusor pressures, and a larger bladder capacity. The improved patients had lower maximum flows, higher detrusor pressures during voiding, slightly lower preoperative residual urine, and a greater contraction strength, or WF. In keeping with their lower flow rates and higher detrusor pressures more (88%) of the improved group were urodynamically obstructed preoperatively compared to the unimproved group (34%). Urodynamic obstruction may be a possible predictor of improvement in incontinence post-TURP (Figure 16a). The mean change in continence in patients who were not obstructed was an increase in incontinence of 33 ml (range -9 to 109) versus a mean improvement of 164 ml (range 13 to 458) in patients who were obstructed (Figure 16b).

One patient who was considered to be urodynamically obstructed preoperatively did not improve significantly in terms of incontinence and he had no change in detrusor instability. This man was also one of the patients who remained urodynamically obstructed postoperatively. As stated earlier, his prostatic fossa and bladder neck opened well on fluoroscopy and his high pressures and low flow were attributed to poor relaxation. Fortunately this man had very little incontinence preoperatively, 23 g, and therefore he was not as inconvenienced by his incontinence as some of the other patients.

Since most of these patients' detrusor instability was not abolished by relieving their obstruction the cause of their instability may be central. In an attempt to identify the area of the brain responsible for this, cognitive testing and SPECT scans have been used. No relationship between cognitive impairment and persistent detrusor instability was found however 3 of the 4 patients who had a greater than 50% improvement in incontinence were cognitively impaired. The fourth patient with marked improvement of incontinence scored 30 on 30 on his minimental status exam (Figure 17a). The mean change in continence in patients who were cognitively impaired was an improvement of 180 ml. Patients who were cognitively intact had a mean increase in their amount of incontinence (Figure 17b).

CHAPTER 5

DISCUSSION

The purpose of this study was to determine if detrusor instability in elderly men (mean age 80) with clinical obstructive benign prostatic hypertrophy would have reversal of detrusor instability following transurethral resection of the prostate. Only 1 of 8 obstructed patients had reversal of instability 6 weeks postoperatively. This is in contrast to other studies[8,27,31] that have shown that relief of obstruction will completely abolish detrusor instability in approximately two-thirds of patients. Abrams[12] studied 55 patients with preoperative instability post-TURP, who had a mean age of 67 years. Relief of obstruction completely abolished detrusor instability in 62% of the patients.

The null hypothesis tested in this study is that in an older population, mean age 80 years, 62% of obstructed patients will have reversal of instability after TURP. As only 1 of 8 patients had reversal, the null hypothesis is rejected with $p < 0.001$, (binomial test).

The timing of the postoperative study may have had some influence on the incidence of reversal of detrusor instability. Abrams' patients were studied at least 4 months after surgery whereas the patients in this study were studied approximately 6 weeks postoperatively. The difficulty with waiting 4 months or 1 year as suggested by Turner-Warwick is that during this period detrusor instability related to age could develop or progress.

Since only 1 patient reverted to detrusor stability after TURP it is unlikely that detrusor instability is secondary to urethral obstruction in this subset of elderly patients. Characteristics common to this subset of patients included dependence in some activities of daily living and cognitive impairment. Patients all had scores of 2 or greater on an activities of daily living scale. This indicates dependence in at least one activity of daily living. Seven of the 12 patients studied were significantly cognitively impaired using the MMSE and the CAMCOG.

Elderly men with bladder instability and clinical obstruction likely have two separate entities. Detrusor instability in these men may be related to cerebral neuropathy that also results in cognitive impairment. This hypothesis is supported by this data as the only patient who had reversal of instability was cognitively intact. Cognitive impairment in the obstructed elderly man with detrusor instability should not be a deterrent to performing a TURP since the cognitively impaired patient may have improvement in their incontinence. No patient had significant postoperative morbidity following TURP. One patient died 12 weeks postoperatively but he was 90 years old and had been on home oxygen for chronic obstructive pulmonary disease prior to his TURP.

Although relieving obstruction will not cure detrusor instability, elderly male patients with obstruction can still benefit from a TURP. When their obstruction is relieved the bladder instability may improve somewhat. The degree of preoperative obstruction as diagnosed by urodynamics can be used as a possible predictor of improvement in incontinence post-TURP. The severity of incontinence is more likely to be substantially reduced in patients with more severe obstruction than those with borderline or no obstruction on urodynamics. Another advantage to doing a TURP in these men is that postoperatively they may be candidates for an anticholinergic. Anticholinergics are useful in inhibiting involuntary bladder contraction by increasing bladder volume to the first involuntary contraction, decreasing the amplitude of the contraction, and increasing the total bladder capacity.[67,68] They have been the mainstay of therapy for urge incontinence but are contraindicated in the presence of obstruction.[69] A commonly used anticholinergic is Oxybutynin chloride which also has musculotropic and local anesthetic properties. It has been shown in a randomized trial to provide symptomatic improvement in two thirds of patients with detrusor instability.[70]

Elderly male patients with obstructive and irritative symptoms would benefit from preoperative urodynamics to determine the amount of obstruction they have and therefore to predict the reversibility of their instability. Obstructed patients should be warned that although their instability may improve it may not reverse completely. The cognitively impaired obstructed patient is at greater risk of not having complete reversal of instability. However the obstructed incontinent patient regardless of cognitive status will likely have improvement in the amount of incontinence post-TURP. Patients who are not urodynamically obstructed could be told that it is unlikely that their detrusor instability and their irritative symptoms, including the amount of incontinence, will be alleviated post-TURP.

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APPENDIX I

RESULTS BY INDIVIDUAL PATIENT

Patient 1 was an 85 year old from a nursing home with frequency, urgency, poor stream and urge incontinence by history. He had been previously seen by a urologist and following cystoscopy was booked for a TURP. He was then referred to us as a suitable candidate for our study. His score on the minimental exam was borderline and he was cognitively impaired on the CAMCOG. During the 24 h study he had a large residual and a poor mean flow with abnormal flow curves. His maximum flow rate however was high, 25 ml/sec. At videourodynamics he was not obstructed. He had bladder instability and demonstrated urge incontinence. The strength of his detrusor contraction was low, 3.4 W/m^2, which likely contributed to his high residual.

Following TURP his incontinence was worse. He had a reduction in his mean residual and a decrease in his maximum flow. His frequency was improved postoperatively. His detrusor instability and urge incontinence persisted despite a fall in his URA value. The only improvements then that this man had was a reduction in his residual urine volume and a reduction in frequency. He was told that he might be a candidate for an anticholinergic if his residual urine volume continued to decrease. He was not however interested in taking any more medications.

Patient 2 was an 82 year old who was in our institution for geriatric assessment and was referred to us because of incontinence. He was cognitively impaired. He had a high mean residual and was very incontinent, 578 ml. His mean flow was 4 ml/sec and his maximum flow was 17 ml/sec. His flow curves were classified as abnormal. He was found to be obstructed during videourodynamics. He also had detrusor instability and urge incontinence was demonstrated. His contraction strength was considered reasonable, 7.5 W/m². Because of his obstruction he was referred to a urologist and subsequently had a TURP.

Post-TURP he had improvements in his mean residual, frequency and in his degree of incontinence. He had been obstructed and was unobstructed post-TURP. He also had a corresponding increase in maximum flow. Although his detrusor instability persisted the pressure was reduced. The WF or strength of voiding contraction also decreased postoperatively.

Patient 3 was an 81 year old who like the previous patient was referred to us for incontinence. He was also cognitively impaired. His mean residual was less than 200 ml. He was incontinent of 362 ml in 24 h. His mean and maximum flows were low, 2 and 9 ml/sec, respectively. He had an abnormal flow curve and was found to be obstructed. He also had detrusor instability. His contraction strength was normal, 10.4 W/m².

This patient's postoperative changes were similar to those of patient 2. He also had improvements in mean residual, frequency and incontinence. His TURP relieved his obstruction and increased his maximum flow. The detrusor instability also persisted but at a reduced pressure. WF also decreased.

Patient 4 was an 83 year old who was also a patient in this institution. He had been seen by a urologist because of poor stream, hesitancy, and incontinence. He was thought to have an obstructing prostate at cystoscopy, however he was also noted to have a very reddened inflamed-looking prostate. He was started on antibiotics and when he failed to respond he was then referred to us for urodynamics. He had a borderline score on the minimental exam and was cognitively impaired on the CAMCOG. His residual urine was high. He was incontinent of a small amount. His mean and maximum flow rates were normal, 6 and 25 ml/sec, however his flow curves were questionable. On videourodynamics he was not obstructed but he did have instability. The strength of his detrusor contraction was moderate, 6.5 W/m^2.

Post-TURP the patient had a reduction in his mean residual urine and only very little change in the amount of incontinence. His frequency was unchanged. He had an increase in maximum flow and improved flow curves. However during videourodynamics he voided with a high pressure and a low flow and was therefore classified as being obstructed. It is unlikely that he was unobstructed pre-TURP and would be obstructed post-TURP. On fluoroscopy his bladder neck and prostatic fossa were wide open which further supports a lack of obstruction. The likely cause of his high pressure, low flow voiding during videourodynamics was failure of relaxation. He had a slight increase in pressure during his unstable detrusor contraction postoperatively.

Patient 5 was a 72 year old referred to us post-cystoscopy from a urologist. He was considered to be obstructed and a TURP was planned. He was referred to us as he was felt to be a good candidate for our study because he had frequency, urgency, and incontinence. He was cognitively intact. He had a small residual and was incontinent 21 g in 24 h. His mean and maximum flows were 4 and 14 ml/sec and his flow curves were abnormal. Using the nomogram to assess for obstruction he was considered to be equivocal, on the borderline for obstruction. His URA value was greater than 27 and therefore obstructed. He also had detrusor instability. His detrusor contraction strength was 7.1 W/m^2.

This man had a slight increase in his post void residual urine post-TURP. He had no change in his incontinence. His daytime frequency fell tremendously from 11 to 6. Postoperatively his URA value fell to an unobstructed level. His detrusor instability persisted.

Patient 6 was an 81 year old who had also been seen by a urologist and for whom a TURP was planned. He was cognitively intact. His residual was less than 200 ml. Although he had a history of incontinence we were unable to measure any.

His mean and maximum flow rates were 4 and 16 ml/sec. and his flow curves were abnormal. At videourodynamics he demonstrated detrusor instability. He had a low contraction strength of 5.3 W/m^2.

Post-TURP he had a reduction in residual urine and an increase in maximum flow rate. He had an improvement in frequency. No change in detrusor instability was measured and again no incontinence was seen during the 24 h study or during videourodynamics.

Patient 7 was a 73 year old who was also referred to us prior to his TURP. He was cognitively intact and had a residual of less than 200 ml. He was incontinent 83 g in 24 h. His flow curves were abnormal with a mean flow of 2 ml/sec and a maximum flow of 4 ml/sec. During videourodynamics this man demonstrated instability but was unable to void. He was considered to be obstructed on the basis of the cystoscopy and his poor flow rates.

As discussed earlier this is the patient who had the tremendous increase in maximum flow rates (from 4 to 25 ml/sec) post-TURP. He also had reductions in mean residual urine, and a reduction in incontinence. His urinary frequency also improved. This was the only patient to have complete resolution of detrusor instability post-TURP.

Patient 8 was a 74 year old who was also referred to us because he was considered to be a good candidate by his urologist. He was not cognitively impaired. His total incontinence in 24 h was 23 g. His mean and maximum flow rates were 3 and 12 ml/sec, his flow curves were classified as being questionable. He was urodynamically obstructed and he had detrusor instability. His detrusor contraction strength was slightly low, 8.7 W/m^2.

Post-TURP this man's improvements consisted of a reduced mean residual, a reduction of frequency and a small improvement in incontinence. He also had an increase in his flow rates.

Patient 9 was a 90 year old who was a patient in this hospital. He was cognitively impaired. He was incontinent and was known to have a large residual. He was clinically felt to be obstructed but there was concern that he was not an operative candidate because of a history of sleep apnea and chronic obstructive pulmonary disease. He was referred to us for urodynamics. During the 24 h study his large amount of incontinence and his large residual were confirmed. The mean and maximum flow rates were low, 2 and 5 ml/sec. He had abnormal flow curves. During videourodynamics obstruction and detrusor instability were demonstrated. The strength of the detrusor contraction was 7.5 W/m^2. He eventually underwent TURP because of recurrent episodes of retention.

He did well postoperatively and was discharged to a nursing home. His family would not permit him to return for post-TURP follow-up but he was allegedly 100% improved. He died approximately 12 weeks post-TURP.

Patient 10 was a 77 year old who had been admitted for geriatric assessment because of a recent onset of confusion. On the minimental exam he scored 15 out of 30. He was noted to be incontinent and was referred to us for evaluation. He was incontinent of 302 g and voided with abnormal flow curves. His residual urine was 82 g. His mean and maximum flow rates were low, 2 and 5 ml/sec. He was obstructed on videourodynamics and was therefore sent to a urologist. The urologist felt that he was a good candidate for a TURP.

Post-TURP the patient initially did well. When he returned for his 6 week post-TURP assessment he appeared more confused than previously. He was a diabetic and his 24 h study had to be rescheduled twice because of severe hypoglycemic reactions. He was eventually studied when medically stable and was noted to have a very substantial improvement in flow rates. His maximum flow rate increased from 5 to 52 ml/sec. His daytime frequency was worse having increased from 2-8. He had an increase in residual urine. He had a slight decrease in incontinence. During videourodynamics his obstruction was improved as was his detrusor instability. His WF fell from 8.6 to 6.3 W/m^2.

Patient 11 was an 82 year old who was cognitively impaired and had been admitted for geriatric assessment because his family was having difficulties managing him at home. He was referred to us because of incontinence. His flow rates were low and he voided abnormally. He was incontinent of 33 g and had a residual of 70 ml. During videourodynamics he was noted to be obstructed and was subsequently referred to a urologist and had a TURP.

His follow-up study showed that his incontinence had increased slightly. Although his residual urine was also increased he was no longer obstructed on videourodynamics. His detrusor instability also improved. He also had a decrease in WF postoperatively.

The last patient was an 86 year old aphasic man who was referred to our institution from the nursing home where he lived because he was becoming increasingly more difficult for them to manage. His incontinence was one of his main problems so he was therefore referred to us for urodynamics. His cognitive status could not be determined. He was very incontinent, 1056 g, and had a residual of 140 ml. His flow curves and flow rates were inaccurate since all his voids into the flowmeter were very small. During videourodynamics obstruction was diagnosed. He was assessed by a urologist and underwent a TURP.

Postoperatively he was still incontinent of large amounts, 798 g, and only had 2 voids, both small volume, into the flowmeter. His residual urine decreased slightly. In assessing his obstruction postoperatively he had a decrease in URA but remained in the obstructed range. His position on the nomogram changed from obstructed to equivocal. Overall his obstruction was not considered to be improved. He had improvement in detrusor instability and a decrease in WF, the contraction strength.

STORAGE CHARACTERISTICS PRE-OP

PATIENTS	1	2	3	4	5	6	7	8	9	10	11	12
FREQUENCY	10	6	5	7	11	11	9	8	4	2	4	6
NOCTURIA	4	1	1	3	5	4	4	6	2	1	1	0
INCONTINENCE (g)	76	578	362	47	21	0	83	23	237	302	33	1056
INSTABILITY	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
CAPACITY (ml)	417	600	383	635	144	410	675	275	689	282	275	258

Table 1

VOIDING CHARACTERISTICS PRE-OP

PATIENTS	1	2	3	4	5	6	7	8	9	10	11	12
MEAN FLOW	3	4	2	6	4	4	2	3	2	2	2	2
PERCENTILE	25	10	5	95	5	10	5	10	10	5	5	25
MAX. FLOW	25	17	9	25	14	16	4	12	5	5	6	4
PERCENTILE	75	50	50	90	25	25	5	50	10	5	10	25
FLOW CURVES	*AB	AB	AB	?	AB	AB	AB	?	AB	AB	AB	AB
AV RESIDUAL (ml)	381	294	131	319	57	127	158	63	373	106	225	79
AM RESIDUAL (ml)	541	293	165	305	74	159	268	77	505		296	50
WF (W/m^2)	3.4	7.5	10.4	6.5	7.1	5.3		8.7	7.5	8.6	6.6	10.1

*AB = abnormal

Table 2

ASSESSMENT OF OBSTRUCTION PRE-OP

PATIENTS	1	2	3	4	5	6	7	8	9	10	11	12
NOMOGRAM	*E	*O	O	E	E	E		O	O	O	O	O
URA	*U	O	O	U	O	U		O	O	O	O	O
FLOW RATE	U	U	O	U	E	U	O	E	O	O	O	O
CLASSIFICATION	U	O	O	U	U	U	O	O	O	O	O	O

*E = equivocal
*O = obstructed
*U = unobstructed

Table 3

EFFECT OF TURP ON STORAGE CHARACTERISTICS:
expressed as the change from pre to post-op

PATIENTS	1	2	3	4	5	6	7	8	10	11	12
FREQUENCY	*-2	-2	1	2	0	-5	-3	1	6	3	-5
NOCTURIA	1	1	1	0	-1	1	-1	-2	1	1	1
INCONTINENCE (ml)	109	-458	-295	-9	0	N/A	-69	-10	-73	15	-258
INCONTINENCE (%)	145	-79	-81	-19	0	N/A	-83	-43	-24	27	-24
INSTABILITY	*INC	*DEC	DEC	INC	INC	DEC	*AB	INC	DEC	DEC	DEC
CAPACITY (ml)	-240	-70	47	-375	24	-100	0	-65	134	103	-11

* a negative number is a reduction post-op
*INC = increased
*DEC = decreased
*AB = abolished

Table 4

EFFECT OF TURP ON VOIDING CHARACTERISTICS:
expressed as the change from pre to post-op

PATIENTS	1	2	3	4	5	6	7	8	10	11	12
MEAN FLOW	0	3	3	5	*-1	2	12	3	8	5	0
PERCENTILE	25	40	45	0	45	0	-45	15	0	70	-15
MAX. FLOW	-8	13	5	16	1	9	39	6	47	15	-2
PERCENTILE	20	45	25	5	50	25	85	25	90	80	-15
FLOW CURVES	*IMP	*SA	SA	IMP	SA	SA	IMP	IMP	IMP	IMP	SA
AV RESIDUAL (ml)	-205	-213	-60	-147	16	-22	-73	-11	9	-84	2
WF (W/m²)	0.4	-3.4	-7.9	1.2	0.5	0.3		0.3	-2.3	-2.3	-4.7

* a negative number is a reduction post-op
*IMP = improved
*SA = same

Table 5

ASSESSMENT OF OBSTRUCTION

PATIENT	1	2	3	4	5	6	7	8	10	11	12
NOMOGRAM PRE-OP	•E	•O	O	E	E	E		O	O	O	O
POST-OP	E	•U	U	O	E	E	E	O	E	E	E
CHANGE	•S	•I	I	•W	S	S		S	I	I	I
URA PRE-OP	U	O	O	U	O	U		O	O	O	O
POST-OP	U	U	U	O	O	U	U	O	O	U	O
CHANGE	S	I	I	W	S	S		S	S	I	S
FLOW PRE-OP	U	U	O	U	E	U	O	E	O	O	O
POST-OP	U	U	E	U	E	U	U	U	U	U	O
CHANGE	S	S	I	S	S	S	I	I	I	I	S
OVERALL CHANGE	S	I	I	W	S	S	I	S	I	I	S

•E = equivocal
•O = obstructed
•U = unobstructed

•S = same
•I = improved
•W = worse

Table 6

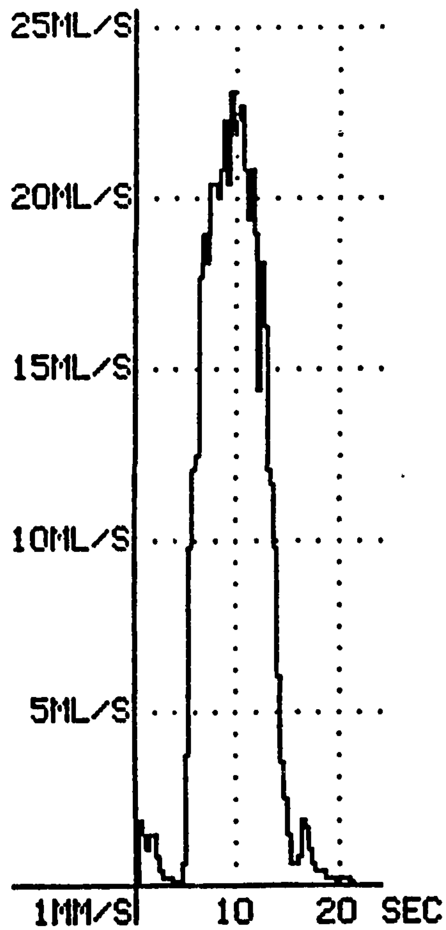


Figure 1: Normal uroflow. Voided volume = 161 ml.

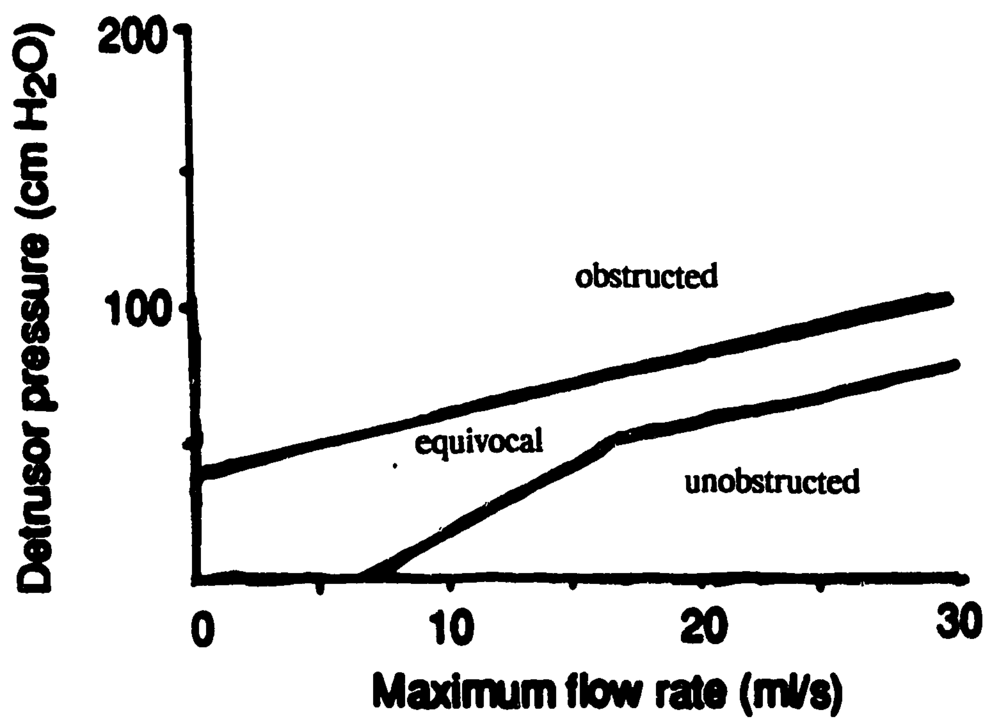


Figure 2: Nomogram of maximum flow rate versus detrusor pressure at maximum flow after Abrams and Griffiths. The two lines divide the figure into three zones of obstructed, equivocal and unobstructed.

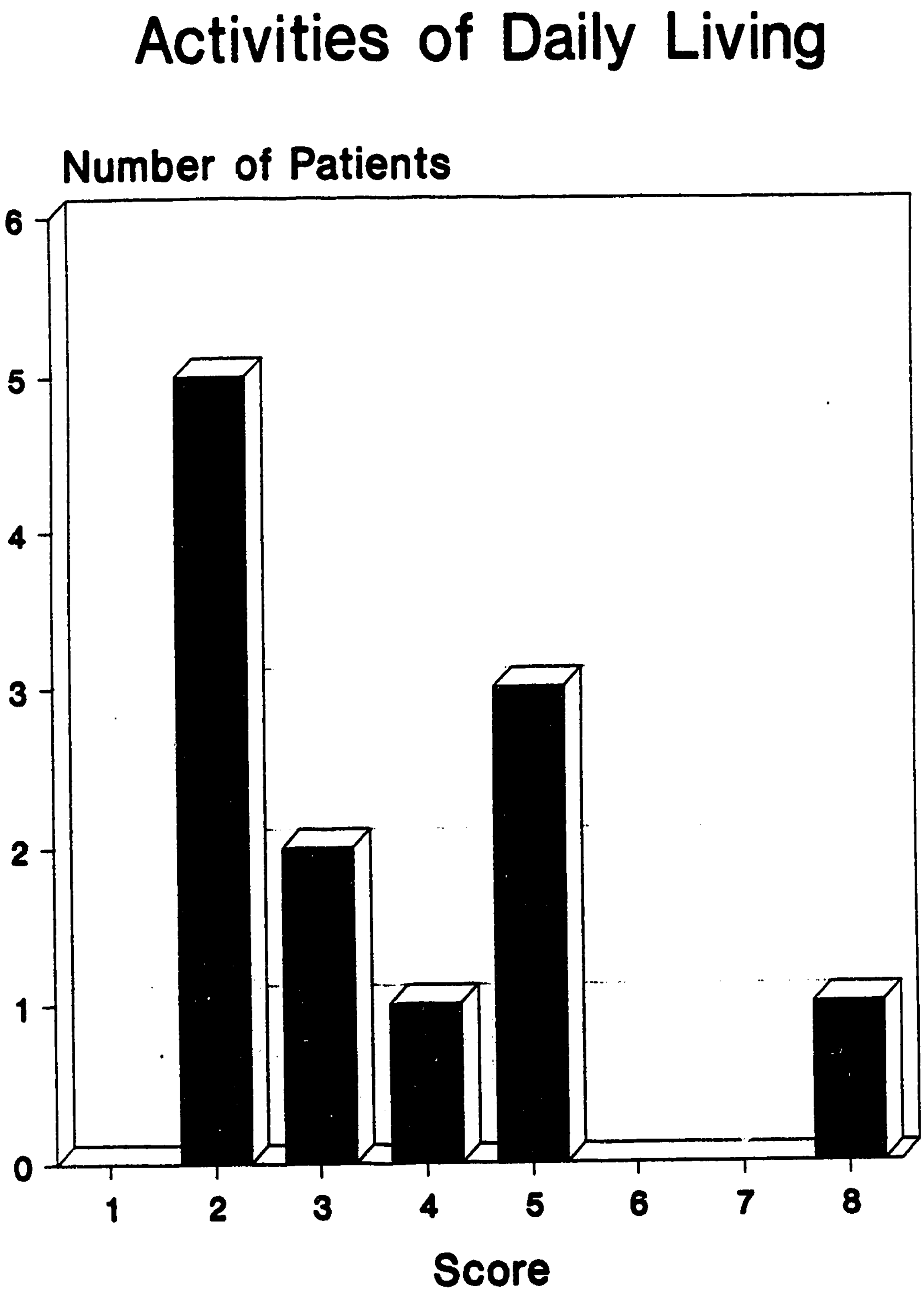


Figure 3: Activities of Daily Living Score: 2 indicates complete independence except for incontinence. A higher score indicates increased dependence. 8 is unclassifiable.

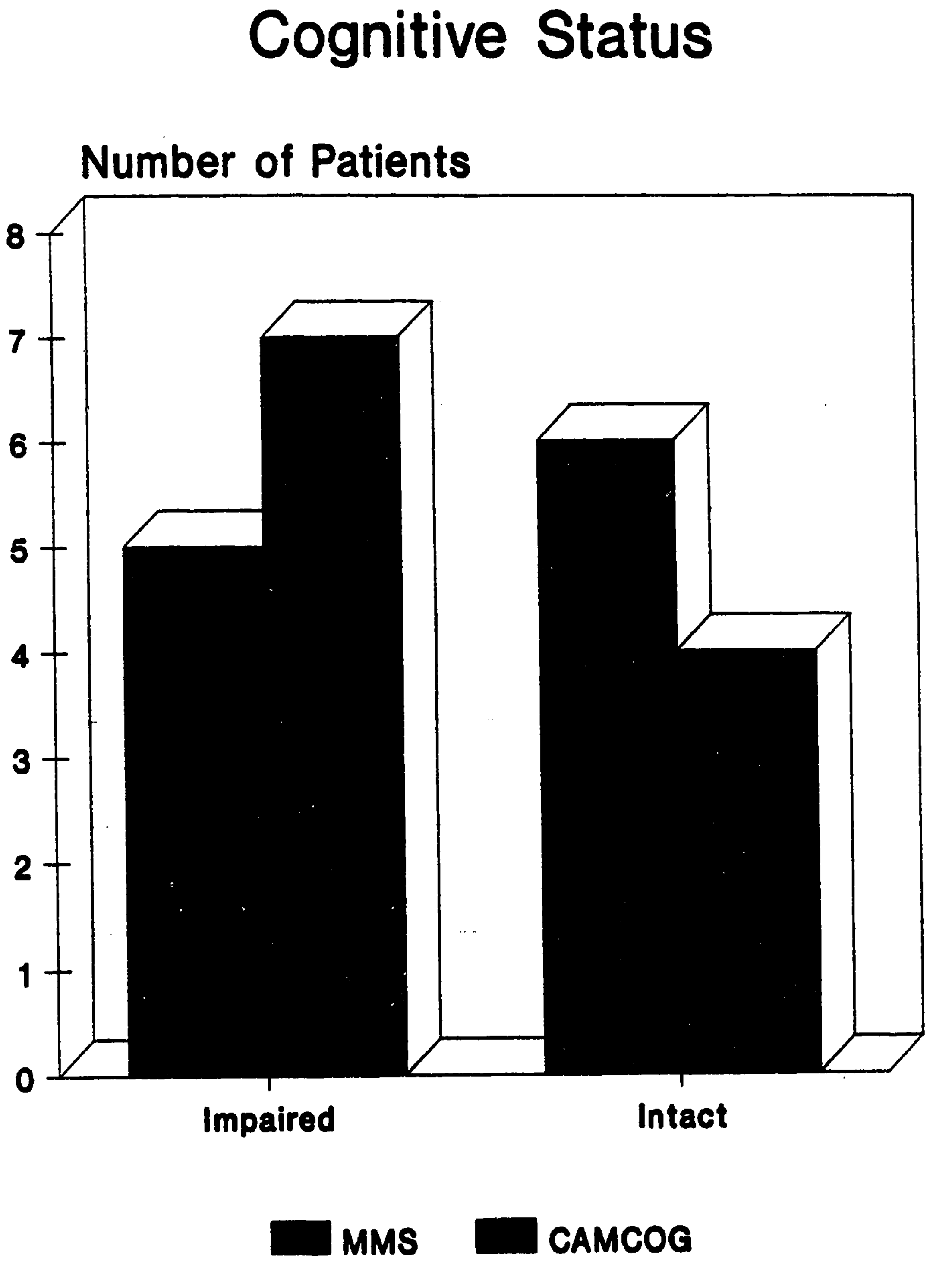


Figure 4: Cognitive Status: A score of 23 or less on the MMS (Folstein Minimental State) indicates cognitive impairment. A score of 79 or less on the CAMCOG (Cambridge Cognitive Exam) indicates impairment.

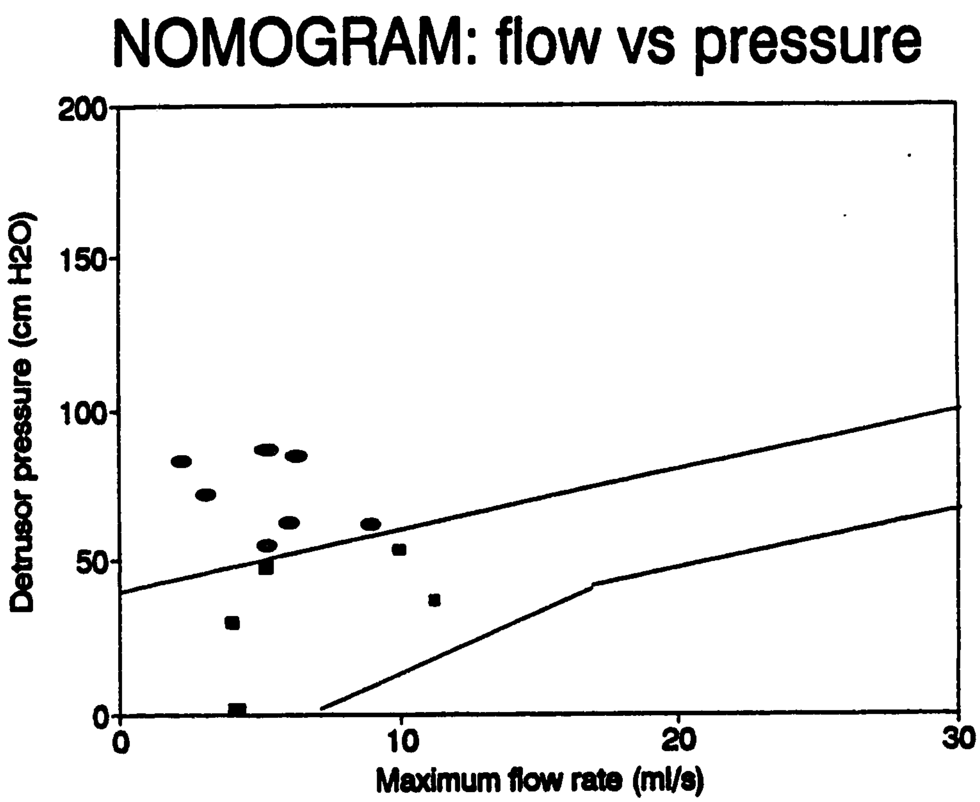


Figure 5: Abrams and Griffiths Nomogram: Patients' preoperative flow rate plotted against preoperative detrusor pressure. Patients represented by ● were obstructed. Patients represented by ■ were equivocal.

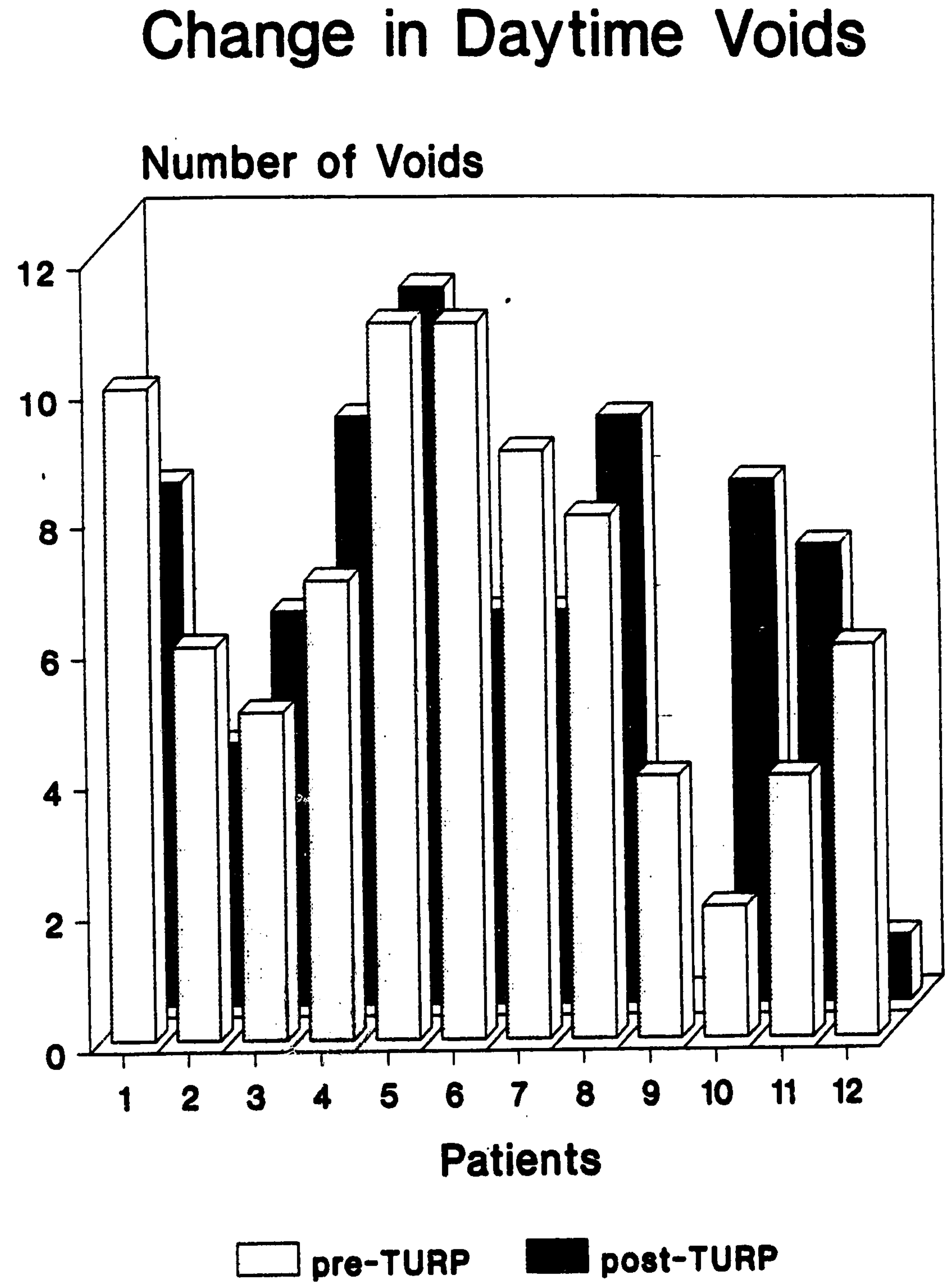


Figure 6: Change in patients' daytime voids, preoperatively and postoperatively. Patient 9 was not tested following his TURP.

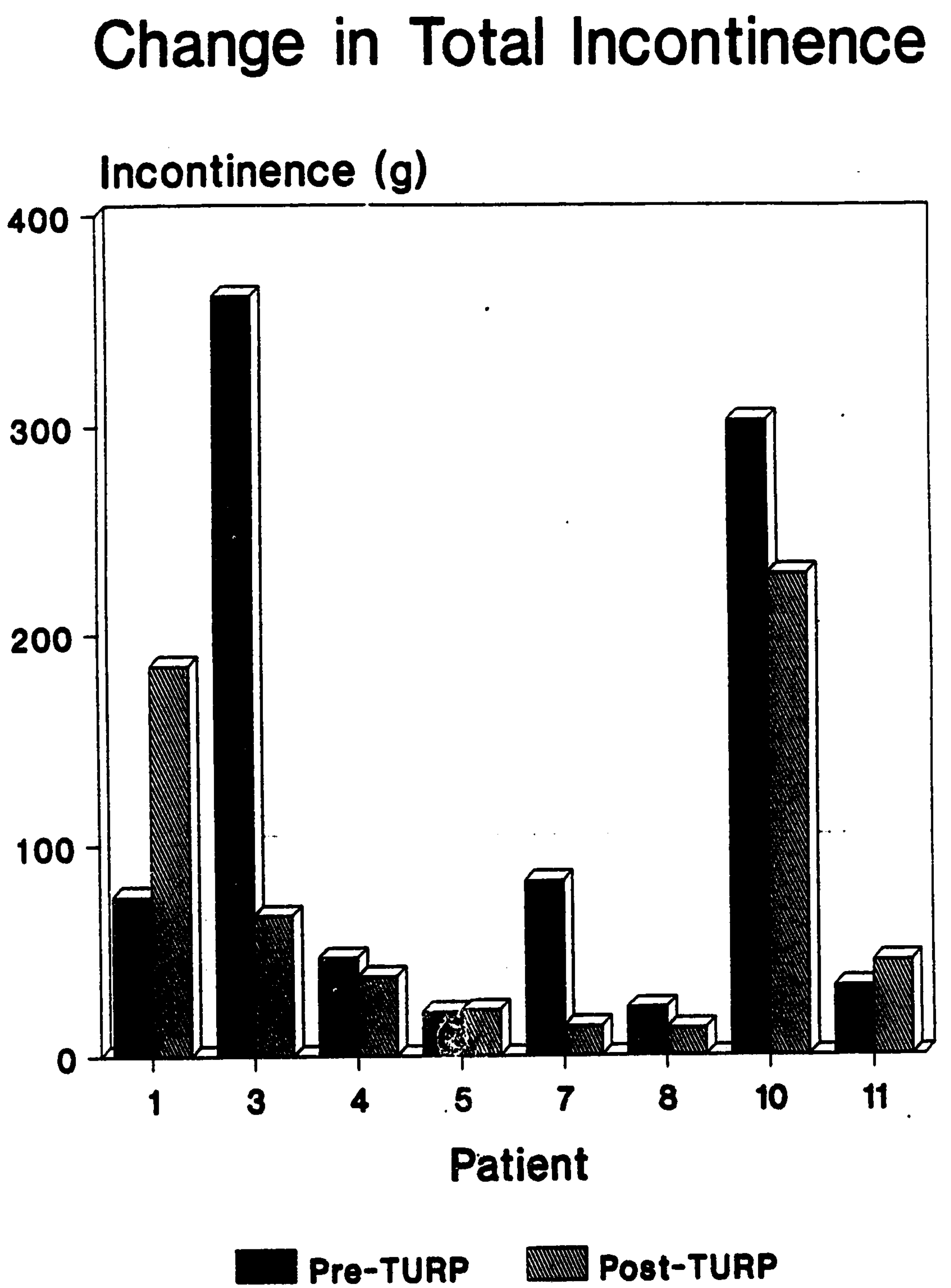


Figure 7a: Change in patients' total incontinence. This graph depicts those patients with preoperative incontinence less than 400 g.

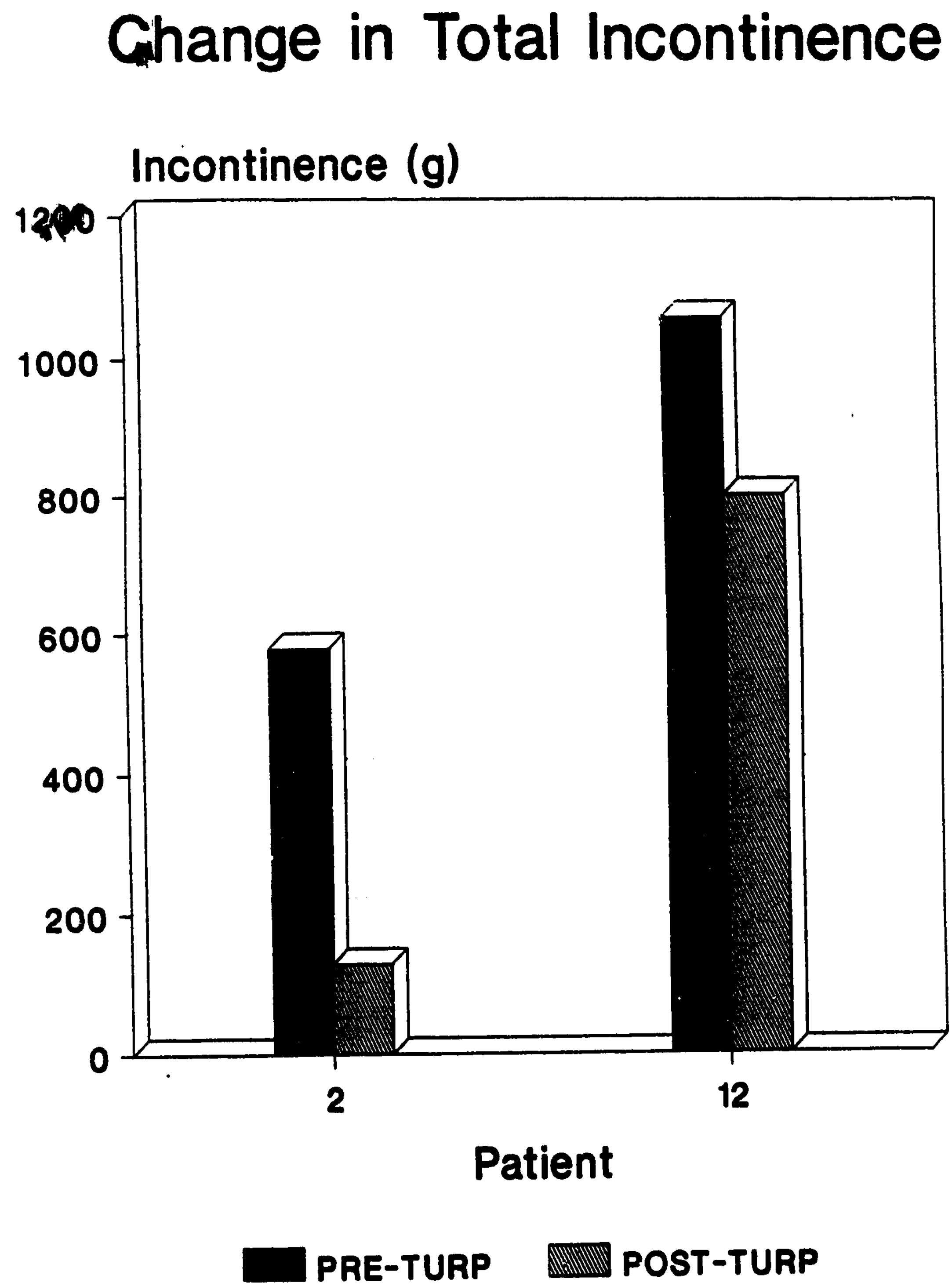


Figure 7b: Change in patients' total incontinence. This graph depicts those patients with preoperative incontinence greater than 400 g.

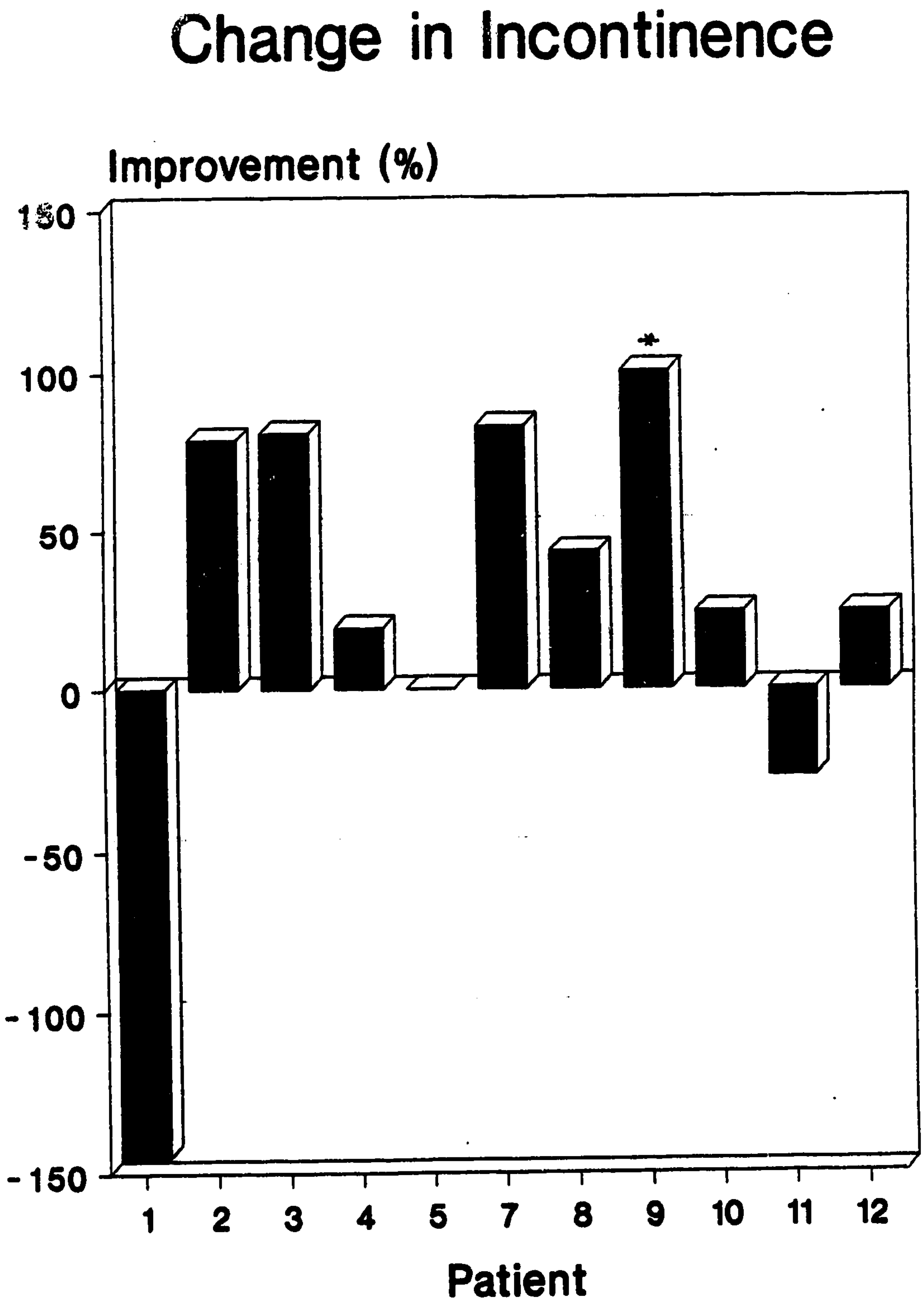


Figure 8a: Percentage change in patients' incontinence after TURP. The asterisk indicates the patient who was not tested postoperatively. By history he was completely improved.

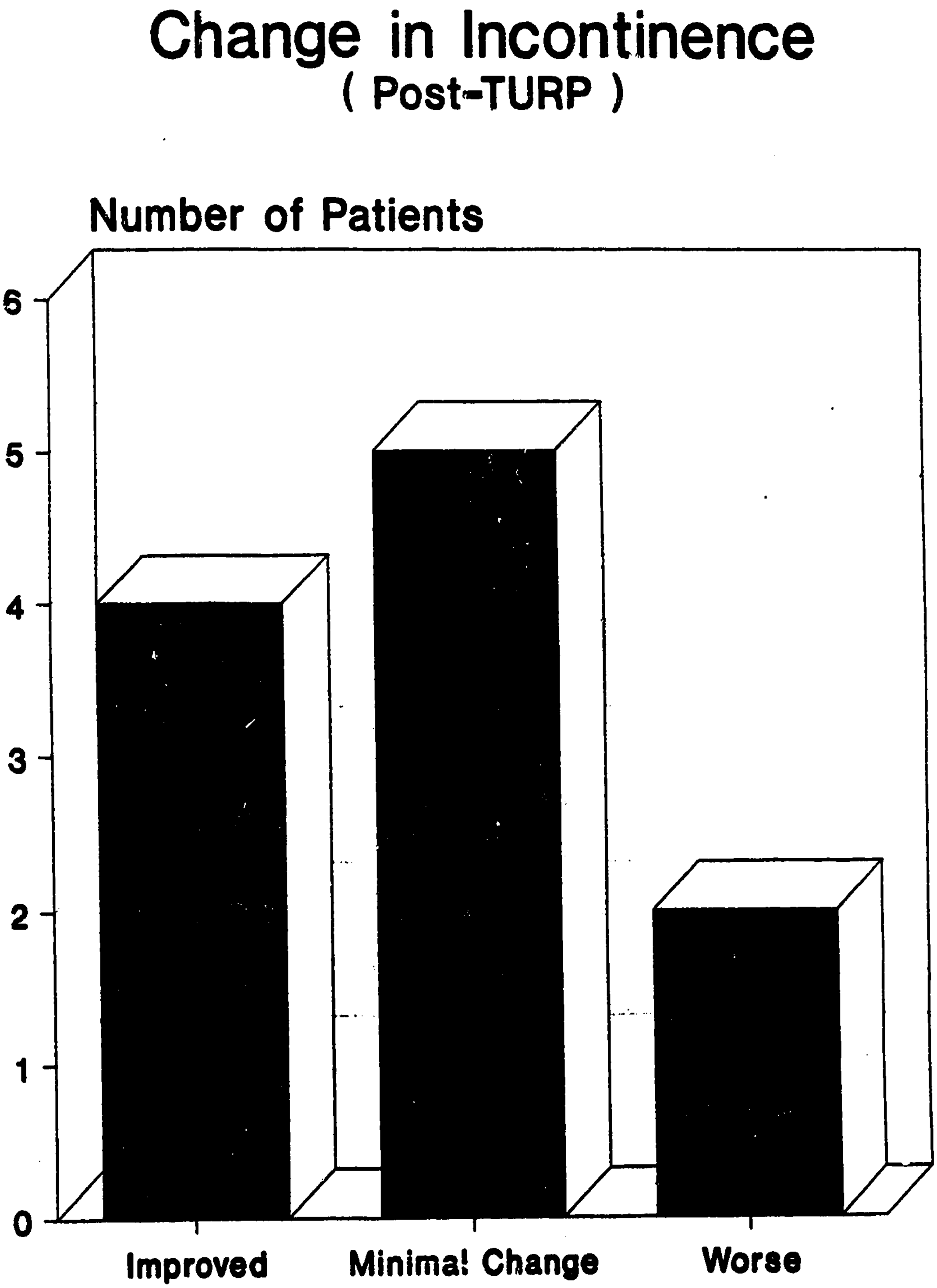
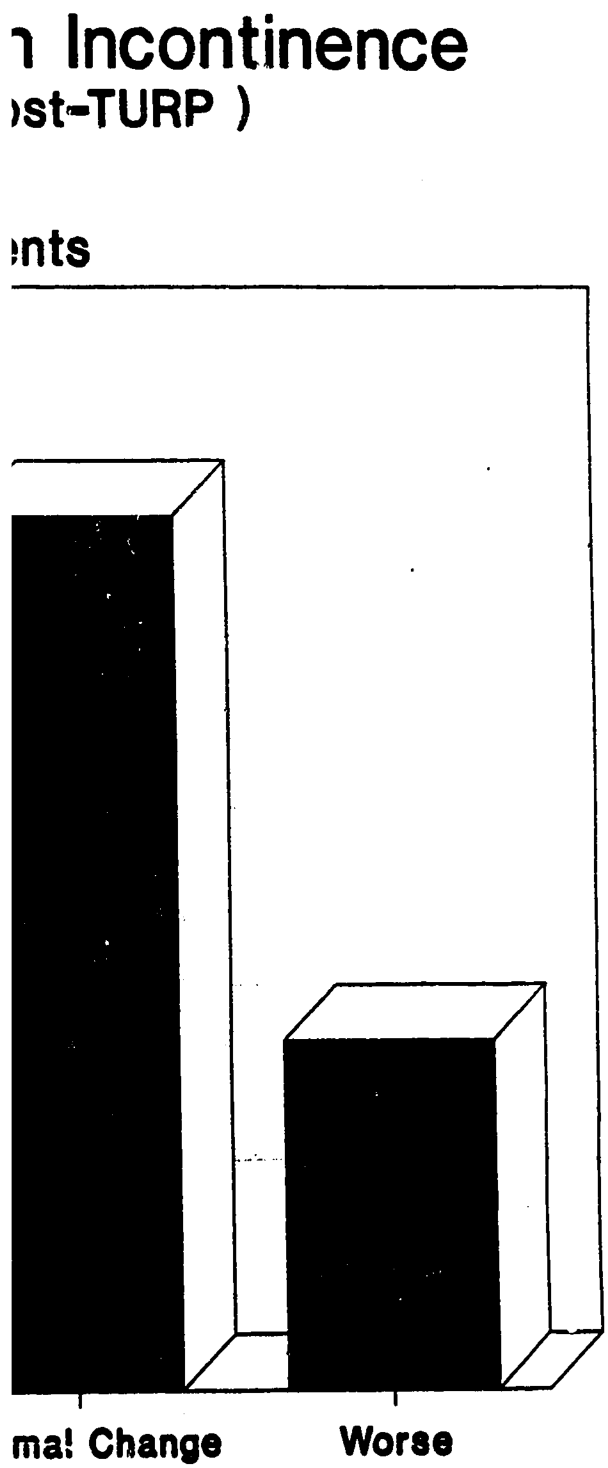


Figure 8b: Change in patients' incontinence after TURP. Four patients had an improvement of more than 50%, 5 patients had minimal change (less than 50% improvement) and 2 patients were more incontinent postoperatively.



ɔntinence after TURP. Four patients had an
ʖ patients had minimal change (less than 50%
ıore incontinent postoperatively.

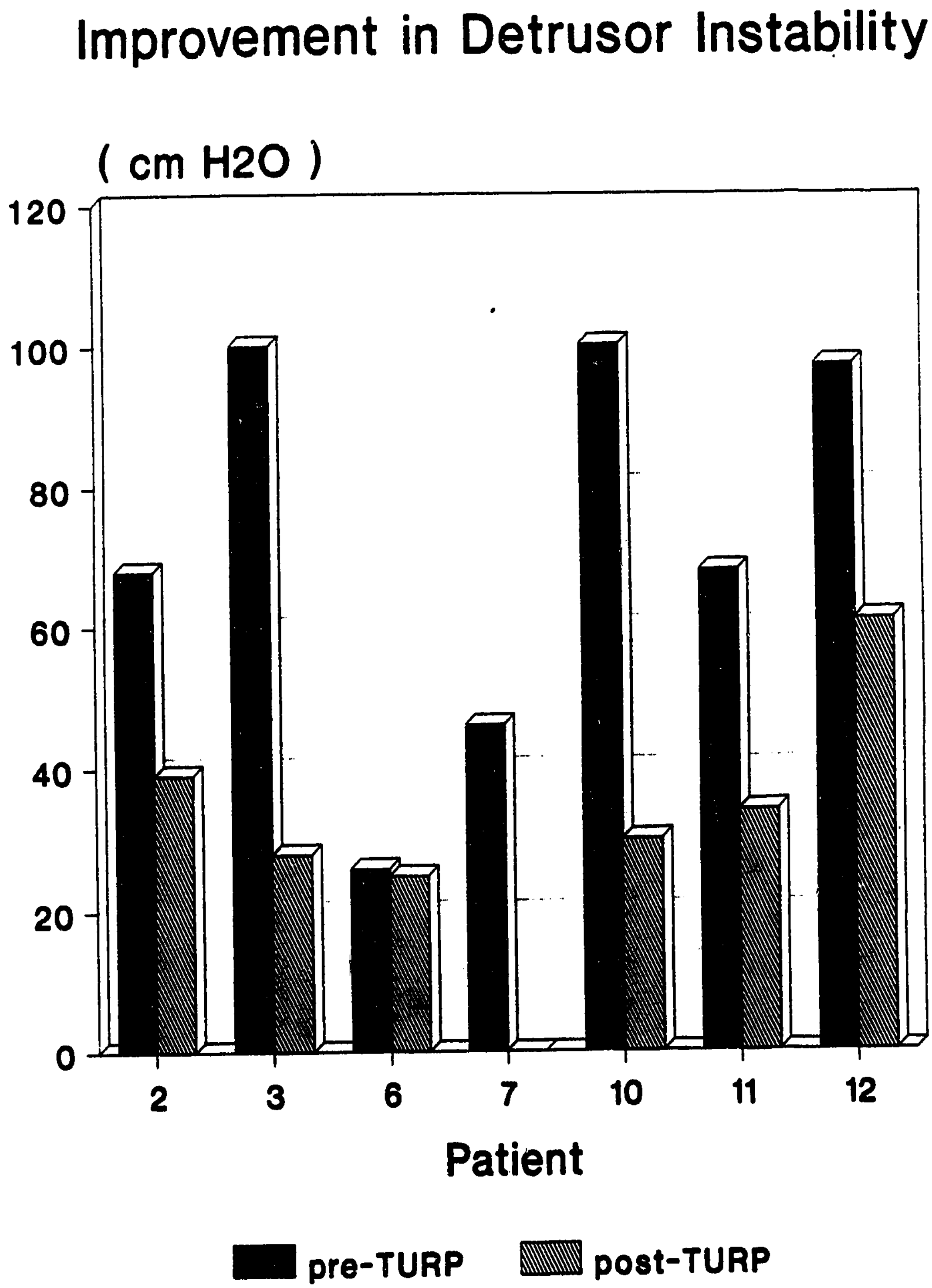


Figure 9a: Change in maximum unstable detrusor pressure following TURP. The 7 patients portrayed on this graph were all improved post-TURP.

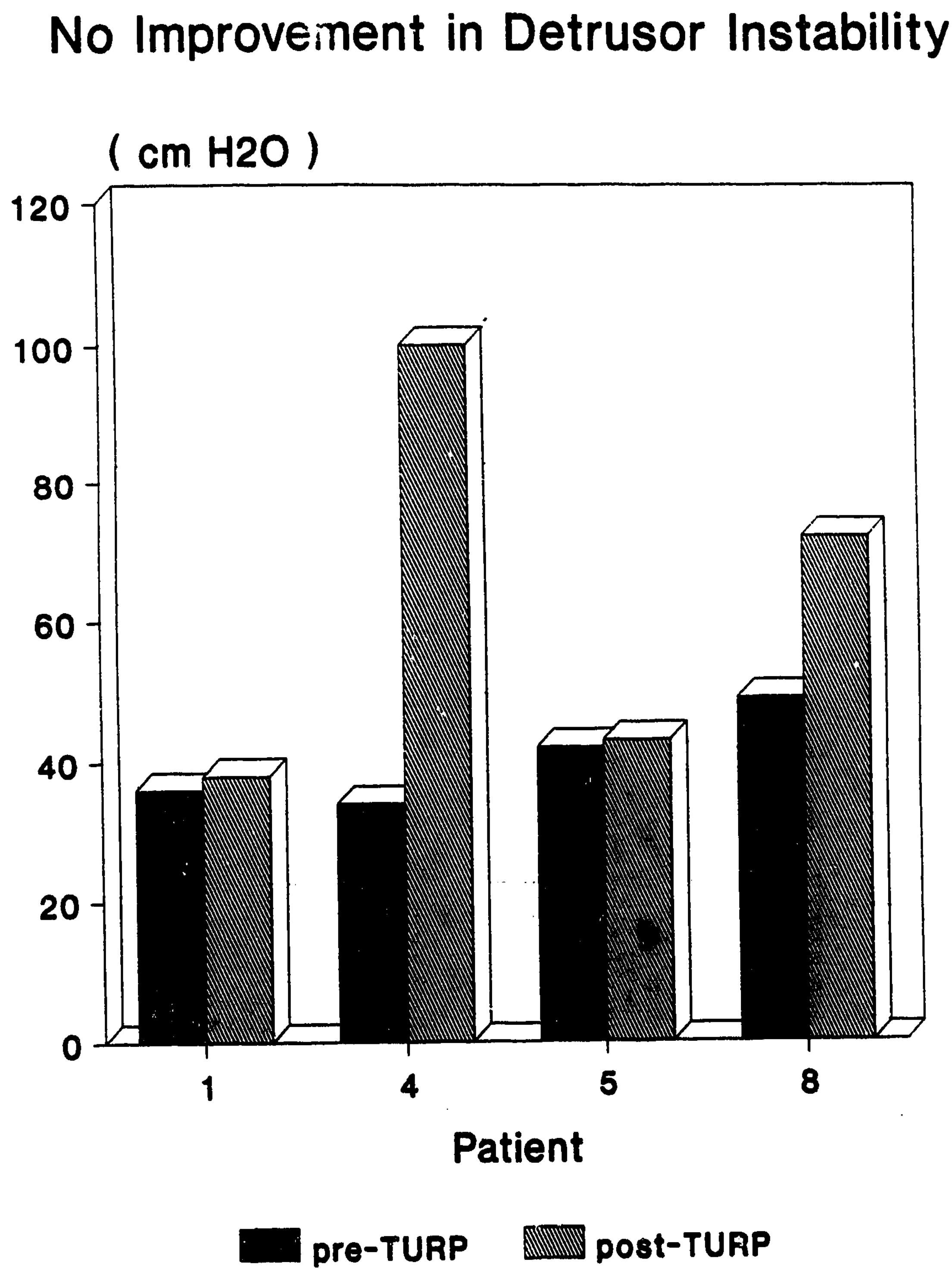


Figure 9b: Change in maximum detrusor pressure following TURP. The 4 patients portrayed on this graph had greater instability post-TURP.

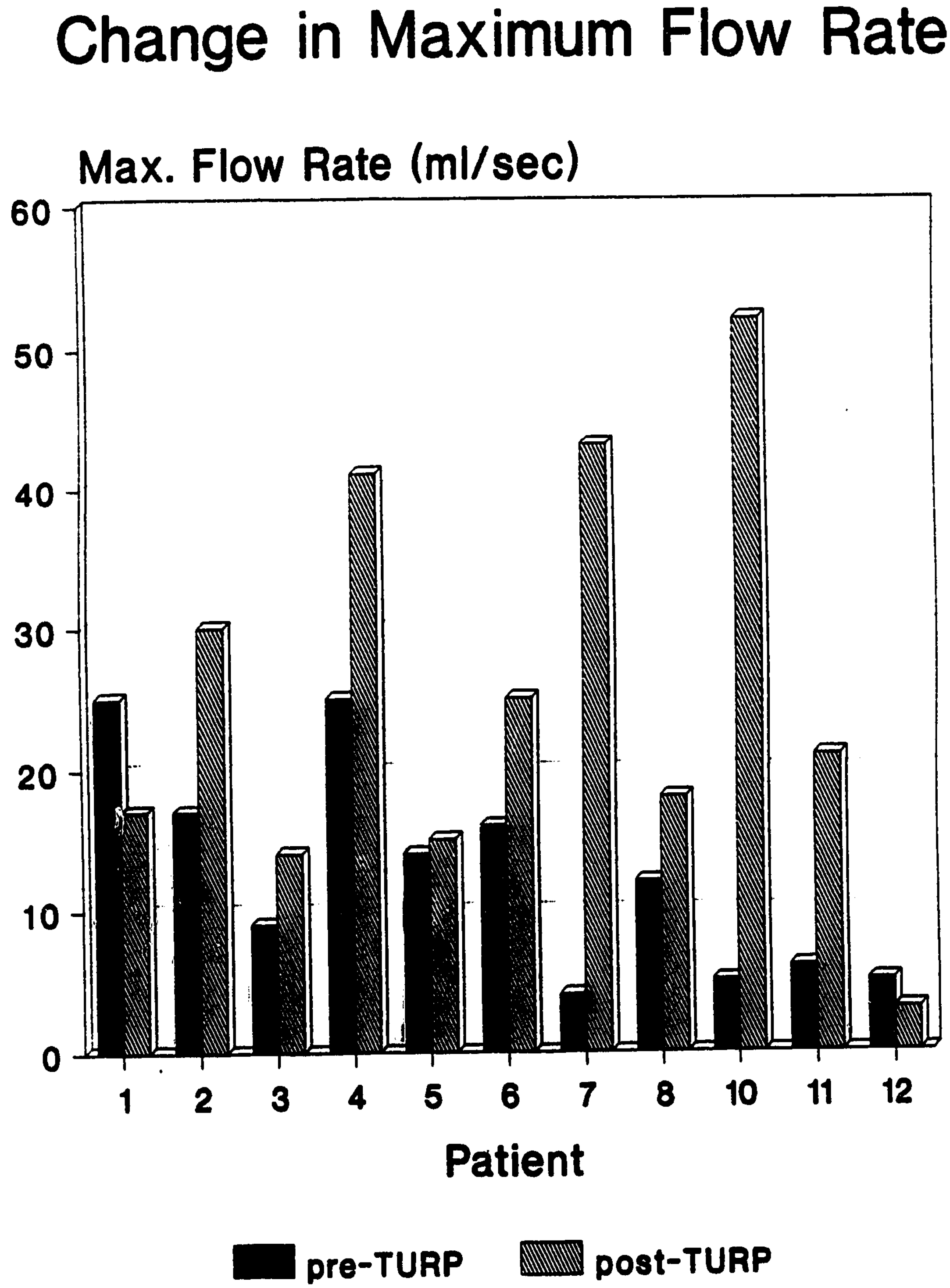
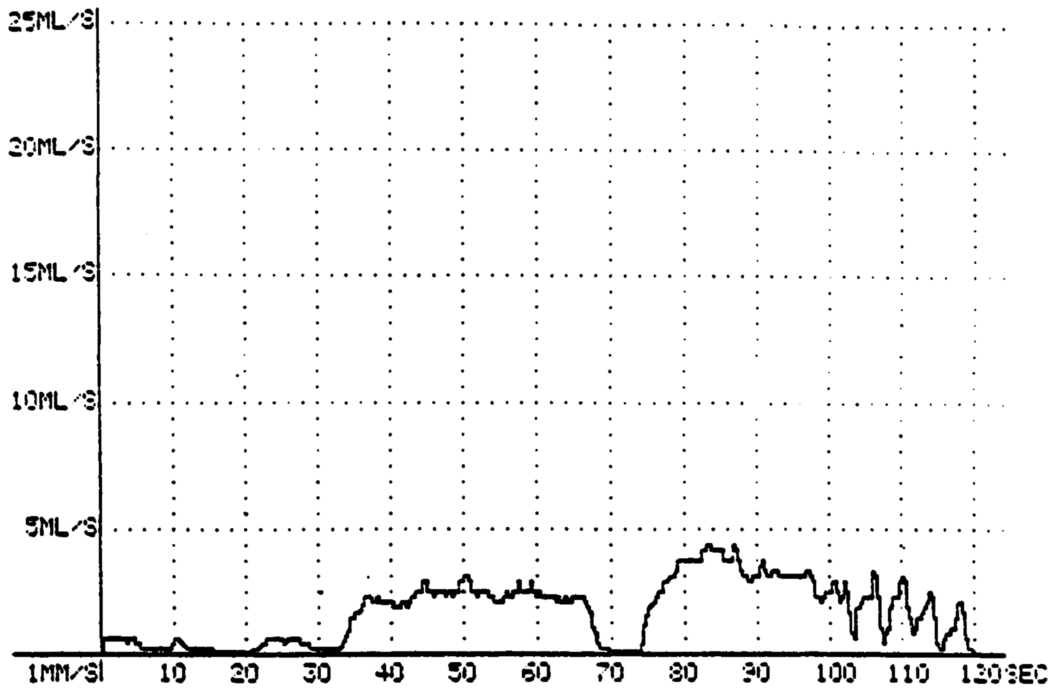
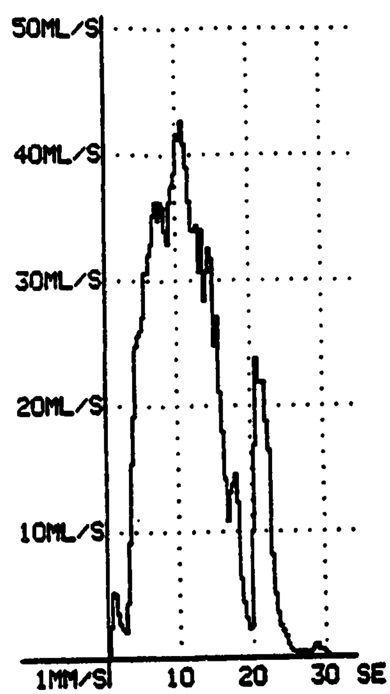


Figure 10: Change in maximum flow rate on 24 h monitoring post-TURP. Nine of 11 patients had an improved flow rate.



a) pre-TURP flow curve



b) post-TURP flow curve

Figure 11: Voiding flow curves for patient 7. Preoperative (a) maximum flow was 4 ml/sec. Postoperative (b) maximum flow increased to 42 ml/sec.

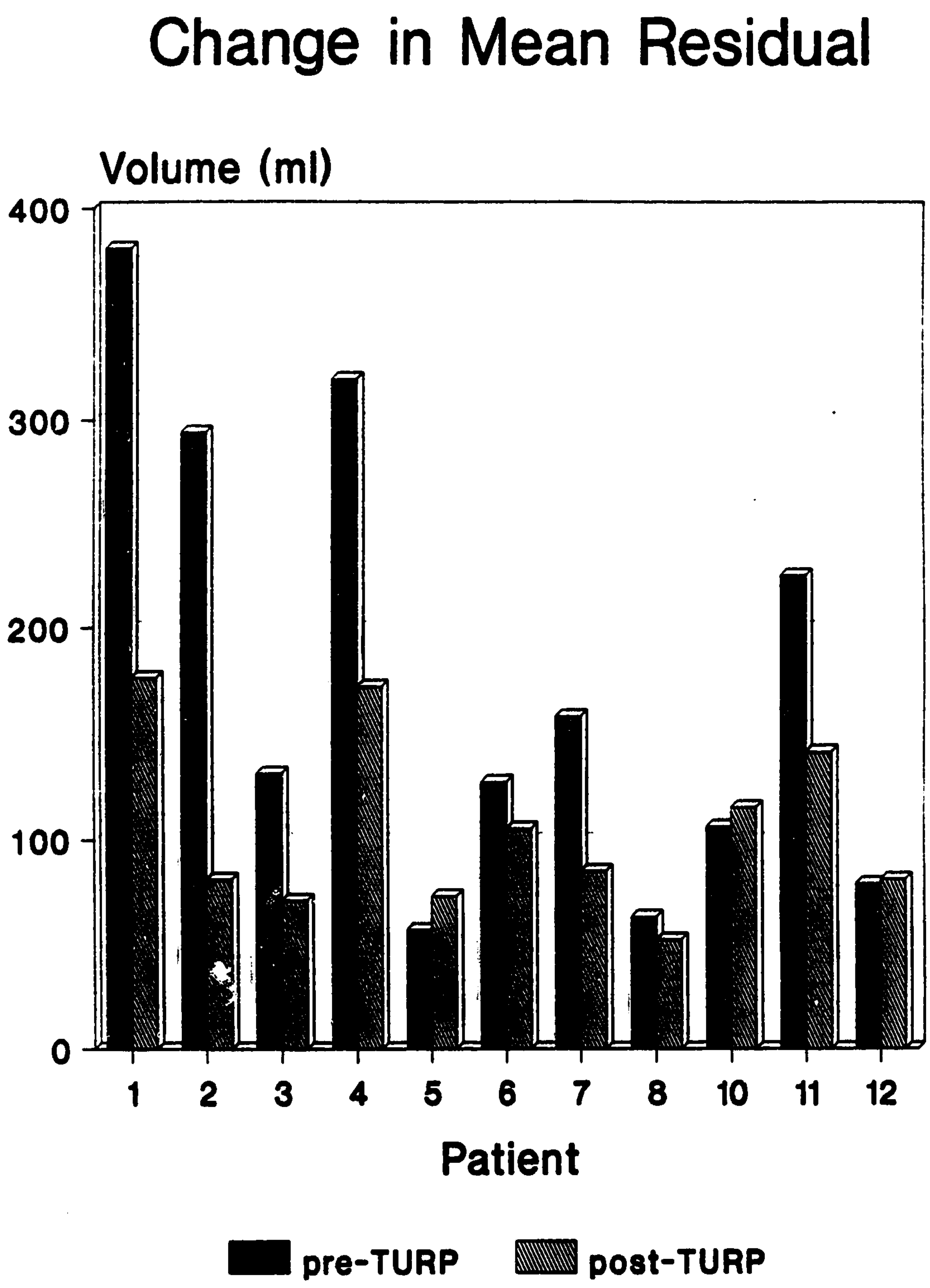


Figure 12: Change in mean residual urine postoperatively. Residual urine was measured by ultrasound 3 times during the 24 h study.

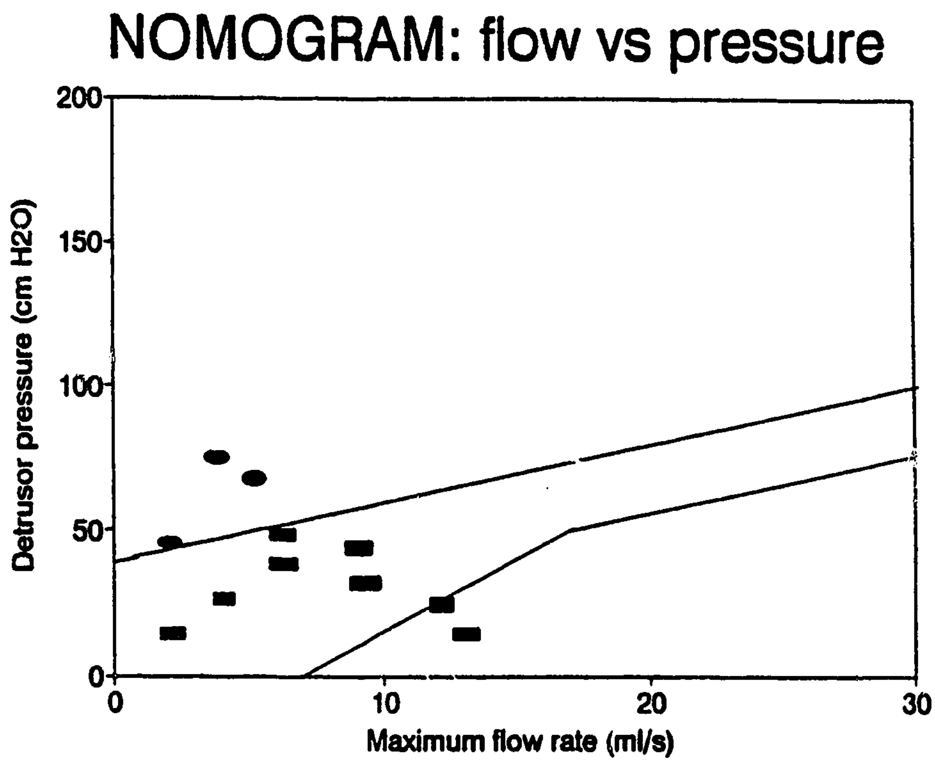


Figure 13: Abrams and Giffiths Nomogram: Patients postoperative flow rate plotted against postoperative detrusor pressure. Postoperatively 2 patients were obstructed ● . The remainder ▬ were equivocal or unobstructed.

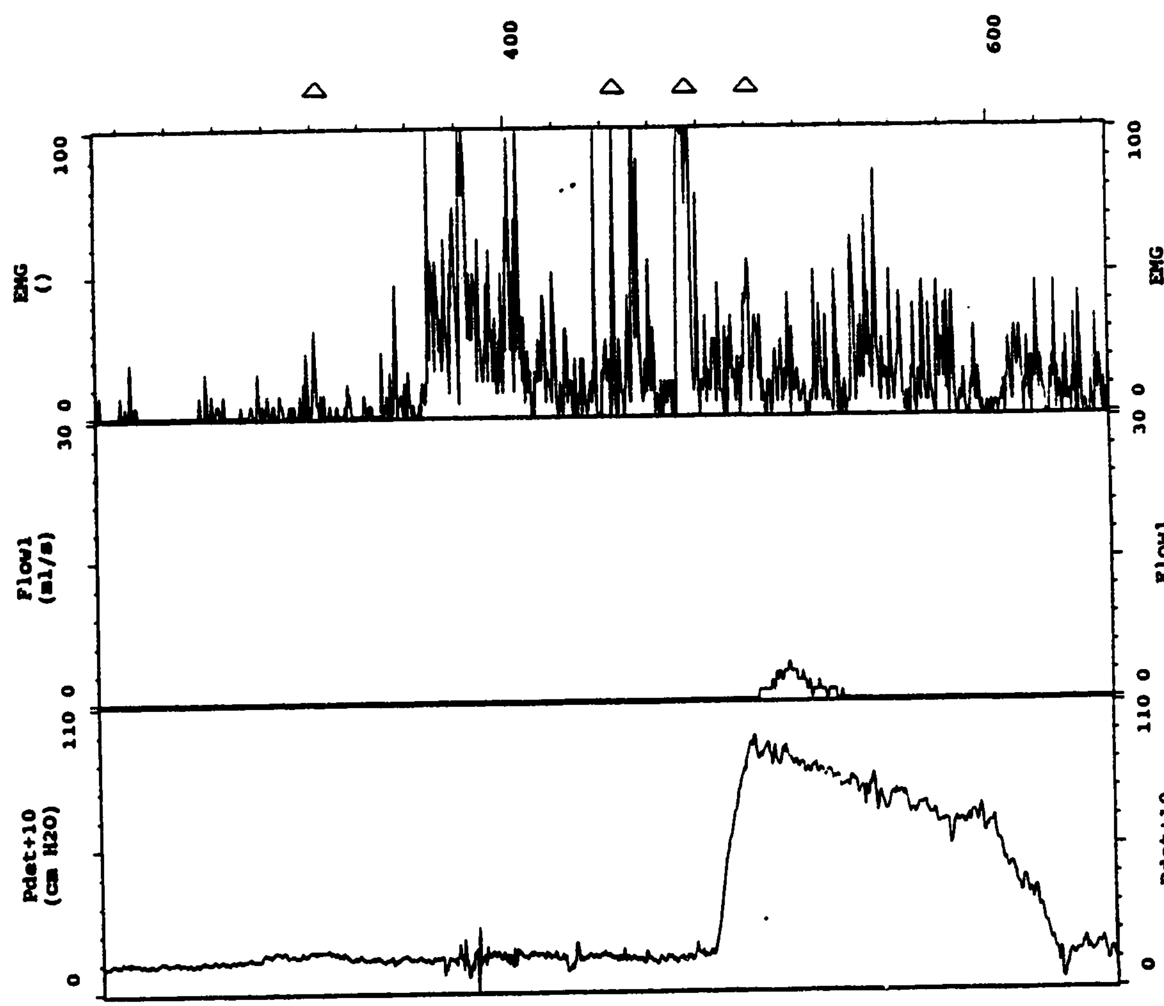


Figure 14: Print out of simultaneously measured sphincter EMG, urinary flow and detrusor pressure. Note that when there is flow of urine there is failure of relaxation of the sphincter.

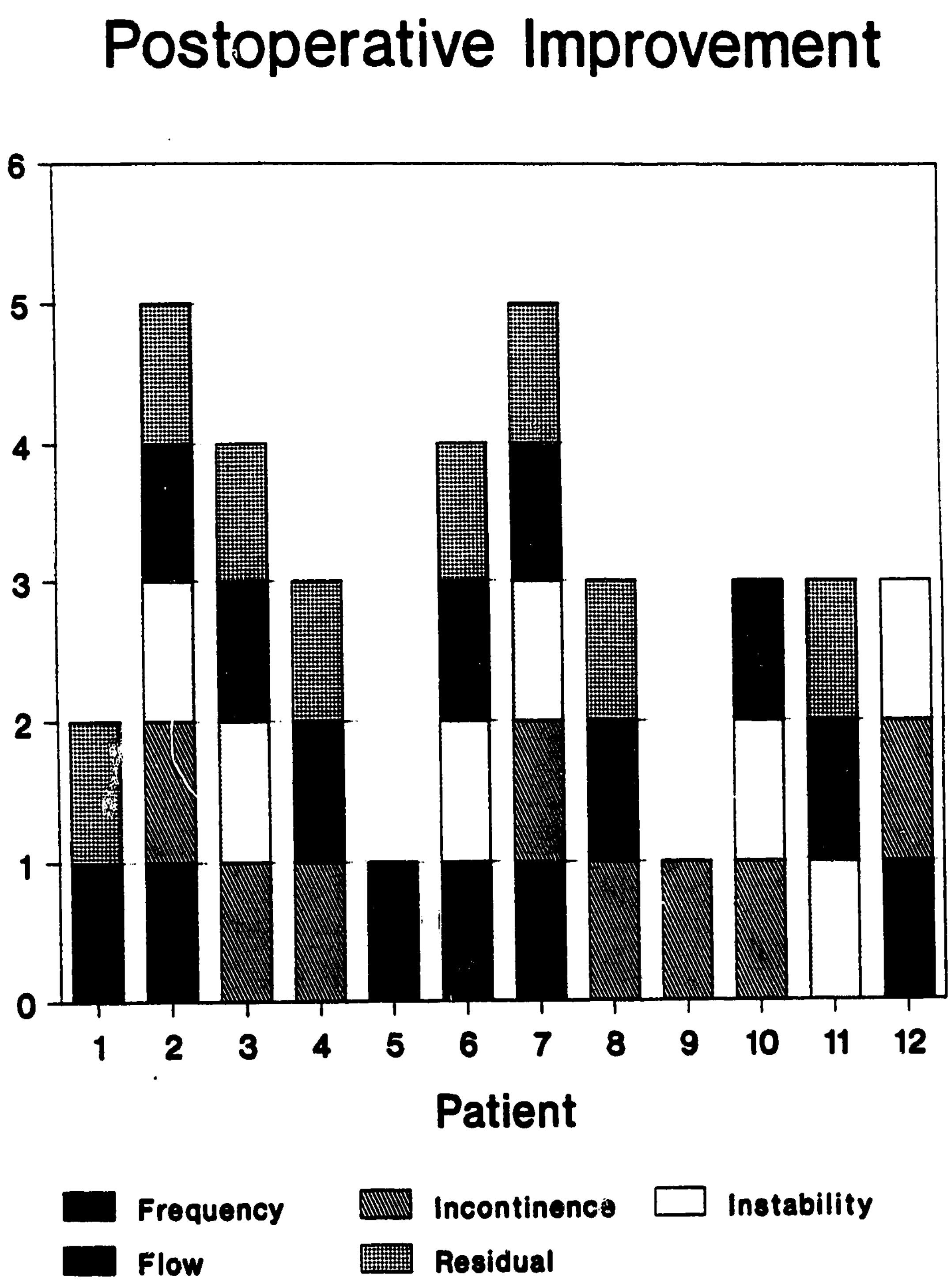


Figure 15: Post-TURP improvement. Each block represents improvement. Only 2 patients, 2 and 7, had improvement in all parameters.

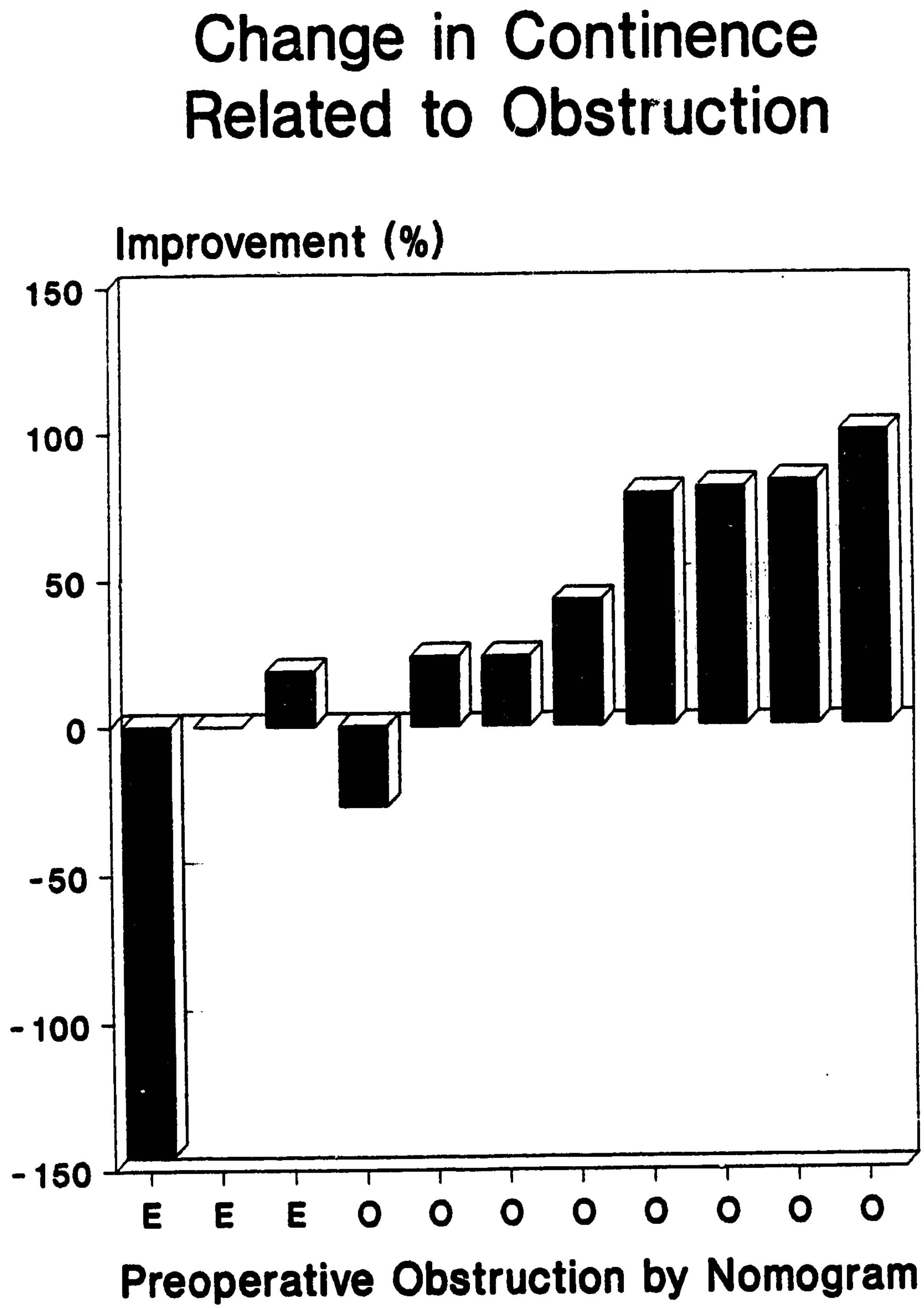


Figure 16a: Change in continence related to preoperative obstruction. Patients who were not obstructed preoperatively (E) have less improvement postoperatively than patients who were obstructed (O).

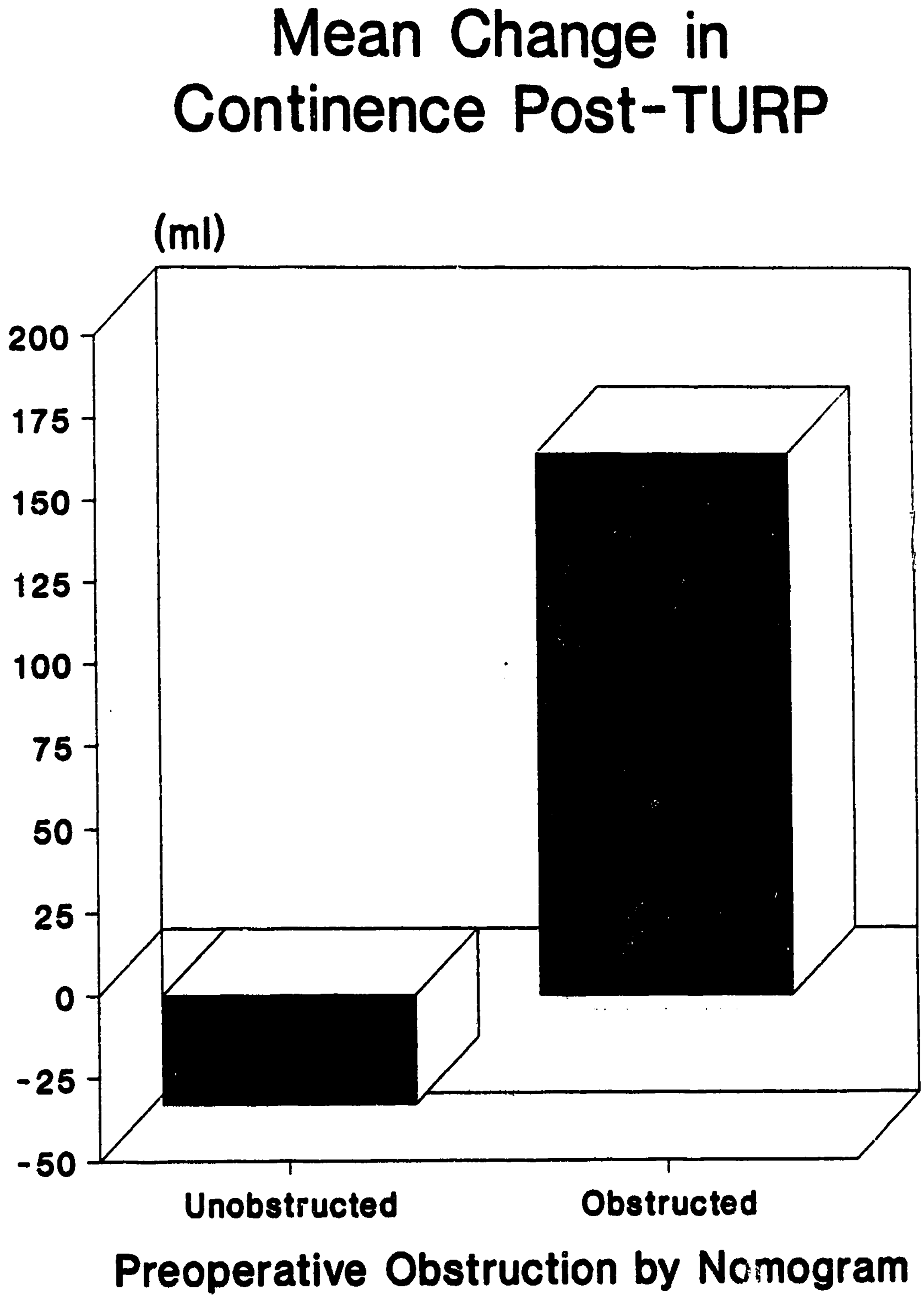


Figure 16b: Mean change in continence postoperatively. The mean change in unobstructed patients was an increase in incontinence of 33 ml. Obstructed patients had a mean improvement in continence of 164 ml.

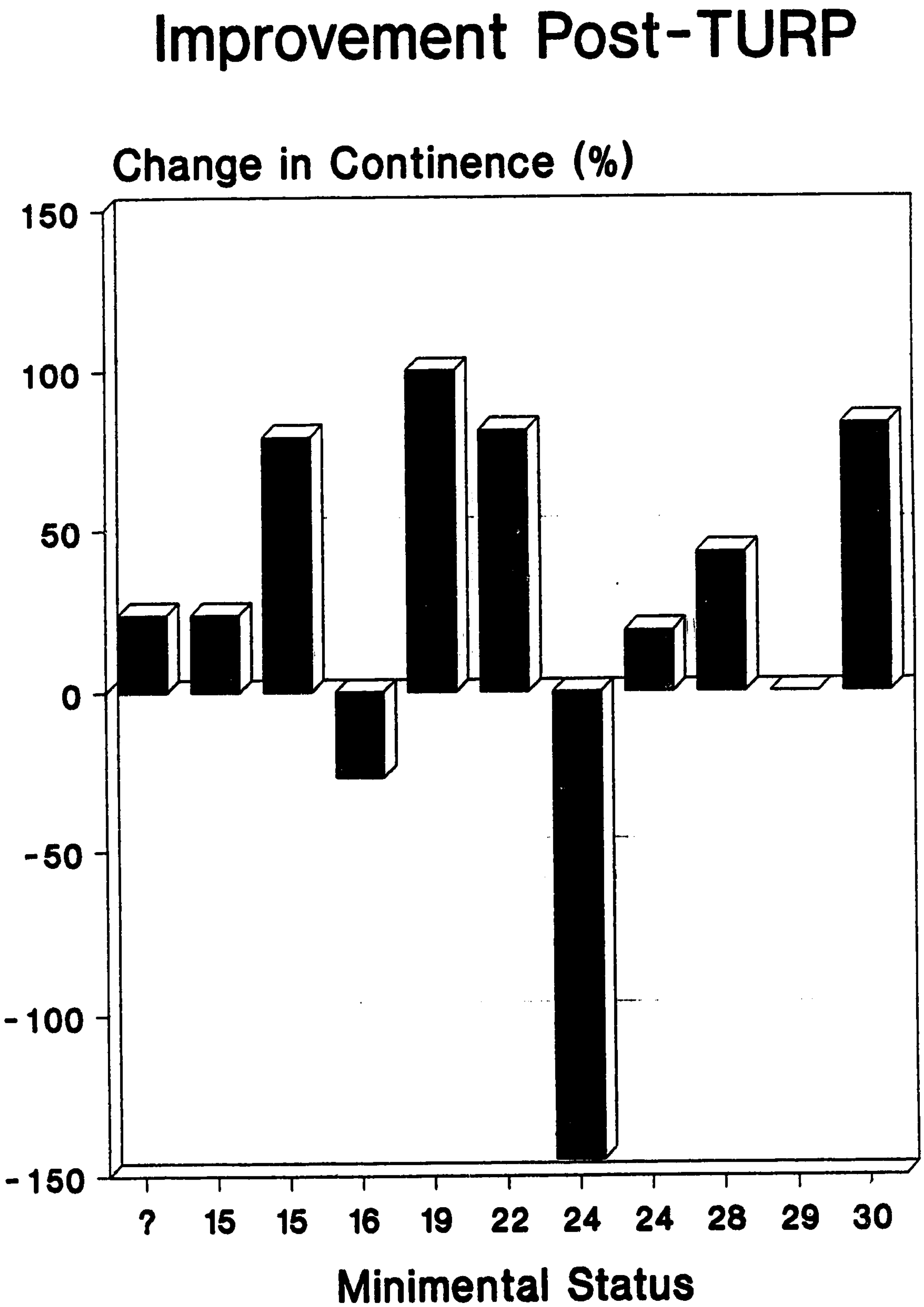


Figure 17a: Postoperative improvement related to cognitive status. Three of the 4 patients who had a significant (>50%) improvement in continence were cognitively impaired (MMS<23).

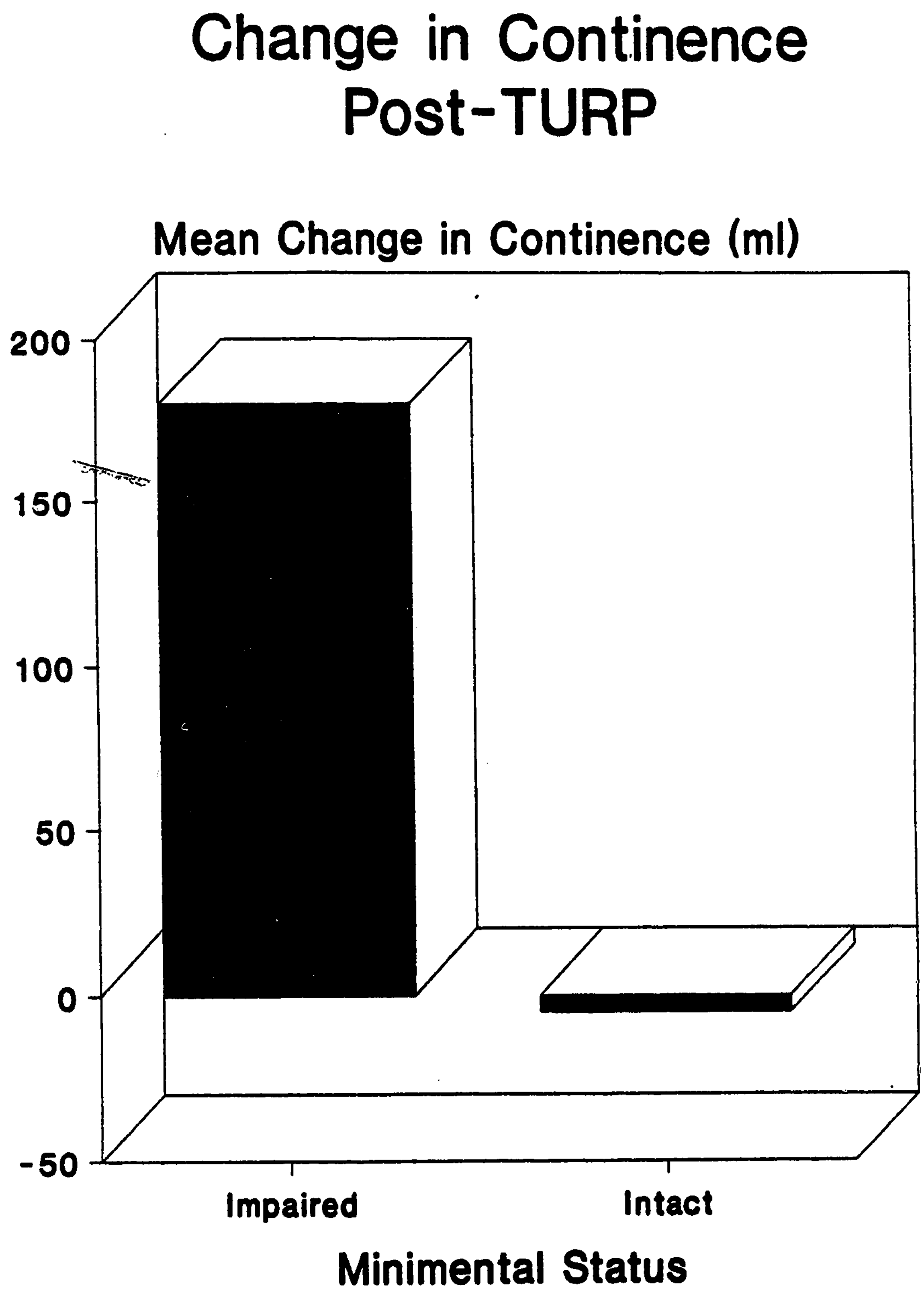


Figure 17b: Change in continence related to preoperative cognitive status. Patients who were cognitively impaired had a greater improvement than patients not impaired.