# CLINICAL STUDIES OF THE DYNAMICS OF MICTURITION

by

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TABLE OF CONTENTS

INTRODUCTION

It has recently become increasingly obvious that the present means (endoscopy and urography) of evaluation of early or minimal cases of lower urinary tract obstruction may be totally inadequate. Endoscopic examination of the lower urinary tract may reveal trabeculation of the bladder, which could be secondary to outlet obstruction, but with no direct evidence of such obstruction. On the other hand, the bladder wall may appear normal in the presence of urethral obstruction. Similarly, urography does not provide the means of adequately evaluating such cases.

Because the degree of obstruction is minimal and appeared gradually, the patient usually cannot be relied upon to relate symptoms to these changes. As well, the physician in evaluating lower urinary tract disease in children, relies upon the history as given by the mother and she may not be a competent observer.

What is needed then is a test for the quantitative evaluation of urethral resistance. Neither the measurement of intravesical pressure or of urine flow alone is the answer. It is well known, for example, that the rate of urine flow may be within normal limits in the presence of obstruction. This is the result of the obstruction being overcome by a high intravesical pressure on voiding due to hypertrophy of the bladder musculature plus contraction of the abdominal muscles. Likewise, voiding pressure may be normal or even low in the case of long-standing outlet obstruction with decompensation of the bladder, but at the expense of rate of urine flow, which decreases to a low value.

The application of Ohm's law to hydrodynamics provides a formula for the measurement of resistance to flow of fluid through a channel. Thus if R represents urethral resistance, P the intravesical pressure and F the rate of urine flow, the formula would be $R = \frac{P}{F}$ .

It is hoped by the measurement of various parameters involved in urination not only to be able to evaluate urethral resistance, and therefore the presence or absence of obstruction, but also to evaluate the dynamics of the entire lower urinary tract before, during and after urination. Such a study will also provide a permanent record of voiding which does not depend upon individual interpretation. It might then be compared with a subsequent study of the same patient to evaluate the results of treatment or the progress of the disease.

A full appreciation of the dynamics of micturition requires a thorough knowledge of the anatomy and physiology of the organs involved. Therefore, before embarking on a review of literature in micturition dynamics and a discussion of our study, it would be appropriate at this point to present the anatomy and physiology of urination.

ANATOMY OF MICTURITION

The act of micturition involves the action and interaction of the bladder, urethra and pelvic diaphragm. Thus, the anatomy and pertinent neuroanatomy of these three structures will be discussed.

I. Bladder:- The urinary bladder has been shown to be composed of a smooth muscle network.[1.] The old concept of three separate and distinct layers of smooth muscle, an inner and an outer longitudinal and a middle circular layer, is no longer acceptable.

The bladder is divided into two parts, the main body or detrusor and the neck or internal sphincter. The structure of the bladder neck, namely a smooth muscle meshwork, is identical to, and continuous with the rest of the bladder. As well, the nerve supply to the two parts of the bladder is identical so that the bladder neck contracts with and not against the detrusor. The previous idea of a sphincter of circular fibers at the neck of the bladder antagonistic in innervation and function to the detrusor is no longer tenable.

The bladder rests on the pelvic diaphragm, which forms its main support. The bladder neck and base are fixed to surrounding structures, but the rest of the bladder is relatively free and mobile. The peritoneum lining the anterior abdominal wall is reflected on to the dome of the bladder.

When the bladder is distended the peritoneal reflection off the anterior abdominal wall is raised well above the symphysis, making it possible to accomplish extraperitoneal entry of the bladder.

II. Urethra:

a) Male:- The male urethra is approximately 25 cm. long and extends from the bladder neck to the external urethral meatus. It is divided into two main parts and each of these into subdivisions as follows:-[2.]

1) Anterior (penile) urethra

i) glandular - 2.5 cm.

ii) pendulous )
) - 15 cm.
iii) bulbous )

2) Posterior urethra

i) membranous - 2 cm.

ii) prostatic - 5.5 cm.

The caliber of the penile portion of the urethra is not uniform, but rather varies from one segment to the next. The external urethral meatus is 8 mm. in diameter, the pendulous urethra is 9-10 mm. and the bulbous urethra is 11-12 mm. in diameter.[2.]

The membranous urethra courses through the urogenital diaphragm and its superior and inferior layers of fascia. The urogenital diaphragm in the deep perineal pouch, consists of two striated (voluntary) muscles, the external urethral sphincter and the transversus perinei profundus.

The external urethral sphincter encircles the urethra as it passes through the deep perineal pouch.

The prostatic urethra courses through the prostate gland, which rests on the urogenital diaphragm between the anterior borders of the pelvic diaphragm. The floor of the prostatic urethra is elevated by the verumontanum.

b) Female:- The female urethra is about 1½ inches long. The internal sphincter of smooth muscle fibers is, as in the male, at the bladder neck. The external sphincter of striated muscle is commonly described encircling the urethra in the deep perineal pouch. It is certainly less well developed than in the male.

III. Pelvic Diaphragm:- The pelvic diaphragm, as mentioned previously, offers the main support to the bladder. This diaphragm is composed of striated muscle (Levator Ani) and arises in front from the back of the body of the pubis, behind from the spine of the ischium, and between these two points from an arched thickening of the fascia of the obturator internus.

The Levator Ani has a free anterior border which is separated from the contralateral muscle by a third of an inch. The urethra, and in the female, the vagina as well, pass between the anterior borders.[3.]

NEUROANATOMY

I. Somatic Nervous System

a) Afferent (sensory) )
) Pudendal nerve S2, 3, 4.
b) Efferent (motor) )

II. Autonomic Nervous System

a) Parasympathetic - Pelvic nerve S2,3,4.

i) Afferent

ii) Efferent

b) Sympathetic

The somatic nervous system is represented by the pudendal nerve, which is a mixed nerve and arises from the second, third and fourth sacral segments. It leaves the pelvis through the greater sciatic foramen to enter the gluteal region. Crossing the ischial spine it re-enters the pelvis by passing through the lesser sciatic foramen to enter the pudendal canal of Alcock. It divides early in the pudendal canal and supplies motor and sensory fibers to the anus. It supplies motor fibers to the muscles of (including the external urethral sphincter) the deep and superficial perineal pouches and to the Levator Ani. Sensory fibers innervate the scrotum, penis, perineum and urethra.

The bladder being a smooth or involuntary muscle structure is naturally innervated by the Autonomic Nervous System. The sympathetic nervous system plays an insignificant role in bladder function. It has been shown that division of the sympathetic fibers to the bladder does not influence urination.

The important nerve supply to the bladder is via the parasympathetic nervous system. The parasympathetic nerves arise as preganglionic fibers from the lateral horns of S2,3,4 and are known as pelvic nerves or nervi erigentes. They proceed to the pelvic plexus where they may synapse or pass through it to synapse with the postganglionic fibers in the bladder wall. The afferent fibers of the parasympathetic nervous system carry sensory impulses from the bladder to the sacral segments of the cord. Thus the reflex center for micturition is at S2,3,4. This center, however, is controlled by higher centers in the brain and there must therefore be pathways in the spinal cord between the higher centers in the brain and the micturition center in the spinal cord. Proprioceptive impulses from the bladder, which include the desire to void and the feeling of bladder fullness, ascend by way of the fasciculus gracilis in the posterior column of the spinal cord. The descending pathway in the spinal cord, carrying inhibitory impulses to the micturition center, is in the pyramidal and extrapyramidal tracts.

The so-called higher centers in the brain concerned with micturition have not been localized with any degree of certainty.

PHYSIOLOGY OF MICTURITION

It has been postulated that there is a constant flow of inhibitory impulses down the pyramidal and extra-pyramidal tracts of the spinal cord from the higher centers in the brain to the micturition center at S2,3, and 4. Let us assume that the frequency of these impulses is represented by 3 negatives as shown in Figure 1.

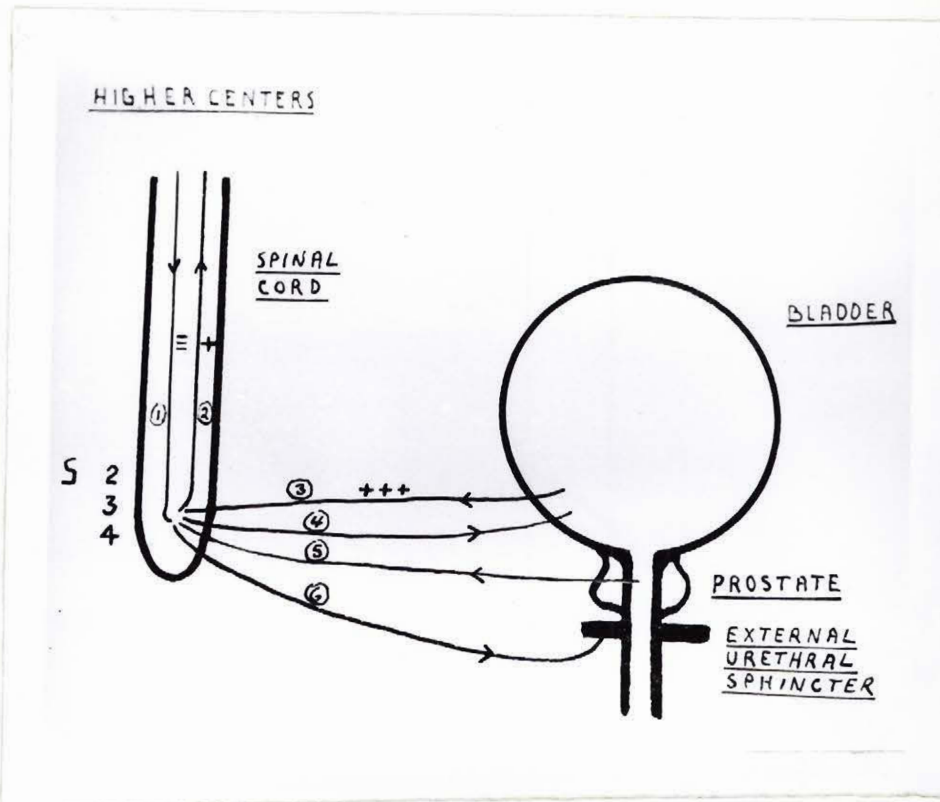



Figure 1 - Neurophysiology of Micturition

1. Pyramidal and extrapyramidal tracts.
2. Posterior column of spinal cord.
3. Parasympathetic afferent ) nervi erigentes
4. Parasympathetic efferent )
5. Somatic afferent ) pudendal nerve
6. Somatic efferent )

As the bladder fills with urine its walls are gradually stretched. A constant low intravesical pressure is maintained as a result of the inherent tone of the smooth muscle. Stretch receptors in the bladder wall are stimulated in increasing numbers. The resultant nervous impulses are carried to the micturition center by the nervi erigentes along parasympathetic afferent fibers. When the frequency of impulses represented by 3 +'s is reached there is still no sensation perceived from the bladder, as the 3 +'s are cancelled by the 3 -'s. With further bladder filling a fourth + appears at the micturition center. This fourth and unchecked + is of insufficient intensity to bring about reflex bladder contraction. Instead it relays to higher centers, via the fasciculus gracilis in the posterior column of the spinal cord, the initial sensation of bladder fullness. This normally occurs with 150 - 250 cc. of bladder filling.

If the individual is not in a location conducive to bladder evacuation, the brain responds by increasing the frequency of inhibitory impulses. This accounts for the common experience of disappearance of the desire to void if the sensation of bladder fullness is suppressed. This may occur several times until eventually the frequency of inhibitory impulses has reached its maximum. At this point bladder capacity has been reached, which in the normal individual represents 500 - 550 cc. of bladder filling. The desire to void is strong and unrelenting and the individual heads for suitable facilities.

At the proper moment the stream of inhibitory impulses is interrupted. The unchecked stream of afferent impulses from the bladder now effects reflex bladder contraction, the efferent limb of the reflex arc being parasympathetic efferent fibers of the nervi erigentes.

The bladder neck contracts simultaneously with the rest of the bladder with which it is continuous. The closed circular internal urethral meatus is opened as the bladder neck contracts and renders the bladder outlet funnel-shaped. This is shown schematically in Figure 2.

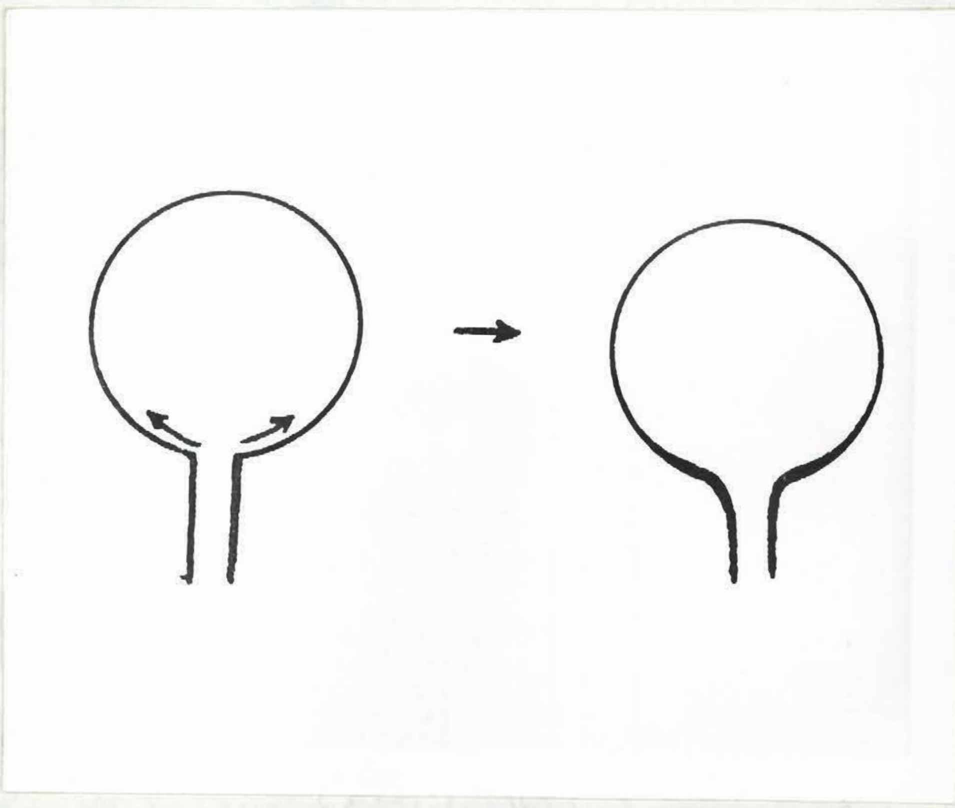


Figure 2 - Opening of the bladder outlet during bladder contraction.

Passive increase in intravesical pressure normally does not suffice to render the internal sphincter patent, however great the volume embraced by the bladder.[4] It would therefore appear that adequate opening of the bladder outlet is only brought about by active bladder (and bladder neck) contraction

When urine enters the posterior urethra, a second reflux results which causes relaxation of the external urethral sphincter and of the pelvic diaphragm. The afferent and efferent limbs of this reflex arc are the sensory and motor fibers respectively of the pudendal nerve. The reflex center is again S2,3, and 4. Relaxation of the pelvic floor brings about a downward and backward movement of the base of the bladder. The bladder outlet is consequently, so to speak, tipped so that the flow of urine from the bladder is facilitated.

It is noted that the internal sphincter opens before the external sphincter and it does this more gradually. At the completion of voiding, contraction of the external sphincter precedes the more gradual closure of the internal sphincter. The external sphincter cannot be relaxed by voluntary effort. It can only be contracted to postpone or stop urination.

Continence is normally maintained by the internal sphincter with reservation of the external sphincter for special situations of increased intravesical pressure or stress, e.g., urgency, straining, coughing, etc. Langworthy[5] found that there was never any incontinence following section of both pudendal nerves in the cat. However, when pressure was applied to the abdomen, urine escaped from the urethra in a large stream. Thus, an incompetent external sphincter results in stress incontinence. With loss of the internal sphincter, such as following prostatectomy or transurethral resection of the bladder neck, the external sphincter assumes the responsibility of maintaining continence. Total incontinence results if both the internal and the external sphincters are injured.

The ability to void and empty one's bladder is determined by the magnitude of two opposing factors. The one, Intravesical Pressure, which tends to force fluid out of the bladder, is opposed by the other, Resistance to flow. In the normal individual, the increase in intravesical pressure with voiding is sufficient to overcome the urethral resistance and effect complete emptying of the bladder. Incomplete emptying or residual urine results when the delicate balance between these two factors is upset. This is seen when there is a decrease of the maximum voiding pressure in the bladder and/or an increase in the resistance to flow of urine through the urethra.

The minimal intravesical pressure required to void against a normal urethral resistance is 30-40 mm. Hg. Not only must this pressure be attained to initiate voiding, but it must be maintained to effect emptying.

Intravesical pressure is the sum of intra-abdominal pressure plus the pressure due to detrusor contraction. The intra-abdominal pressure may be increased by straining or by suprapubic manual pressure (Crede maneuver). This increased intra- abdominal pressure is transmitted directly to the inside of the bladder. The normal individual need not strain to either initiate or maintain voiding. Detrusor contraction alone is sufficient to raise the intravesical pressure to levels which will completely evacuate the bladder. Straining while voiding will only raise the intravesical pressure above already sufficiently high levels with a resultant increase in the rate of urine flow.

The patient with the hypotonic or atonic bladder cannot attain sufficient bladder pressure by detrusor contraction alone. He must rely on straining and/or manual pressure. Sufficiently high pressure may be attained by these means to result in voiding, but it cannot be maintained to cause emptying. Furthermore, as mentioned previously, adequate opening of the bladder neck requires adequate bladder contraction.

Resistance to flow of urine may be increased by:

1) Bladder neck contracture
2) Prostatic hypertrophy or carcinoma
3) Spasticity of external sphincter
4) Stricture of the urethra.

As noted in the introduction the evaluation of urethral resistance entails study of both intravesical pressure and rate of flow.

Calculation of urethral resistance is complicated by the fact that not only does it vary during a single act of urination, as will be shown later, but also from one act to another in the same individual depending on the volume of urine in the bladder at the onset of voiding. Urethral resistance varies inversely with the degree of bladder distension. That is, the greater the initial bladder volume, the greater the opening of the bladder neck on voiding and therefore the less the resistance to flow. This is illustrated in Figure 3.

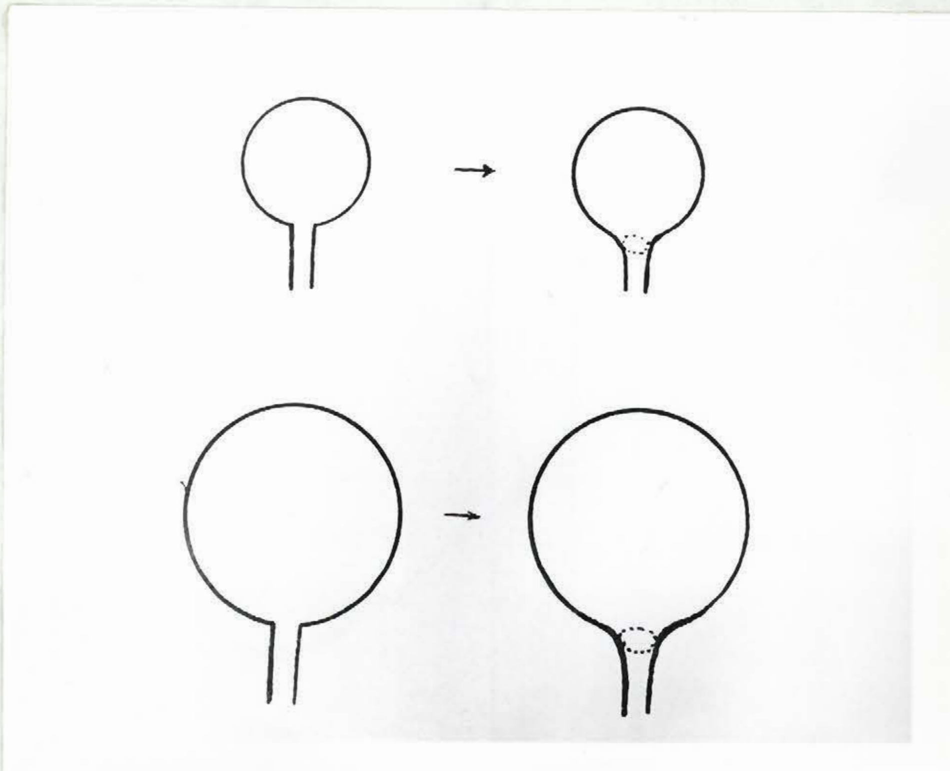


Figure 3 - Degree of bladder neck opening varies directly with bladder volume at onset of micturition.

Langworthy, Kolb and Lewis note that the opening of the internal sphincter occurs earlier during bladder contraction; the greater the volume of fluid in the bladder.[6.]

REVIEW OF LITERATURE

Multi-event recording was first performed by Denny-Brown and Robertson[7] in 1933. They studied the changes in intravesical, intrarectal and abdominal wall pressures simultaneously using a urethral catheter, a balloon inserted rectally and another fastened to the abdominal wall.

The first work of significance in the study of the dynamics of micturition was published by von Garrelts[8] in 1957. Intravesical pressure and flow rates were studied in 10 normal persons. In 3 of these the intra-abdominal pressure was also studied by means of a gastric balloon. A fine polyethylene tube was introduced into the bladder via the urethra and was used to both fill the bladder with saline and to record the intravesical pressure before, during and after voiding. The bladder was refilled with volumes varying between 100-450 cc. so that voiding parameters at various bladder volumes could be studied in the same individual. The conclusion of his studies were as follows:

1) "In the erect position, the pressure in the bladder at rest shows very slight variation with volume in the individual.

2) The varying resting pressures in different persons are probably attributable to different degrees of superimposed abdominal pressure rather than to variations in the tone of the detrusor muscle.

3) The detrusor contraction associated with micturition is not related to the intra-abdominal pressure either for its initiation or its maintenance.

4) The rise in pressure on micturition is fairly constant in the individual and is independent of the volume of the bladder contents.

5) Intravesical pressure falls during micturition, rapidly when the initial volume to be voided is small and slowly when it is large.

6) Bladder pressure is lower after micturition than before.

7) Towards the end of micturition, an 'after contraction' occurs in roughly half the cases; this is a detrusor contraction which is not related to intra-abdominal pressure.

8) The variation in the urinary flow with varying initial bladder volume observed in normal persons (greater when the volume of bladder contents is large and vice versa) is, therefore, not due to changes in micturition pressure with varying bladder volume, but is due to changes in urethral resistance."

A second fine study was that carried out by Cardus, Quesada and Scott.[9.] The purpose of their paper was primarily to present a technique in the study of bladder dynamics. Only one normal and three urologic patients were studied.

Their study of changes in bladder pressure on voiding is more physiological than that of von Garrelts, in that they measured the intravesical pressure by means of a polyethylene catheter inserted into the bladder suprapubically. This leaves the urethra free of a foreign body and therefore more closely approximates the true condition of voiding. The idea of measuring intravesical pressure by means of a suprapubic tube was first introduced by Sandoe and Bryndorf[10.] in 1959.

Urine flow was measured by Cardus et al. with a flowmeter which operates on the principle of electromagnetic induction. Electromyographic activity of the external anal sphincter was recorded using a bipolar needle electrode. As well, the configuration of the bladder and urethra during voiding was studied fluoroscopically.

Murphy and Schoenberg[11.] recorded intravesical pressure via a polyethylene suprapubic catheter. They concluded that patients with urethral obstruction have a higher initial and maximum voiding pressure than do normal individuals.

Much has been written in the literature on the measurement of rate of urine flow which is the parameter which offers the greatest difficulty. Uroflowmeters of varying types and complexity have been used, varying from simple mechanical devices to complex meters operating on the principle of electromagnetic induction.

Kaufman[12.] produced the first practical uroflowmeter, which was a modification of Drake's original machine. It operates on the principle of recording increasing weight (translated into volume) of urine passed per unit time, (cc./sec.) He found that a minimum voided volume of 150 cc.

was necessary for an accurate determination of the urine flow.

Holm[13.] utilized the principle of the anesthetic gas flowmeter in which the volume of urine passed/sec. displaced a corresponding volume of air from a closed container. The displaced air elevated a float indicator to a height corresponding to the rate of flow. He studied 141 normal males and concluded that 15 cc./sec. was the minimal value for maximum flow at bladder volumes of 200 cc. or more.

Stewart[14.] using a Kaufman uroflowmeter found that 20% of normal individuals voided with maximum flow rates as low as 15-20 cc./sec., while 20% of obviously obstructed individuals voided with maximum flow rates as high as 15-20 cc./sec. He therefore concludes that a maximum flow rate of over 23 cc./sec. is probably normal, but that a flow rate of below 23 cc./sec. may be normal or may indicate obstruction, bladder weakness, dysuria or psychic inhibitions.

Scott and McIlhaney[15.] studied 139 normal females and 169 normal males. They found that in the normal adult male and female there is considerable variation in voiding rate with constant as well as varying bladder volumes. The variation was most pronounced with volumes less than 200 cc., but significant variations did occur with volumes in excess of 200 cc.

Studying children[16.] these same two authors found that urine volumes in excess of 100 cc. appeared to be adequate for accurate determination of voiding rate. 78 children between the ages of 4-12 were examined. They found a strong positive correlation between maximum voiding rate and body surface area but not between maximum voiding rate and age. The voiding rate in the group studied varied between 10-20 cc./sec.

Lattimer and Gleason[17.] were the first to attempt to calculate urethral resistance. 19 normal patients, of which 12 were children, were studied. Intravesical pressure was measured by a small polyethylene tube inserted into the bladder per urethra and connected to a Lewis cystometer. Flow rate was measured with a Kaufman flowmeter. The study consisted of filling the bladder to the point of extreme urgency via the urethral catheter which was then connected to the cystometer. The patient would then void around the urethral catheter into the flowmeter.

Urethral resistance was calculated by dividing the maximum voiding pressure by the maximum flow rate. The upper limits of normal were found, in this study, to be $3 \times 10^3$ $dyne.sec.cm^{-5}$.

Pierce et al.[18.] studied 42 normal adult males and 5 pre- and post-prostatectomy males. Intravesical pressure was measured here as well by a transurethral catheter.

They note that the equation $R = \frac{P}{F}$ is valid for the calculation of urethral resistance, in spite of the fact that the radius and length of the urethra do not enter into the calculation.

Poiseuille's law for the calculation of flow through a channel takes into consideration the length and radius of the channel.

$$\text{Thus } \frac{P}{F} = K\frac{l}{r^4}$$

The ratio of P to F, which equals R, therefore takes into consideration differences in length and radius of the conduit and so can be ignored.

Their measurements of urethral resistance in the adult male were:

5-7 Gm.sec.$cm^{-5}$, as the normal range, 10-15 Gm.sec.$cm^{-5}$, as indication of early obstruction and, 16-25 Gm.sec.$cm^{-5}$, as indication of moderate obstruction.

Arbuckle and Paquin[19.] studied 27 normal females using a transurethral catheter to both fill the bladder and then to record intravesical pressure as the subject voids around the catheter.

They found that the average urethral resistance decreased slightly with increasing voided volumes from 5.46 to 3.38 dyne.sec. $cm^{-5} \times 10^3$ for a range from 100-600 cc. The minimal urethral resistance for the same range was 3.38 to 2.08 dyne.sec.$cm^{-5} \times 10^3$. The upper limits of normal for average and minimal urethral resistance were 8.32 and 4.42 dyne.sec.$cm^{-5} \times 10^3$ respectively. These authors note that Pierce and Braun obtained considerably higher resistance values. They attribute this difference to the slightly larger urethral catheters used.

Ritter and associates[20] question the validity of using the equation $R = \frac{P}{F}$ to represent urethral resistance. They reason that the urine flow is turbulent and therefore the equation should be replaced by a similar relationship which holds for friction loss due to turbulent flow. The equation they offer as an alternative is complex and would only be applicable to the female. Because of the greater length and varying diameter of the male urethra the calculation of urethral resistance in the male would then be infinitely complex or impossible.

TECHNIQUE OF THE STUDY

On the day prior to the performance of the study, a small suprapubic tube is inserted into the bladder, using the Seldinger needle technique. The Seldinger technique involves puncture of the distended bladder with a stylet needle. (Figure 4)

Figure 4. Seldinger needle (PE - 160 No. 17), flexible wire mandrin and Oedman Ledin catheter (6-10 inches long).

The site of entry of the needle is in the midline of the abdomen, one to two finger-breadths above the symphysis pubis. Correct placement of the needle is verified by the flow of fluid from the bladder when the stylet is removed.

A soft wire mandrin is then introduced through the lumen of the needle and advanced well into the bladder. The needle is withdrawn leaving the mandrin in situ. The catheter, which is of the same gauge as the needle, is then threaded on the mandrin and gently advanced through the abdominal and bladder walls until it is judged that 3-4 inches of the catheter has entered the bladder. The mandrin is then withdrawn and the suprapubic catheter taped in place.

This procedure can be safely carried out provided:

1) Patients with suprapubic scars are either eliminated from the study or handled with great caution.

2) The bladder capacity is at least 300 cc.

Placement of the tube requires previous distension of the bladder. Every effort is made to distend the bladder physiologically by having the patient drink ample fluids and refrain from voiding. When the patient complains of discomfort and a strong desire to void, he is examined for a suprapubic mass with dullness to percussion in this area. If the physical signs are present and unequivocal, the suprapubic tube may then be safely and easily introduced under local anesthesia (2% xylocaine).

The advantage of allowing the bladder to distend physiologically is that the urethra is not traumatized by catheterization. The tested act of voiding will therefore more closely approximate the usual voiding pattern for that patient. The disadvantages of this method are:

1) The time required to distend the bladder sufficiently (up to several hours) during which time the doctor must be available.

2) The uncertainty of bladder distension in certain cases.

3) Inability to insert the suprapubic tube in cases of spastic neurogenic bladder dysfunction because of small bladder capacity.

In the latter two instances the alternate method of distending the bladder is chosen. A Foley catheter is introduced trans-urethrally and the bladder distended with a known volume of sterile saline. In the case of the spastic neurogenic bladder, 30 mg. of Probanthine is given intravenously to relax the bladder and increase its capacity temporarily. Following the insertion of the suprapubic tube, the Foley catheter is removed. That night the patient is given a cleansing enema and the perianal region is shaved.

On the morning of the following day, the patient is given two glasses of water to drink with his breakfast. An intravenous of 10% Mannitol (Osmitrol) is started at the bedside and run at about 60 drops per minute. About 1 hour later, the patient is brought to the test room, where a rectal balloon is inserted and distended with 15cc. of water. (Figure 5 and Figure 6). He is then seated upon a specially constructed chair and the bladder and rectal tubes attached to the respective pressure transducers.

A 2 inch co-axial needle electrode (Figure 7) is inserted perineally and advanced towards the apex of the


Figure 5 - Rectal balloon (finger cut from rubber glove) collapsed prior to insertion into rectum.


Figure 6 - Rectal balloon distended with 15 cc. water following insertion into rectum.

Figure 7 - Co-axial needle electrode.

prostate, guided by an examining finger in the rectum. In the female the electrode is placed in the bulbo-cavernosus muscle. The sound and sight of the characteristic firing of striated muscle motor units is listened and watched for on the loudspeaker and oscilloscope screen respectively. (Figure 8). When the electrode is felt to be in proper position, (the external urethral sphincter in the male), it is taped in place. (Figure 9).

Finally, the funnel of the flow-meter is placed so that the urinary stream can be caught and recorded. (Fig. 10).

The two pressure transducers, (rectal and bladder), the flow-meter and the oscilloscope are connected to their respective channels on the polygraph recorder (Figure 11) and the four parameters studied are recorded simultaneously.

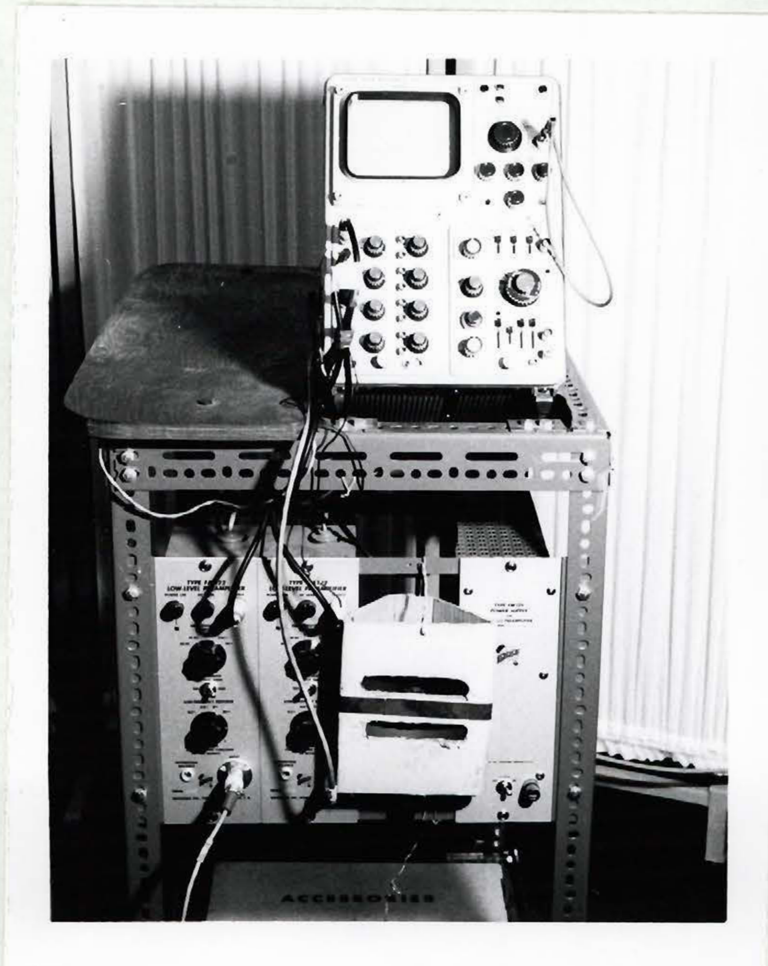


Figure 8 - Tektronix oscilloscope and loudspeaker.

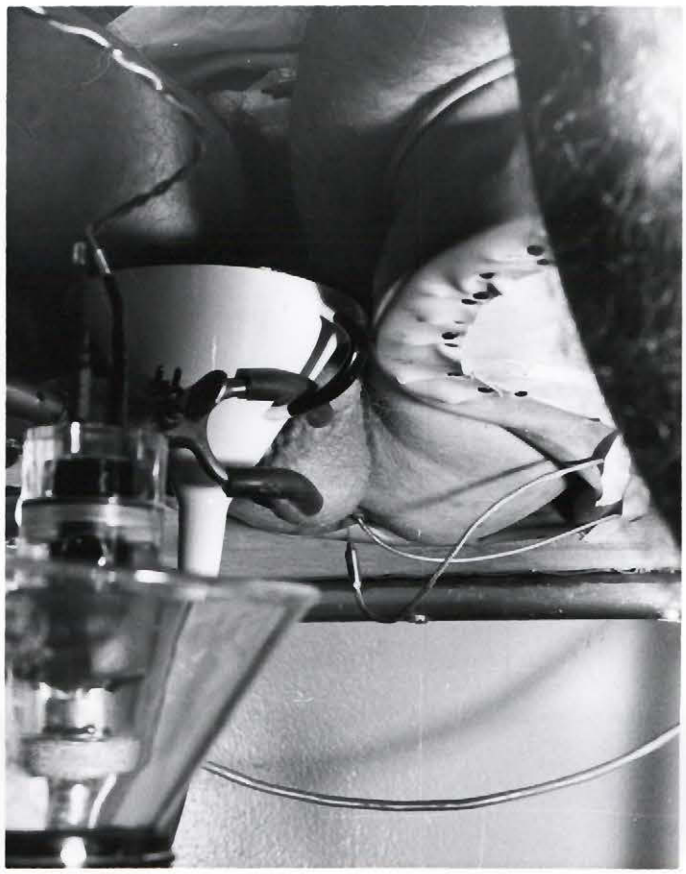


Figure 9 - Patient seated with electrode and rectal balloon in place. Funnel and flow-meter is seen in the foreground.

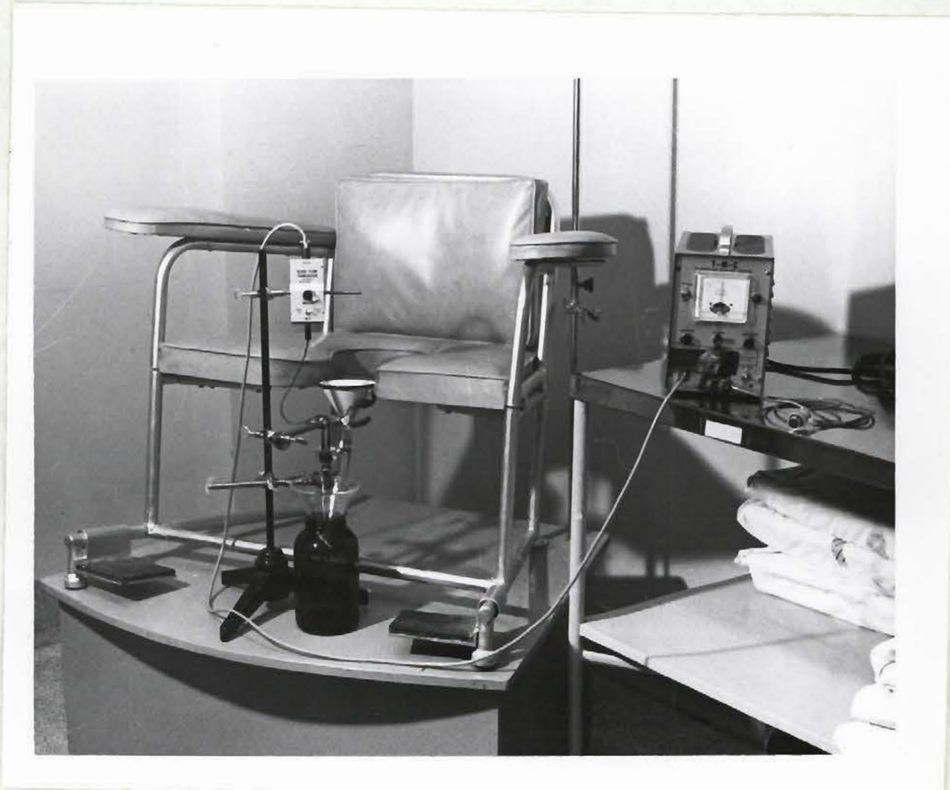


Figure 10 - Urine flow-meter and chair on platform.

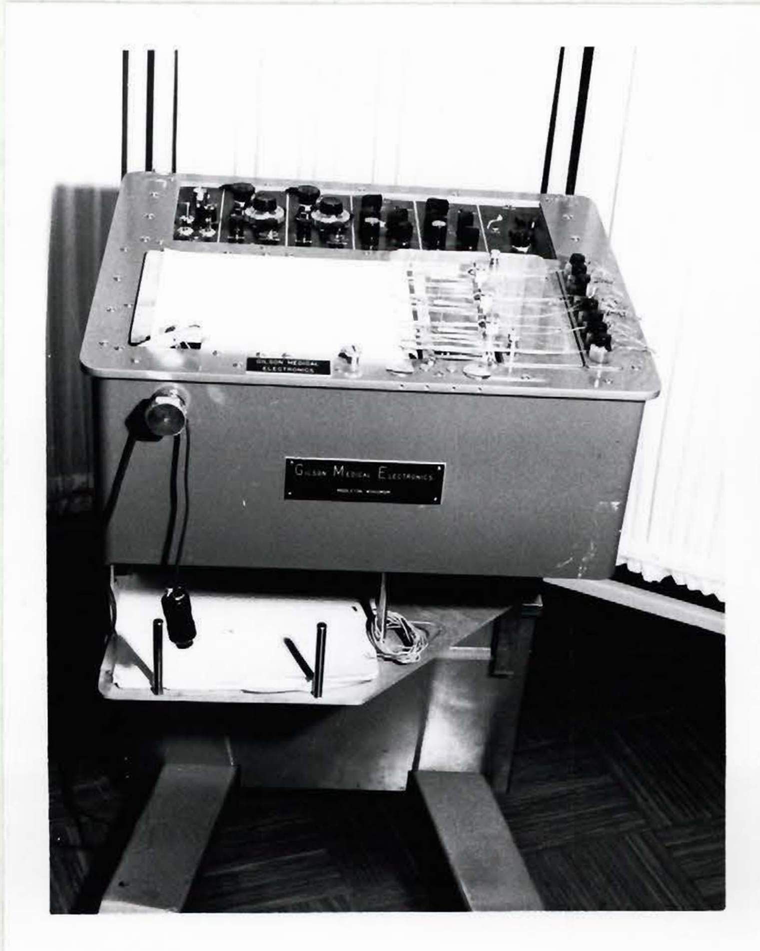


Figure 11 - Gilson polygraph recorder.

A screen is placed around the subject isolating him from the apparatus and examiner (Figure 12) and the recording is begun. (Figure 13) The polygraph recorder is run at a speed of 0.5 mm/sec.

The bladder and rectal pressure recording may be checked by instructing the patient to cough. The resulting spike in bladder and rectal pressures indicate proper function of the recording device.

Figure 12 - Isolation of the subject by a screen placed around him.

Figure 13 - Patient being tested.

The placement of the electrode is checked by stroking or pinching the glans of the penis or clitoris (Bulbo-cavernosus reflex) or by asking the patient to voluntarily contract his sphincter as if he had to "hold back urine". In both instances, proper placement of the electrode is indicated by the characteristic electromyographic pattern of striated muscle potentials.

When the patient expresses the desire to void, the polygraph speed is accelerated to 2.5 mm/sec. and he is allowed to proceed.

At the completion of voiding, the volume of urine passed is measured and the amount of residual urine is determined by aspiration of the bladder contents via the suprapubic tube.

All tubes and needles are removed and a small dressing applied to the suprapubic puncture wound.

If there was evidence of moderate to severe outlet obstruction or of bladder hypotonia, then an indwelling Foley catheter is left in place for 2-3 days to prevent extraperitoneal urinary extravasation.

In the presence of pyuria or bacteruria the patient is placed on sulfas for 1 week following the study.

## RESULTS

Twenty-nine studies were performed on twenty-three patients. One patient (L.B.) was studied twice and 2 subjects (J.P. and K.K. - both normal) were studied on one occasion each but with multiple voidings at different bladder volumes. Of the 23 patients studied, 5 were normal, 12 had clinical evidence of obstructive uropathy and 6 of neurogenic bladder dysfunction.

Because of technical difficulties the flow-meter was not available at first and only 20 of the 29 studies are complete as outlined in the technique. In the 9 earlier studies the maximum flow rate and therefore the pressure at maximum flow could not be determined. Therefore, the minimum resistance in these cases could not be calculated. Using average pressure and average flow however, the average resistance only was determined in these cases.

The results will be presented under the headings of the 5 parameters studied. (Figure 14), illustrates the order of recording of the parameters on the tracing.

### Intravesical Pressure

The Resting Pressure may be defined as the pressure within the bladder during filling. It therefore extends from the time the bladder is empty to just prior to detrusor contraction. The resting pressure in the normal individual when the bladder was empty was found to be 5-10 mm. Hg. As the bladder filled, the resting pressure gradually increased

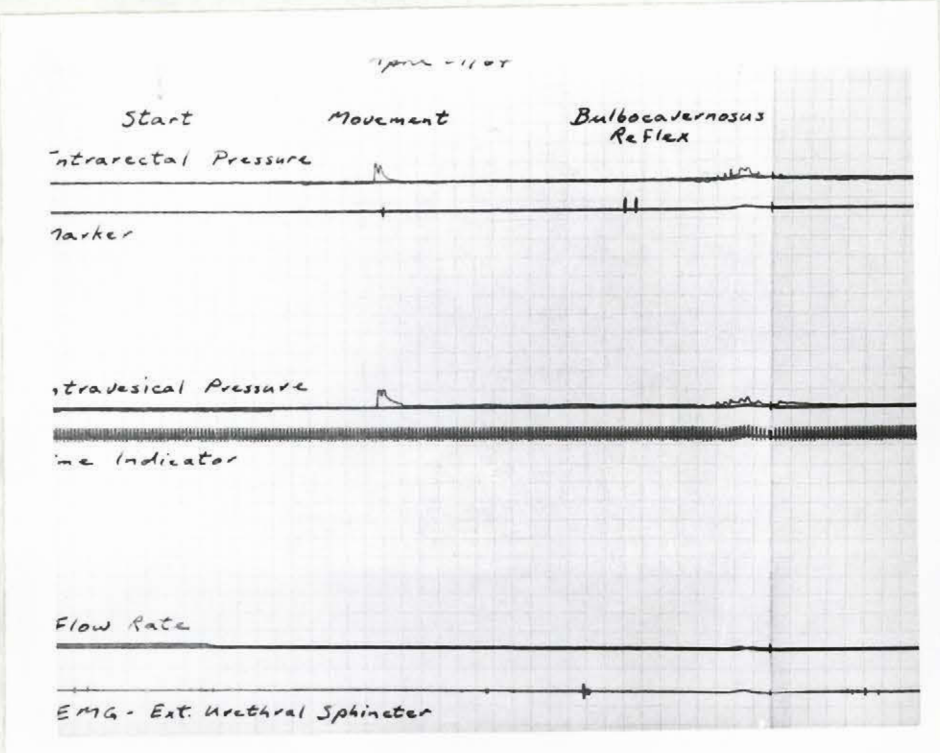



Figure 14- Location of parameters on the tracing.

until it was about 5 mm. Hg. higher just prior to detrusor contraction. Thus, the terminal portion of the resting pressure or the Pre-micturition Pressure was 10-15 mm. Hg. in the normal individual. The resting pressure increase was gradual and did not show evidence of involuntary bladder contractions. The resting pressure curve in the obstructive patients was found to be essentially the same as for the normal individuals. In the cases of neurogenic bladder dysfunction the outstanding difference in the resting pressure curve was the presence of multiple ineffectual low amplitude deflections representing involuntary bladder contractions.

The Initial Voiding Pressure is that pressure at at which urinary flow appears. The intravesical pressure continued to rise until the Maximum Voiding Pressure is reached. The range of maximum voiding pressure in the normal individuals tested was 25-35 mm. Hg. The values in the obstructive group were unequivocally higher - 42-60 mm. Hg.

The intravesical pressure then drops from a maximum value, simultaneously, as the flow rate increases. The Pressure at Maximum Flow is therefore less than the maximum pressure. It was found to be 20-32 mm. Hg. in normals and 36-46 mm. Hg. in the obstructive group.

The intravesical pressure continues to drop at a variable rate until it eventually returns to a resting level which corresponds to the resting pressure as found early in the study when the bladder is empty. The cessation of urine flow invariably precedes the return of the intravesical pressure to pre-micturition levels. In one case, Figure 15, the intracystic pressure remained elevated for 50 seconds following the cessation of voiding.

In none of the cases studied was an "after contraction" as described by von Garrelts noted. Several had minimal deflections in the intravesical pressure curve following voiding, but these were accompanied by corresponding deflections in the intrarectal pressure curve signifying straining in an attempt to completely empty the bladder. This is illustrated in Figure 16.

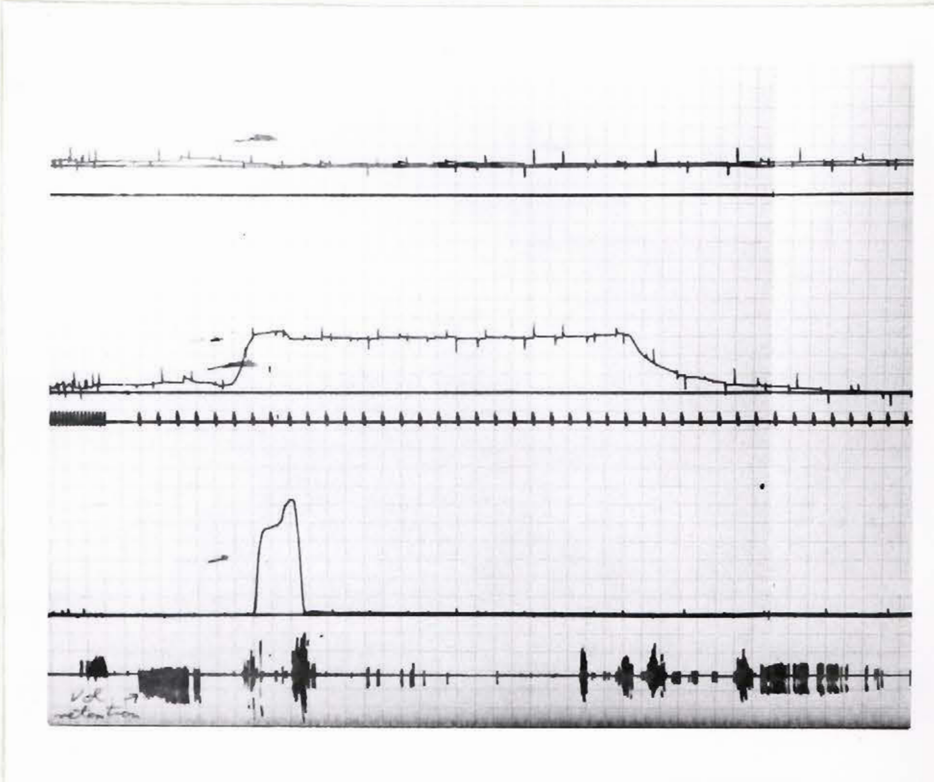


Figure 15 - Subject A.M. - Normal Pressure - Flow - EMG Study. Bladder and rectal pressure curves show deflections due to artifact (electrical interference from outside source).

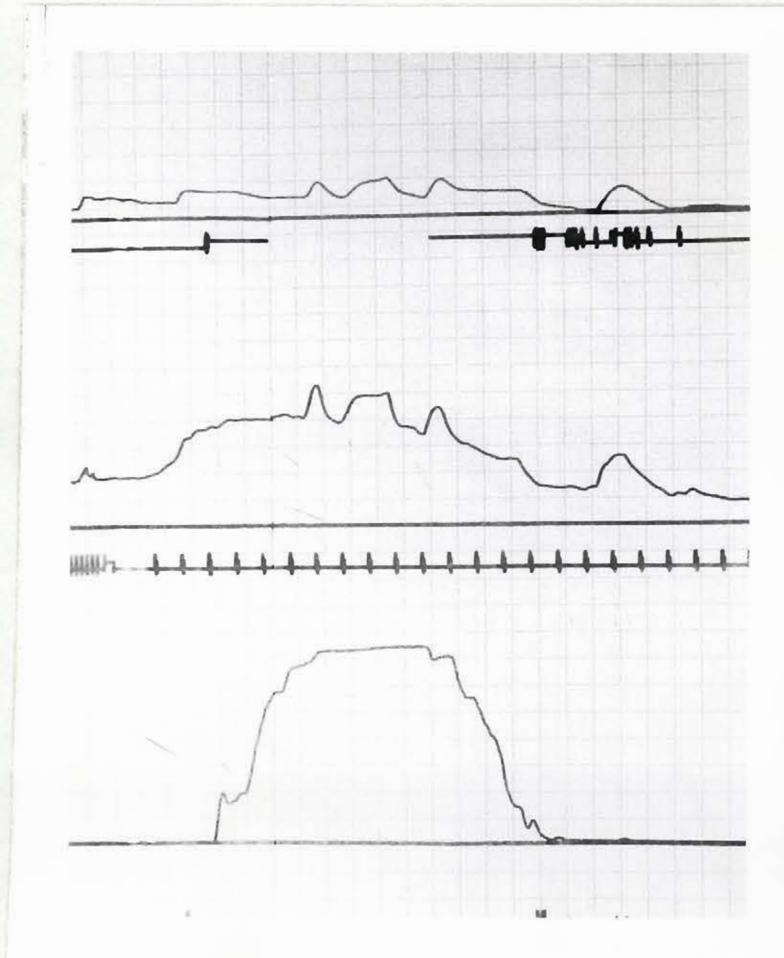


Figure 16 - Study on patient (G.N.) with obstructive uropathy showing effect of straining on intravesical pressure.

In two cases (a normal male and a normale female) voiding parameters at different volumes were studied. In the case of Mr. J.P., for volumes of 80, 130, 250, 400 and 575 cc., the maximum voiding pressure was 35, 40, 32, 32 and 32 mm. Hg. respectively. Mrs. K.K. voided 140 and 210 cc. with maximum pressures of 22 and 25 mm. Hg. respectively.

Intrarectal Pressure

The pen recording rectal pressure was set at the zero level at the beginning of the study. Thus, only relative pressure changes in the rectum were recorded. This is in contrast to the bladder pressure study where absolute pressure was recorded. The changes in intrarectal pressure were not measured but their presence and degree were noted.

In the normal individuals there was little or no change in intrarectal pressure on voiding. On the other hand, patients with obstructive uropathy and neurogenic bladder dysfunction usually required the added pressure provided by straining, Figure 17. The intrarectal pressure changes in these cases were variable.

Urine Flow

The tracing obtained for urine flow is variable depending on the rate of flow, the flow duration and the continuity of the stream. The usual picture obtained in the normal individual was that of a rapid rise in the rate of flow commencing 3-4 seconds after the intravesical pressure had begun to rise. The flow reached a maximum

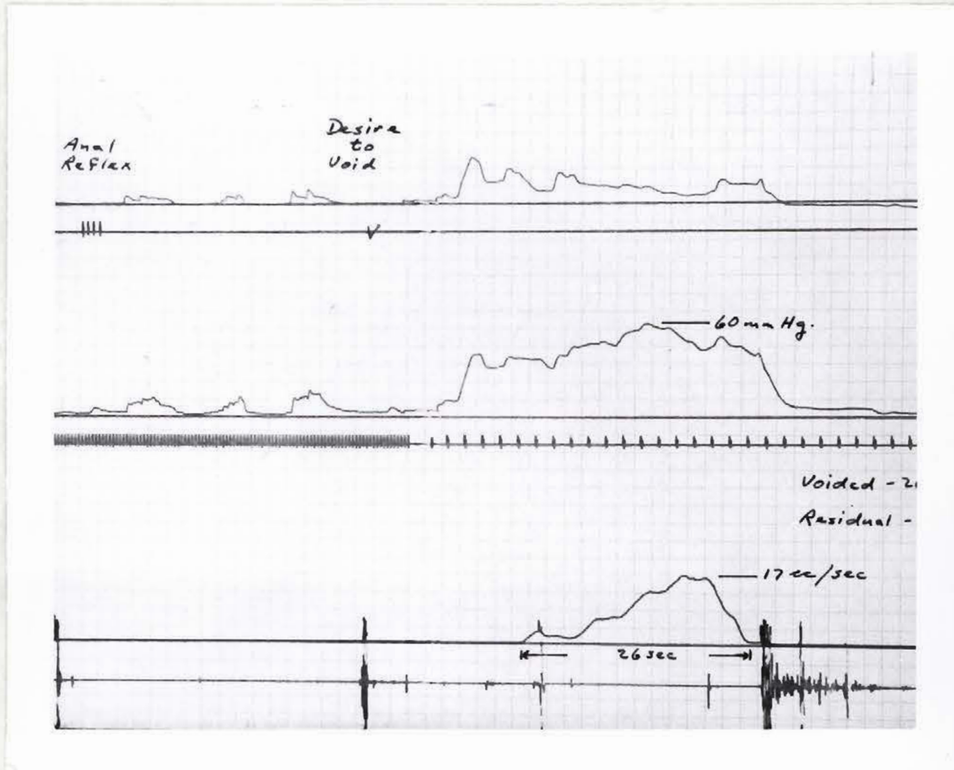



Figure 17 - Patient (B.G.) with bladder neck contracture straining to void.

and then decreased at a variable rate, Figure 18. A similar tracing was seen in the obstructive cases, Figure 19.

An interesting observation was made with respect to the time interval between the time of initial flow and the time of maximum flow. This was found to be significantly longer in the obstructive group (10-18 sec.) than in the normal group (2-8 sec.).

The range of maximum flow in the normals tested was 14-27 cc/sec. in females and 20-32 cc/sec. in males.

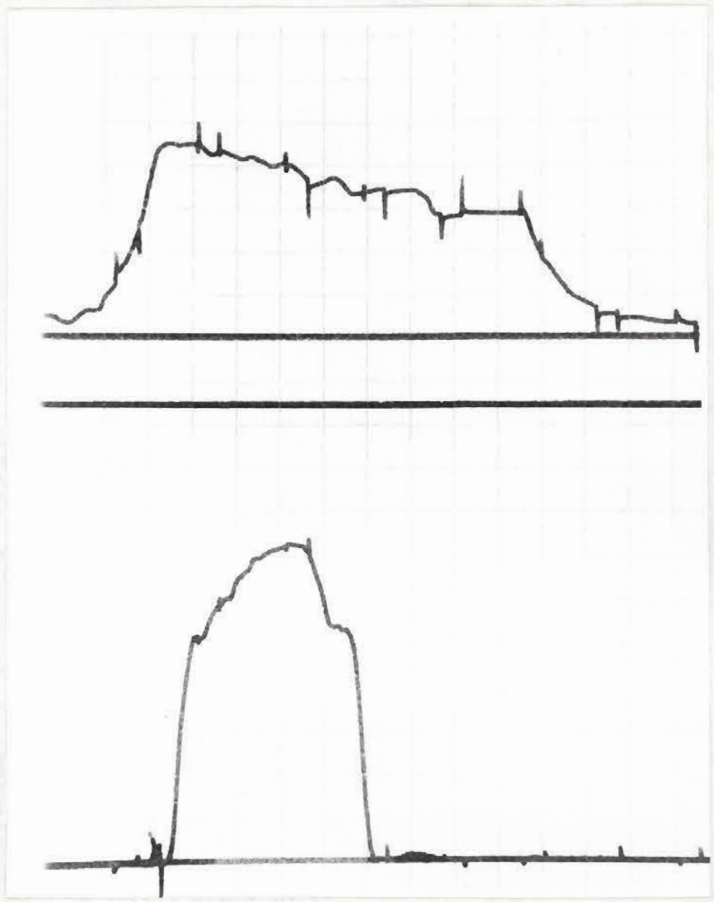


Figure 18 - Tracing of a normal individual
(M.H.) illustrating flow curve

In the obstructive cases it was 11-29 cc/sec. The tracing of urine flow in cases of neurogenic baldder dysfunction often took the form of multiple deflections of low amplitude and short duration. This represented an interrupted weak stream, Figure 20.

The maximum flow rate increased as the volume of urine voided increased. Mr. J.P. voided 80, 130, 250, 400 and 575 cc. with maximum flow rates of 16.5, 18, 20,

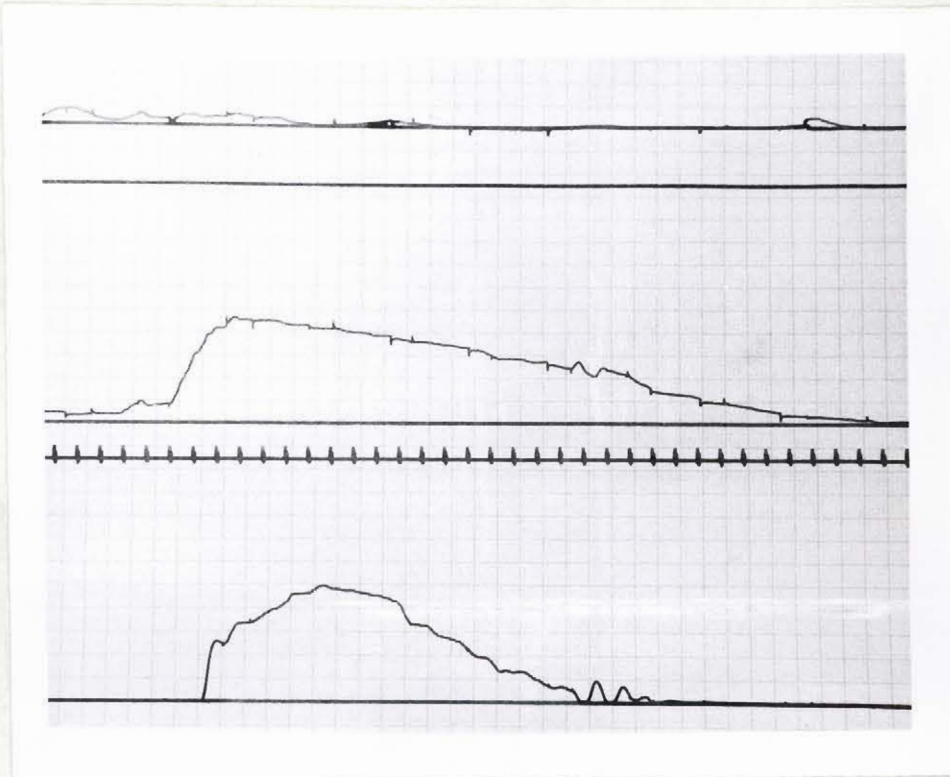


Figure 19 - Tracing of a patient (G.E.) with obstructive uropathy illustrating flow curve.

32 and 32 cc/sec. respectively.

EMG of External Urethral Sphincter

The electromyographic pattern in the normal individual was one of ample activity during placement of the electrode and for some time thereafter. This eventually subsided and the major period of bladder filling was characterized by inactivity of the sphincter with occasional bursts of potentials. In the latter portion of filling and especially after the patient expressed the

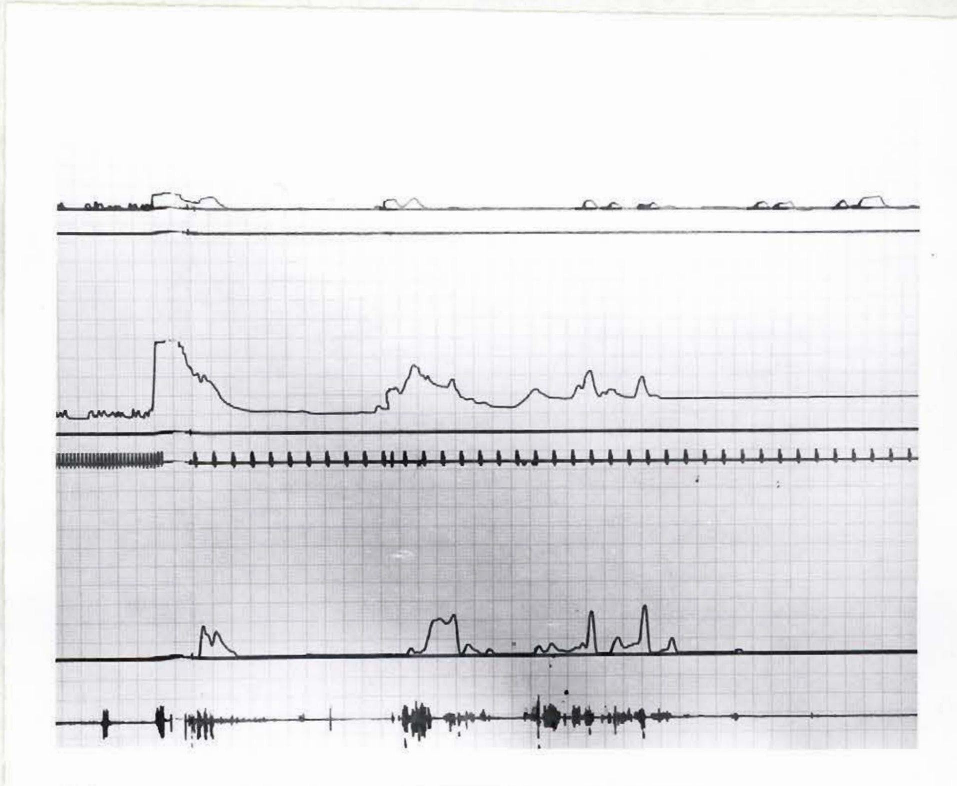


Figure 20 - Tracing of a patient (F.B.) with multiple sclerosis illustrating flow curve.

desire to void the bursts of motor activity became more prominent. Just prior to voiding sphincteric activity was pronounced. With the onset of voiding however, complete relaxation of the external urethral sphincter occurred, as evidenced by the absence of potentials on the oscilloscope screen and by silence from the speaker. Silence persisted until voiding was complete following which evidence of sphincteric contraction returned. The frequency of electrical potentials gradually decreased and disappeared except for occasional bursts during refilling, Figure 21.

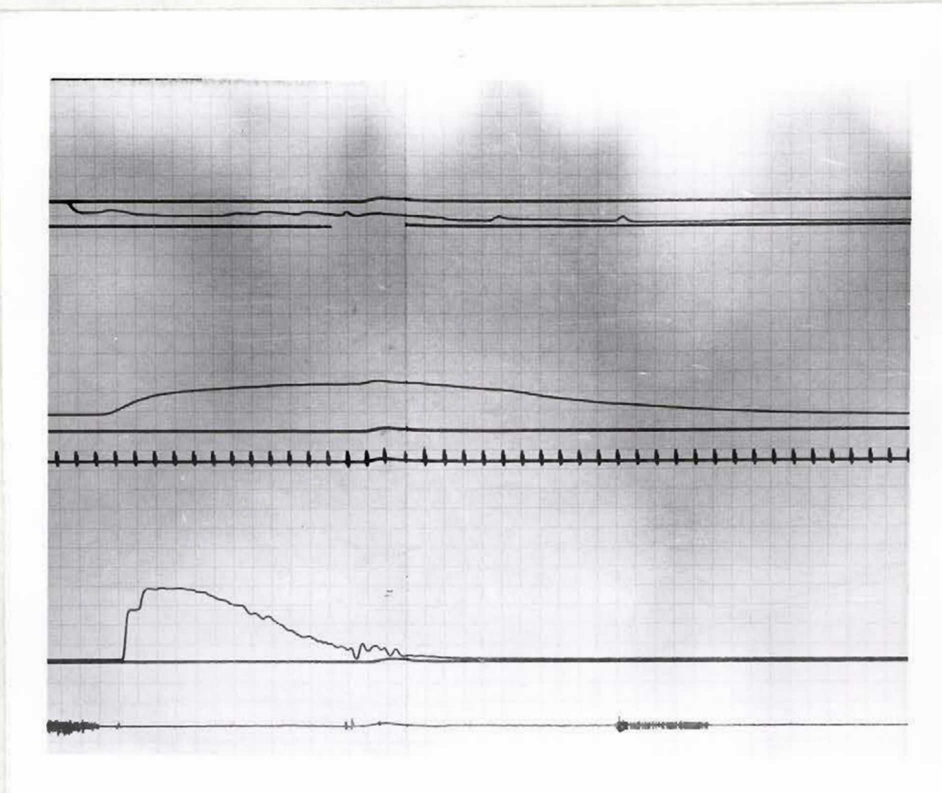


Figure 21 - Normal subject (K.K.) illustrating disappearance of EMG activity during voiding. The explanation for the delay in the return of activity following voiding is not clear.

Voluntary retention of urine when the desire to void is strong results in a most intense degree of sphincteric contraction. This was seen in Figure 22, in which the patient was instructed to "hold it" following a strong desire to void. The intravesical pressure was seen to rise to high levels during isometric bladder contraction and then to drop as the degree of bladder contraction decreased. This change in intravesical pressure correlated well with

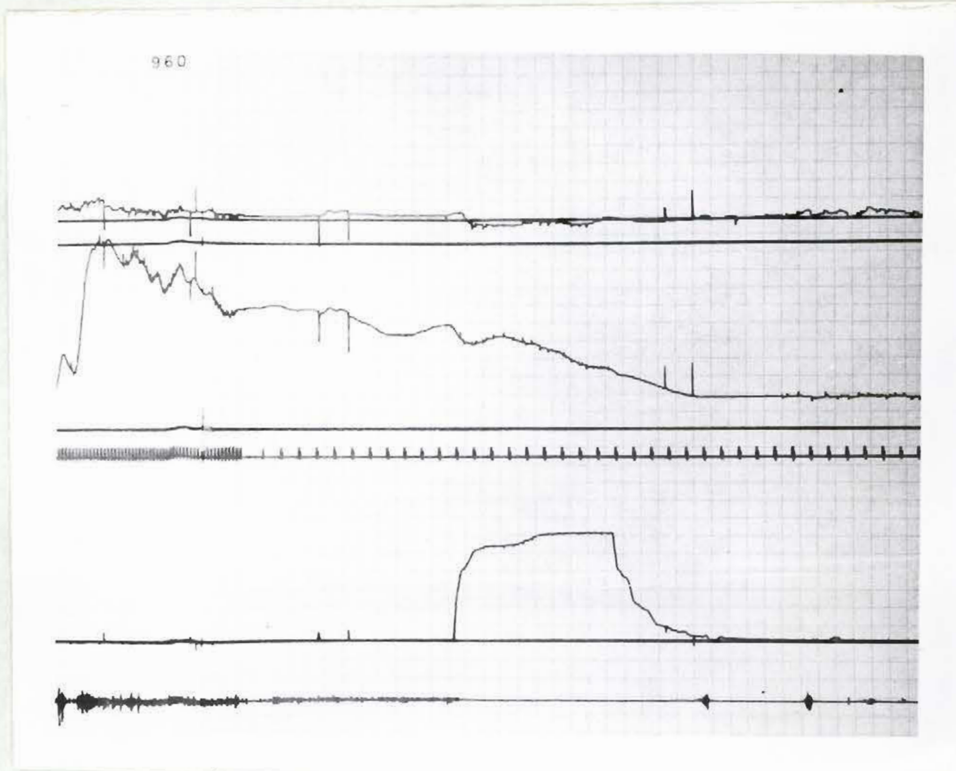



Figure 22 - Patient (E.G.) asked to voluntarily refrain from voiding following a strong sensation of bladder fullness. Artifacts on pressure curves due to electrical interference.

the degree of sense of urgency and with the activity of the EMG. The higher the intravesical pressure the greater the urgency and the greater the EMG activity. As the bladder relaxed and the intravesical pressure fell the EMG activity decreased. With voiding, EMG activity disappears.

Activity of the EMG was evidenced during coughing. It was noted that there was more activity during a cough when the bladder was full than there was earlier in the study when the bladder was not quite as distended.

As well, activity could be elicited by stroking or pinching the glans of the penis (Bulbo-cavernosus reflex) or by anal dilatation (Anal reflex).

In two patients who had had transurethral resection of the bladder neck, a tonic activity of the sphincter was noted throughout bladder filling. One normal female exhibited this pattern. The activity disappeared with voiding.

Patients with neurogenic bladder dysfunction of the Automatic (Upper Motor Neuron) or Uninhibited type showed marked activity of the sphincter during voiding. In many cases this activity was confined to the period of voiding, but in some it lasted for a time thereafter.

Whereas suprapubic pressure (Crede maneuver) had no effect on external sphincter activity in the normal, it caused intense activity in the patient with neurogenic bladder dysfunction, Figure 23.

Anal dilatation, as well, resulted in intense activity of the external sphincter in the patient with neurogenic bladder dysfunction. The intense activity eventually subsided however and urethral resistance was reduced sufficiently to enable the patient to void in spite of renewed activity during voiding, Figure 24.

Urethral Resistance

As discussed earlier, the formula for the calculation of resistance to flow of fluid through a conduit, is

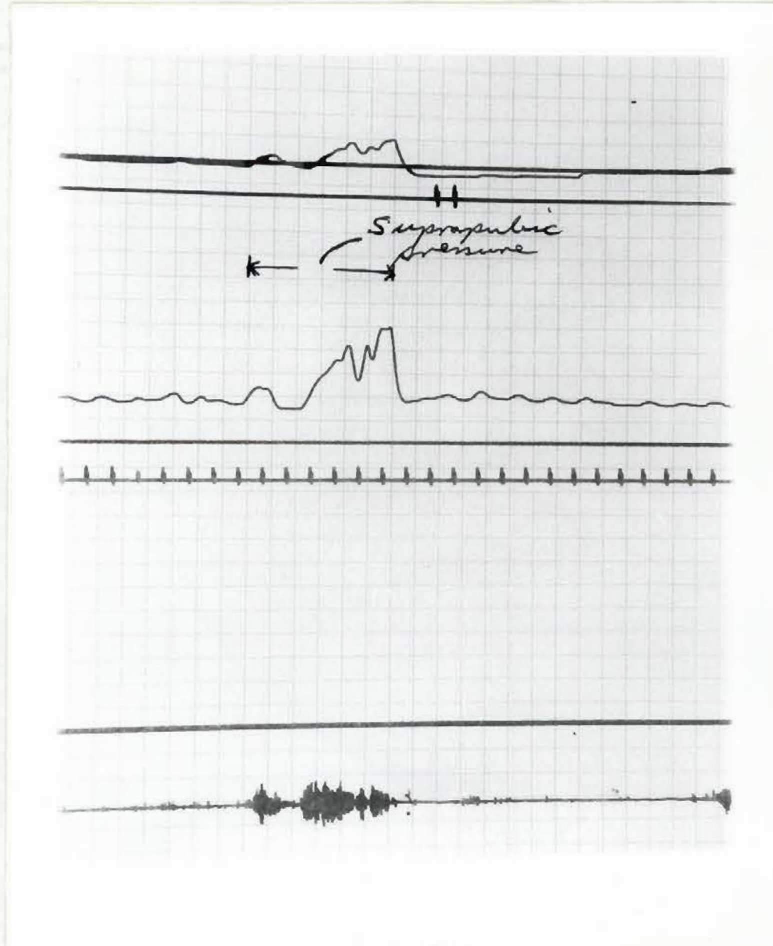



Figure 23 - Effect on activity of external urethral sphincter of suprapubic pressure in a patient (M.C.) with neurogenic bladder dysfunction.

the pressure at one end of the conduit driving the fluid, divided by the rate of flow of fluid out the other end.

The pressure measured in mm. Hg. is converted to dynes/cm.$^2$ by multiplying by 1333 ( 1 mm. Hg. = 1333 dynes/cm.$^2$)

$$\text{Thus:-}\quad R = \frac{P\ (\text{dynes/cm.}^2)}{F\ (\text{cm.}^3/\text{sec.})}$$

$$= \frac{\text{dyne.sec}}{\text{cm.}^5}$$

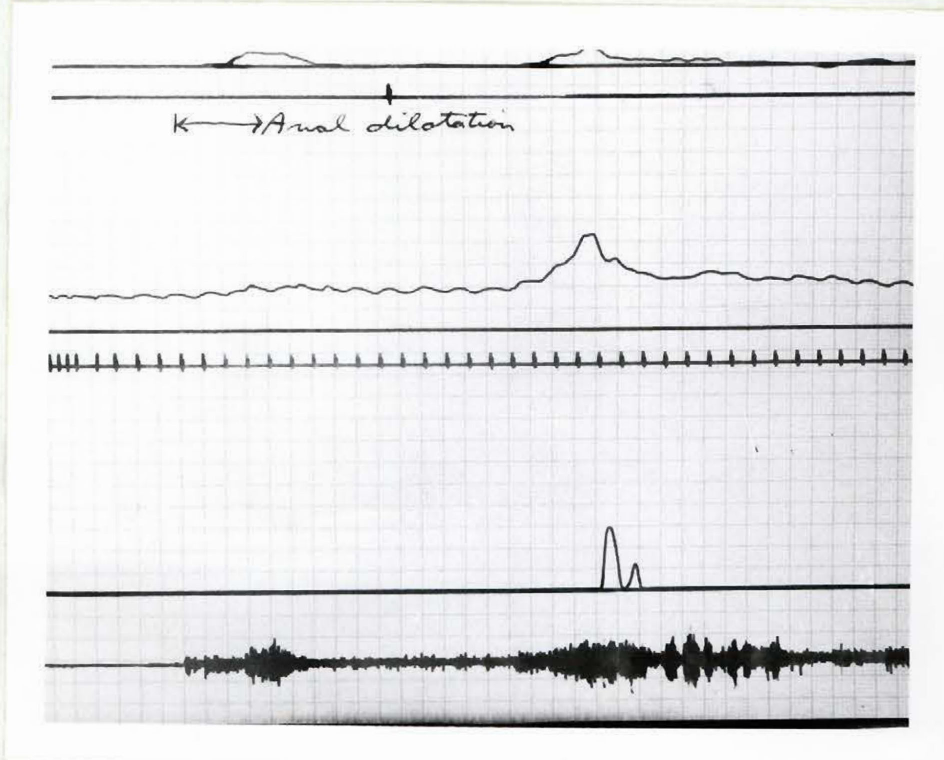



Figure 24 - Effect on EMG activity of anal dilatation in patient (M.C.) with neurogenic bladder dysfunction.

The figure is made less cumbersome by multiplying by $10^{-3}$.

It is obvious from the formula that the resistance to flow is increased if the intravesical pressure is increased and/or the flow of urine is decreased. In the normal individual the urethral resistance is low because of good urine flow with low intravesical pressure, Figure 25.

The average urethral resistance was calculated by dividing average intravesical pressure during voiding by the average rate of urine flow, Table I. The amount and duration

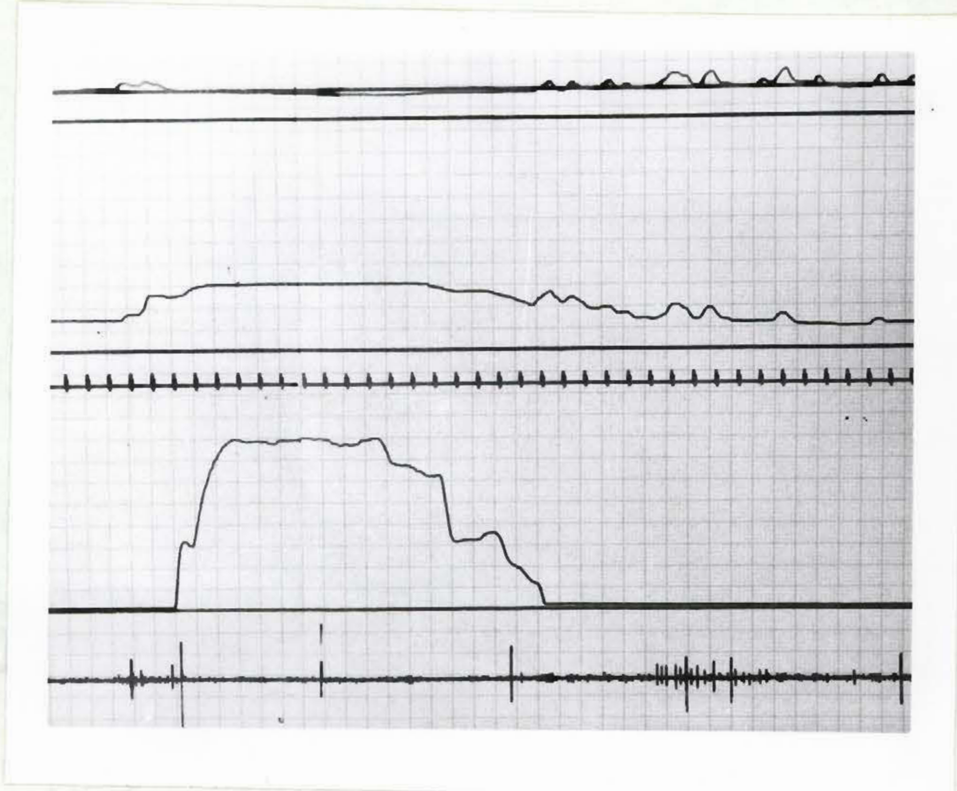


Figure 25 - Tracing in normal subject (J.P.). Note low voiding pressure with good urine flow.

of flow must be known to calculate average flow. The average pressure is calculated by determining the average height of the area under the pressure curve during voiding. The calculation of average resistance therefore does not require the use of a flow-meter.

The range in the 5 normal subjects tested was 2.49 - 3.53 R units. ( 1 R unit = 1 dyne.sec.$cm^{-5}$ x $10^{-3}$) All patients with proven urethral obstruction of moderate to severe degree had values above this except E.G., age 13.

The range of normal values in children is undoubtedly lower than that in adults. Two patients with mild benign prostatic hypertrophy had values in the normal range. Neither required treatment. All patients with neurogenic bladder dysfunction, but one (G.C.), had average resistance values above normal. The one exception was a patient whose bladder dysfunction had been treated by transurethral resection of the prostate and bladder neck with partial division of the external urethral sphincter. His urethral resistance was intentionally reduced to the point where he exhibited stress incontinence. By reducing his urethral resistance to low levels he was able to empty his bladder on voiding.

Average urethral resistance could not be calculated in one patient (F.B.) due to the fact that he voided multiple (6) small amounts.

The minimum resistance was calculated by dividing the pressure at maximum flow by the maximum flow, Table II. A urine flow-meter is necessary for the determination of peak flow and therefore, as well, of the the pressure at peak flow.

The minimum resistance was in all cases lower than the respective average resistance. The normal range of minimal resistance was 1.33 - 2.00 R units. It is interesting that a male subject had a lower minimum resistance that either of the two normal females tested. The same was true of average resistance values.

All values for the obstructive group, except one, fell above the normal range. The exception (G.N.) was a patient who had a transurethral resection of the prostate one year prior to examination. He was admitted because of diurnal frequency and nocturia and was found to have pseudomonas infection of the urine. No obstructive element was found.

A marked drop in minimum resistance is noted post-operatively in patient L.B. who had a transurethral resection of the bladder neck for contracture.

Both cases of neurogenic bladder dysfunction had abnormally high values for minimum resistance.

Table III illustrates a definite inverse relationship between volume of urine voided and average and minimum resistance. That is, the greater the volume of urine voided the less the urethral resistance and vice versa. This relationship is less obvious for average resistance that it is for minimum resistance. The inverse relationship is only noted when the volumes of urine voided are significantly different. The figures of Table III are placed in graphic form in Graph I.

The urethral resistance during a single act of voiding along with changes in intravesical pressure and rate of urine flow was plotted against time in one patient (G.E.), Graph II. It is noted that the urethral resistance decreases as the rate of flow increases and that the minimum urethral resistance corresponds to the peak flow. This is in agreement with the findings of Arbuckle and Paquin.[19.] The resistance is then seen to increase as the rate of flow diminishes. The time of maximum intravesical pressure precedes that of maximum flow and minimum resistance by 4-8 seconds.

TABLE I

$$\text{Average R}\left(\frac{\text{dyne.sec}}{\text{cm.}^5}\right) = \frac{\text{Average P}\left(\frac{\text{dynes}}{\text{cm.}^2}\right)}{\text{Average F}\left(\frac{\text{cm.}^3}{\text{sec.}}\right)}$$

| Patient | Age | Diagnosis | Average R(x $10^3$) | Voided (cc) | Residual(cc) |
|---|---|---|---|---|---|
| | | NORMAL | | | |
| V.C. | 49 | Normal | 3.53 | 350 | 0 |
| A.M. | 31 | Normal | 3.33 | 82 | 0 |
| M.H. (F) | 44 | Normal | 2.96 | 160 | 0 |
| J.P. | 32 | Normal | 2.67 | 575 | 0 |
| K.K. (F) | 38 | Normal | 3.07 | 210 | 4 |
| | | OBSTRUCTIVE | | | |
| G.E. | 76 | Ca of prostate | 5.07 | 350 | 5 |
| P.P. | 71 | Benign Prostatic Hypert. | 10.37 | 100 | 85 |
| B.G. | 28 | Bl. neck contr. | 6.45 | 260 | 0 |
| W.C. | 21 | Urethral stric. (Post-dilat.) | 3.67 | 400 | 35 |
| A.G. | 66 | B.P.H. | 7.27 | 550 | 0 |
| L.B. | 23 | Bl. neck con. | 46.65 | 170 | 550 |
| L.B. | 23 | Post - TUR | 4.67 | 200 | 590 |
| G.N. | 73 | B.P.H.-minimal | 3.33 | 410 | 35 |
| E.G. | 13 | Bl. neck contr. | 3.14 | 475 | 240 |
| M.B. | 20 | Bl. neck contr. | 8.82 | 150 | 0 |
| A.D. | 57 | B.P.H.-minimal | 3.51 | 270 | 8 |
| R.R. | 48 | Bl. neck con. | 33.33 | 200 | 600 |
| H.L. | 57 | B.P.H. | 4.67 | 80 | 5 |
| | | NEUROGENIC BLADDER DYSFUNCTION | | | |
| J.T. | 22 | Paraplegia | 7.62 | 80 | 60 |
| G.C. | 37 | Paraplegia | 2.96 | 300 | 0 |
| C.C. | 41 | M.S. | 13.33 | 300 | 630 |
| M.C. | 44 | M.S. | 16.00 | 35 | 40 |
| F.B. | 31 | M.S. | - | 100 | 10 |
| A.C. | 59 | M.S. and median bar | 6.27 | 340 | 40 |

## TABLE II

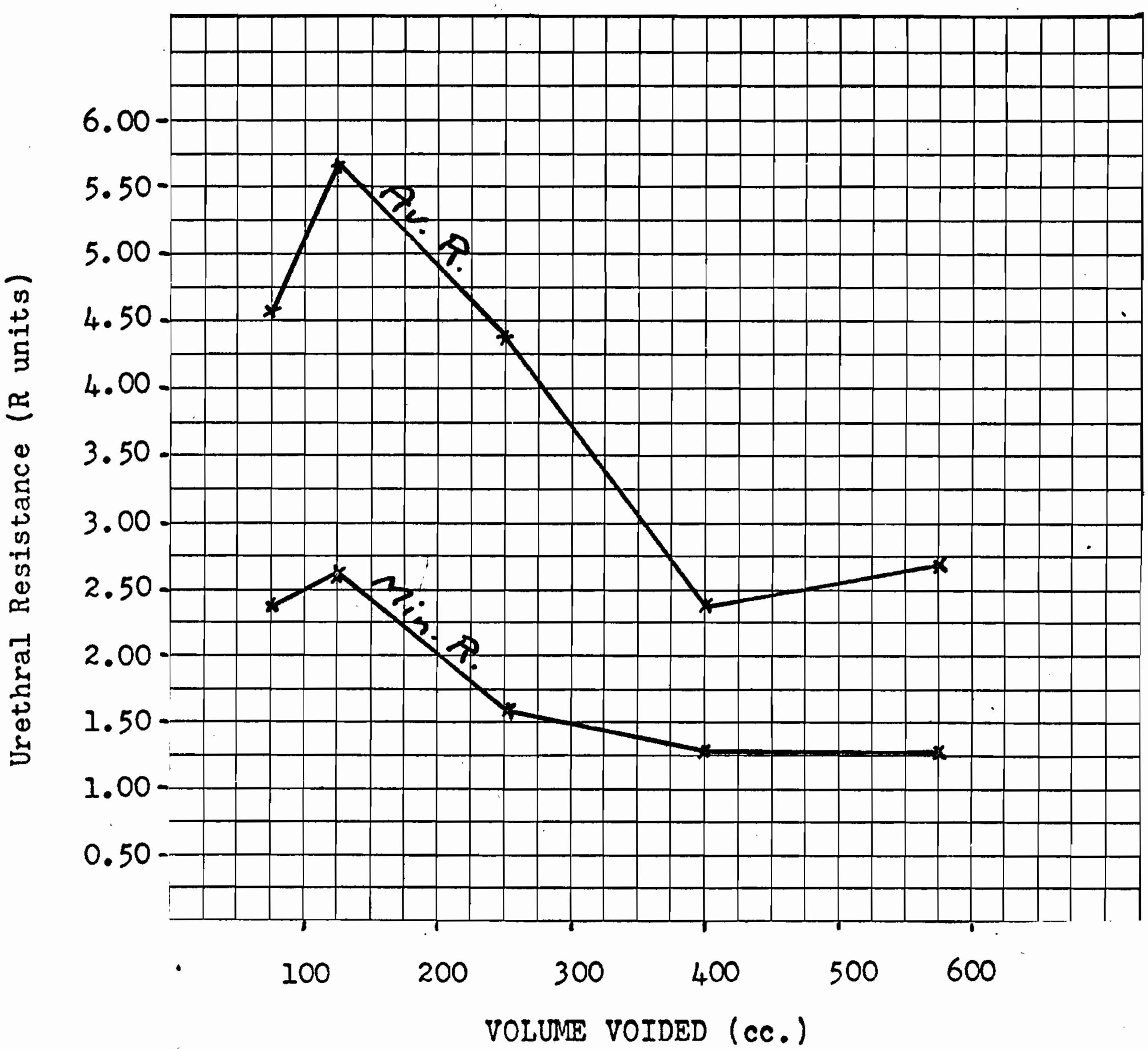
$$\text{Minimum R (dyne.sec/cm.}^5) = \frac{\text{Pressure at maximum F (dynes/cm.}^2)}{\text{maximum F (cm}^3\text{/sec.)}}$$

| Patient | Max. flow(cc/sec) | P at max. flow(mm Hg) | Min. R x $10^3$ |
|---|---|---|---|
| | | **NORMAL** | |
| A.M. | 20 | 30 | 2.00 |
| M.H. (F) | 27 | 40 | 1.97 |
| J.P. | 32 | 32 | 1.33 |
| K.K. (F) | 14 | 20 | 1.90 |
| | | **OBSTRUCTIVE** | |
| G.E. | 16.5 | 44 | 3.55 |
| P.P. | 11 | 38 | 4.60 |
| B.G. | 17 | 46 | 3.61 |
| L.B. | 6.5 | 110 | 22.55 |
| L.B.(Post TUR) | 14 | 36 | 3.43 |
| G.N. | 29 | 40 | 1.84 |
| E.G. | 19 | 40 | 2.81 |
| H.L. | 12.5 | 35 | 3.73 |
| | | **NEUROGENIC BLADDER DYSFUNCTION** | |
| C.C. | 11 | 50 | 6.06 |
| A.C. | 25 | 45 | 2.40 |

## TABLE III

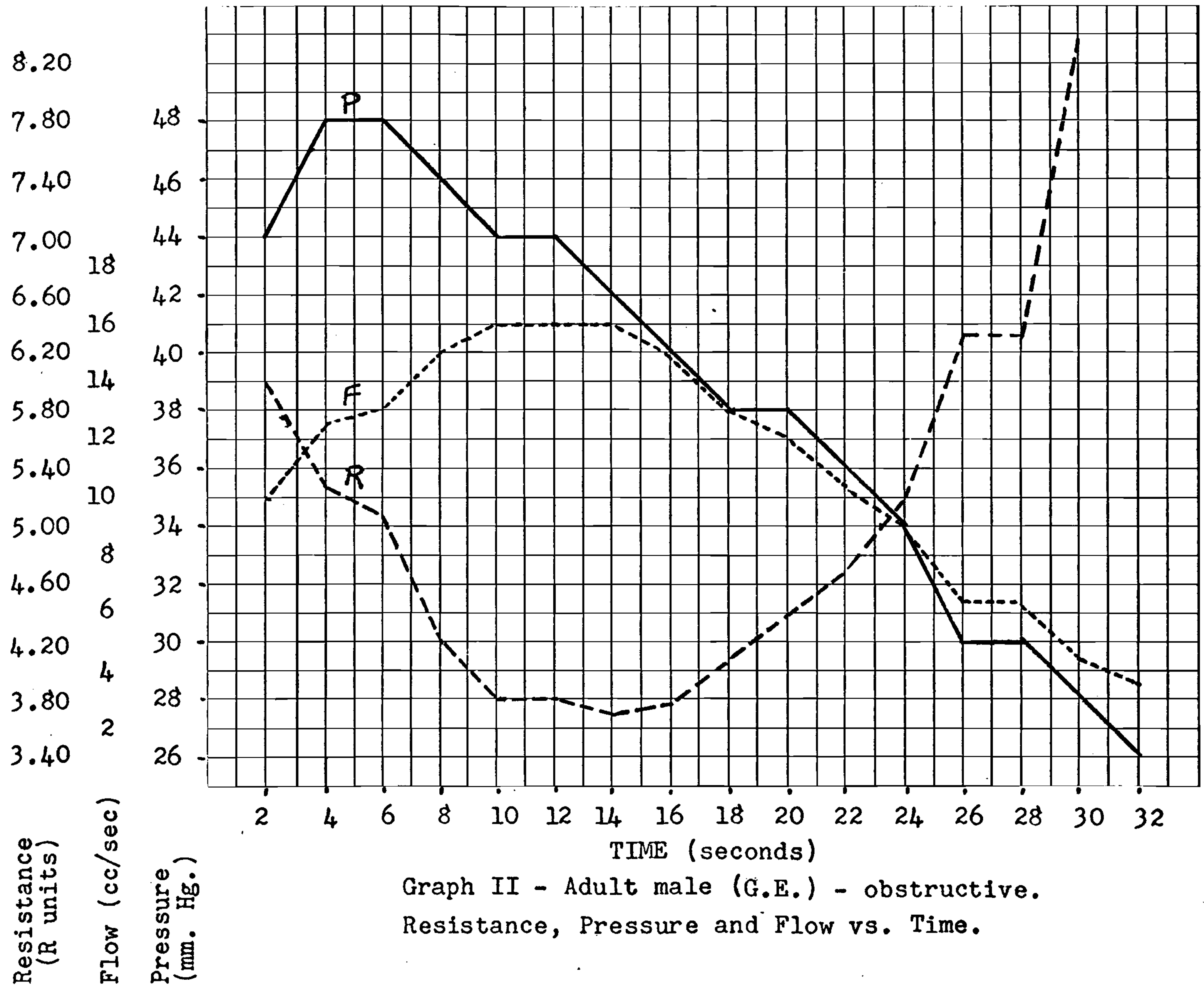
Average and Minimum Resistance in a normal adult male (J.P:) voiding different volumes.

| Voided(cc) | Residual(cc) | Average Resistance(R units) | Min. Resist. (R units) |
|---|---|---|---|
| 80 | 0 | 4.67 | 2.42 |
| 130 | 12 | 5.71 | 2.59 |
| 250 | 0 | 4.44 | 1.67 |
| 400 | 2 | 2.36 | 1.33 |
| 575 | 0 | 2.67 | 1.33 |




Graph I - Normal adult male (J.P.)

Urethral Resistance vs. Volume Voided.




Graph II - Adult male (G.E.) - obstructive.
Resistance, Pressure and Flow vs. Time.

## DISCUSSION

The discussion of the results obtained will be presented, as were the results themselves, under the headings of the parameters studied.

### Intravesical Pressure

The usual means of measuring intravesical pressure during bladder filling is by cystometry using the Lewis cystometer. Fluid is run into the bladder from an overhead reservoir via a urethral catheter. The rate of flow of the fluid is maintained at approximately 100 cc. every 5 minutes, and the intravesical pressure during filling is recorded in mm. Hg. The resultant tracing, produced on a 4 x 9 inch card, is called a cystometrogram. The subject is asked to notify the examiner of the first sensation of a desire to void and of subsequent discomfort due to bladder fullness. He is also asked at various intervals to strain as if to void.

The cystometrogram reveals approximate values for bladder capacity and for the degree of bladder fullness at which the patient has the first desire to void. Inaccuracy in these values is due to the presence of a catheter in the urethra, to the accelerated rate of bladder filling and to the temperature of the fluid used, which is lower than that found physiologically. A desire to void is often reported following the introduction of the urethral catheter at the beginning of the test. Filling the bladder quickly or with cold fluid may give rise to early discomfort and therefore a false impression of reduced bladder capacity.

When the patient is asked to bear down a spike in intravesical pressure is recorded. This spike is usually due entirely to increase in intra-abdominal pressure. A detrusor contraction is seldom seen, except in those cases of spastic neurogenic bladder dysfunction where straining initiates a reflex bladder contraction.

The presence or absecne of involuntary bladder contractions is revealed by cystometry, but here too inaccuracy is caused by the rate of flow and the temperature of the filling fluid. In the case of spastic neurogenic bladder dysfunction, the colder the fluid and the faster the rate of flow, the earlier and the more forceful will be the involuntary bladder contractions.

As the same channel is used both to fill the bladder and to record pressure, inaccuracy in the latter is bound to occur due to turbulence of flow. Furthermore, a serious deficiency in the test stems from the fact that an isotonic bladder contraction, as occurs physiologically, cannot take place as the urethra is obstructed by the catheter. Isometric bladder contractions are often seen in cases of spasticity with leakage of fluid around the catheter.

Weyrauch[21.] has abandoned this test as a diagnostic procedure. He feels that the technique fails because of:

"1) The extremely wide variation in the normal as well as the abnormal cystometrogram.

2) the broad overlap of the pathological and the normal and,

3) the fact that numerous abnormal conditions of the bladder produce identical changes in the cystometric curve."

Conventional cystometry continues to be widely used as a crude test of bladder sensation, capacity and tone. Its limitations and inaccuracies however are recognized. The intravesical pressure curve as obtained in our study of bladder dynamics provides a much more accurate representation of the true or physiologic pressures than that obtained by the Lewis cystometer. The two studies however are not competitive as their aims are different.

The resting intravesical pressure is felt to be due to the sum of the pressure due to the weight of the intra-abdominal viscera resting on the bladder plus the pressure due to bladder tone. It seems logical to state that the intravesical pressure when the bladder is empty is due entirely or mainly to the weight of the viscus and that the increase in bladder pressure as the bladder fills is due to bladder tonicity. These statements are supported by two observations. Firstly, a rough correlation of initial bladder pressure with body weight was noted. The lowest pressure recorded when the bladder was empty (5 mm.Hg.)

was that in a 13 year old boy of average weight. Secondly, the intravesical pressure with the bladder emptied was measured in a normal female (Mrs. K.K.) in the standing, lying and sitting positions. As each position was assumed, the diaphragm of the pressure transducer was placed at the level of the symphysis pubis using a carpenter's level to assure accuracy. The intravesical pressure on standing was 25 mm. Hg., lying flat it was 0 mm. Hg. and sitting it was 6 mm. Hg.

On lying down the viscera fell away from the empty bladder and exerted little or no effect on it. The intravesical pressure in this position registered 0 mm. Hg. and therefore it can be said that the bladder was without tone.

As the weight of the viscus is constant, it follows that the increasing pressure as the bladder fills is due entirely to increasing bladder tone. This increasing tone was found to be very gradual and to add little (5 mm.Hg.) to intravesical pressure during the resting or filling phase.

A possible explanation for the difference in intravesical pressure on standing and on sitting may be that on standing the abdominal musculature is contracted to maintain posture, thereby adding to the intra-abdominal and consequently to the intravesical pressure.

The effect of posture on bladder pressure was first studied by Gould[22.] in 1955. He noted higher pressures in the upright position as compared with the supine position and attributed the difference to the weight of the intestines.

Arbuckle and Paquin[19.] found that the standing pressure was slightly higher than the sitting pressure and the sitting pressure was twice as high as the supine pressure. They do not however state whether the bladder was empty or full at the time of the measurement or whether the level of the pressure transducer was altered with changes in position.

It was thought that the pre-micturition pressure in the obstructive cases would be higher than that in normal individuals because of hypertrophy and hypertonicity of the detrusor. This was not found to be the case. The pre-micturition pressures in both groups were essentially the same.

The initial voiding pressure is difficult to determine accurately because of the mechanical delay in recording the exact point of onset of urination. Although the delay may only be of the order of 1-2 seconds, intravesical pressure changes at this point are great and therefore the inaccuracy unacceptable.

The finding of higher maximum voiding pressure in the obstructive group confirms the findings of Murphy and Schoenberg.[11.] It is to be expected that a higher intravesical pressure is required to overcome outlet obstruction.

The greater intravesical pressure is the result of compensatory hypertrophy of the detrusor and probably more important the greater role played by the isometric phase of contraction because of the obstruction. Recall the high intravesical pressure seen with voluntary urethral obstruction, Figure 22.

The relative constancy of the maximum voiding pressure with varying volumes of urine voided in the two cases studied agrees with the findings of von Garrelts.[8] Arbuckle and Paquin,[19] on the other hand, found that the pressure decreased very slightly with increasing volume instead of remaining absolutely stable.

Our findings do not confirm the statement of von Garrelts[8] that bladder pressure is lower after micturition than before. The pressure after micturition was found to be equal to the early resting pressure. Both these values however were lower than the pre-micturition pressure and it may be that this is what von Garrelts is referring to.

The internal sphincter is closed during the phase of bladder filling. Initially the bladder contracts isometrically and the intravesical pressure rises rapidly until the bladder neck opens sufficiently to allow some urine to flow. This point corresponds to initial voiding and it would appear that at this point the bladder neck is minimally opened. The intravesical pressure continues to rise as the bladder neck opening progresses, resulting in increasing rate of urine flow. A peak pressure is reached before peak flow is obtained. At the time of peak flow the pressure may have dropped to some extent. The time of peak flow is felt to correspond to maximal opening of the bladder neck.

It would appear that bladder contraction up to the point of initial flow is entirely isometric. As opening of the bladder neck progresses following initial flow, the isometric component of the bladder contraction predominates as the isotonic component increases until the maximum intravesical pressure is reached. At this point the neck is sufficiently open to allow for good flow of urine, thereby reducing the isometric component. Following the maximum intravesical pressure, the isotonic component of bladder contraction predominates as the isometric component decreases. At maximum flow the internal sphincter is fully open and the isometric component is greatly reduced. Bladder contraction from the point of maximum flow to the point of cessation of flow is therefore mainly isotonic.

It may be that the configuration of the vesical pressure curve may reflect the character of the internal sphincter. Study of these pressure curves revealed some interesting findings. In the normal cases there was not much difference between the maximum voiding pressure and the pressure at maximum flow. That is, there was not much of a drop in pressure after the maximum pressure had been reached. On the other hand, four of the five obstructive cases that had had flow studies, showed a definite drop in pressure between these two points. This pressure change may therefore reflect the degree of resistance to opening of the bladder neck. Thus, it would appear that there is little pressure change and therefore little resistance to opening of the bladder neck in the normal individual. In two cases of bladder neck contracture in which flow studies were obtained, the drop in pressure from maximum pressure to pressure at maximum flow was 15 - (55-40) and 14 - (60-46) mm. Hg. respectively. The pressure drop was generally less in cases of prostatic obstruction.

The "after contraction" was first noted by Denny-Brown and Robertson. They noted that the contraction was not due to changees in intra-abdominal pressure. Its occurrence was later confirmed by von Garrelts,[8.] in 73 out of 147 voidings. von Garrelts found that the rise in intravesical pressure at the end of urination, although appreciable and occasionally of greater magnitude than the maximum voiding pressure, did not seem to influence the flow of urine. Its appearance could not be explained by this author.

Arbuckle and Paquin[19.] demonstrated the after contraction in 24% of the voidings. They postulated that the after contraction is probably the result of the internal sphincter closing tightly before the detrusor contraction has terminated. This seems to be the most likely explanation for thephenomenon, if it exists. Why then was it not demonstrated in our present studies? Could it be that it is the result of a bladder spasm caused by a transurethral catheter which the aforementioned investigators used to measure intravesical pressure?

Although the intravesical pressure as measured in these studies is felt to be accurate, sources of error must be considered. Great care is exercised to prevent the introduction of excessive length of tubing into the bladder. Some stimulation of the bladder mucosa and trigone by the catheter is inevitable and this may lead to inaccuracy in the recording. One of the patients had a voiding cine-cystourethrogram following our study. The suprapubic tube was left in place so that the contrast material could be introduced into the bladder. Fluoroscopic examination revealed that although the intraluminal portion of the tubing was short, its end was in the prostatic urethra. This was unexpected as the patient experienced no discomfort and he voided well with good recording of intravesical pressure during the study that day. Using the present technique this problem cannot be prevented. However, should fluoroscopy be incorporated in the study it could be readily avoided or corrected.

In another case (B.G.), similarly studied, voiding cine-cystourethrogram revealed massive bilateral ureterovesical reflux. The reflux siphoned off some of the vesical pressure so that the voiding pressure was lower than it would otherwise have been. The calculated resistance therefore did not correlate with the severe degree of obstruction observed clinically. Aspiration of the bladder immediately after voiding revealed no residual. The cine-studies revealed complete emptying of the bladder at the completion of voiding, followed by refilling as the contrast material that had refluxed up the ureter returned to the bladder. If the bladder had been aspirated 5 minutes later at least 200 cc. of "residual urine" would have been noted. The true dynamics of micturition in this case were not revealed by the pressure-flow studies alone. These experiences emphasize the importance of the addition of cine-fluoroscopy to the study of lower urinary tract dynamics, both normal and abnormal.

<u>Intrarectal Pressure</u>

Gleason and Lattimer[23.] emphasize the need to measure rectal pressure for the proper interpretation of voiding pressure. Only through a knowledge of the presence or absence of extrinsic forces acting on the bladder, can one determine the intravesical pressure that is due to detrusor contraction alone. By subtracting the rectal pressure from the total intravesical pressure a pressure due to pure detrusor contraction is obtained.

A tracing indicating changes in rectal pressure enables one to distinguish involuntary bladder contractions during filling from deflections in the bladder pressure tracing due to straining.

Calculation of urethral resistance requires measurement of the total intravesical pressure, not just that due to detrusor contraction. Therefore, it was not necessary to measure the changes in intrarectal pressure even though straining may alter the intravesical pressure and therefore the flow. Stewart[14] noted that the flow rate can be increased up to 10cc./sec. by abdominal straining alone. Since both the intravesical pressure and the flow are increased by straining, the calculated urethral resistance should not be appreciably altered.

Urine Flow

If initial flow reflects minimal opening of the bladder neck and prostatic urethra, and maximum flow complete opening, then it would be reasonable to expect that the time interval between the two be longer in obstructive cases than in normal individuals. This indeed, was found.

As noted previously, the exact time of onset of flow could not be obtained accurately with the flow-meter. Arbuckle and Paquin[19] had their subjects press a recording button for the length of time of voiding. This method is grossly inaccurate. It is very difficult to both void normally and concentrate on recording the onset and cessation of voiding. The author tried and failed miserably.

The examiner could observe the subject and record onset and cessation of voiding. This however, introduces the problem of psychic inhibition of voiding. Fortunately, the determination of the exact time of onset of voiding and from this the initial voiding pressure is not essential and can be dispensed with.

Psychic factors were found to be important. In the early days of the study, patients were often found to void poorly or not al all when being tested but voided well in the toilet immediately afterwords. It is impossible to eliminate all psychic factors as the mere thought of being tested my produce some inhibition. The mental aspect however, may be minimized by isolating the patient behind a screen (although preferable in a separate small room) and keeping the noise and human activity to a minimum.

Stewart[14.] and Arbuckle[19.] both stress the importance of reducing psychic factors as much as possible. Only then can a normal or usual voiding pattern be recorded.

Stewart[14.] feels that only flow rates on voided volumes exceeding 200 cc. should be used, as those on less than 200 cc. vary greatly and ore not reliable.

von Garrelts[8.] refers to the direct relationship between rate of urine flow and volume of urine voided. He reasons that since the micturition pressure is constant with varying bladder volume, that the increase in urine flow with increase in bladder volume is due to decrease in urethral resistance. An attempt to explain this is presented in Figure 3.

<u>EMG of External Urethral Sphincter</u>

Several authors[2, 24, 25.] have referred to a tonic activity of the external urethral sphincter. This is not in agreement with our findings.

Lapides[26.] found that continence was maintained during paralysis of striated muscles with muscle relaxants (D-tubocurarine and succinylcholine) as it was following pudendal block. If the external urethral sphincter is not necessary to maintain continence in the normal individual under resting conditions, why then should it be tonically contracted?

During conditions of stress, however, such as coughing or straining, one would expect contraction of the external urethral sphincter in an attempt to assist the internal urethral sphincter in preventing incontinence. This is supported by the finding of greater activity of the external sphincter during coughing when the bladder is full as when it is only partly full. A full bladder may result in minimal opening of the bladder neck on coughing, placing more responsibility on the external sphincter in maintaining continence.

It was also noted that there was increased activity of the external sphincter when the bladder was at rest but full. The abrupt disappearance of action potentials during voiding is striking and confirms the findings of other authors.[24, 25.] Bursts of activity at the completion of voiding represent closure of the external sphincter.

The tonic activity of the external sphincter in the two patients that underwent transurethral resection of the bladder neck indicates the takeover of function of the external sphincter in the absence of the internal sphincter in preventing incontinence. The presence of tonic activity in the normal female studied could not be explained, except on the basis of cortical factors, as this patient was quite apprehensive throughout the test.

Spasticity of the external sphincter during voiding in cases of neurogenic bladder dysfunction is to be expected. This forms the basis for the rationale of the treatment of these patients by pudendal neurectomy or resection of the external sphincter.

It is noteworthy that suprapubic pressure in these patients increases the intravesical pressure, but as well brings about marked reflex contraction of the external urethral sphincter. This spasticity may sufficiently increase resistance to prevent or impede urination. Thus, the Crede maneuver tends to obstruct rather than aid voiding in certain cases of neurogenic bladder dysfunction. Although anal dilatation produces a similar responce of severe reflex contraction of the external sphincter, the activity subsides in time and may allow the patient to void. This difference in reaction to suprapubic pressure versus anal dilatation in paraplegics is noted by Abramson.[25.] He recommends anal dilatation as an aid to urination and condemns the Crede maneuver.

The amplitude of deflection of the EMG tracing is variable from one individual to another. In some the amplitude is so low that the excursion of the writing pen must be amplified. In others the amplitude must be reduced. This difference in amplitude probably depends on the differences in distance between the electrode point and the muscle motor units. Thus, it is not possible to state the voltage potentials in any case, or to compare one case with another. Instead we must be satisfied with analysing relative activity or inactivity of the sphincter in the case being studied.

As the placement of the electrode is essentially blind and relies upon the picking up of motor unit firing, it might be argued that the activity of one of the other muscles in the vicinity and not the external urethral sphincter is being recorded. The other muscles in the area are the bulbo-spongiosus, the ischio-cavernosus and the Transversus Perinei Superficialis in the superficial perineal pouch and the Transversus Perinei Profundus in the deep perineal pouch. These muscles are innervated by the pudendal nerve, as is the external urethral sphincter, and they contract simultaneously. Thus, the activity of one reflects the activity of the others.

An example of the simultaneous activity of different muscles innervated by the same nerve is provided by the Bulbo-cavernosus and Anal reflexes. The usual response tested in both reflexes is anal contraction, the efferent pathway being the pudendal nerve. However, the pudendal nerve also supplies the external urethral sphincter. Stimulation of the afferent limb of these reflexes is noted to bring about contraction of the external urethral sphincter as well as the external anal sphincter.

Petersen and Franksson[27.] inserted their electrodes into the external urethral sphincter via the urethra using a panendoscope. Whereas placement of the electrodes under direct vision can be achieved by this means, instrumentation of the urethra before testing and the presence of a foreign body in the urethra during testing would surely alter urethral resistance.

Urethral Resistance

It is realized that the formula presently being utilized, (i.e., $R = P/F$) for the calculation of urethral resistance may be oversimplified and inaccurate. On the other hand, it gives one a working tool which though inaccurate in terms of absolute resistance values, is most valuable in the determination of relative resistances. This presupposes a constant inaccuracy. The calculation of true resistance, if possible, would undoubtedly be much more difficult.

The average urethral resistance is relatively simple to compute and has the advantage of not requiring a flow-meter. The information derived however, is of questionable value. The figure for average resistance implies the average of all the resistances extending from the point of initial flow, when the resistance is high, through the peak flow when the resistance is lowest, to the end of flow when the resistance is again high. At wither end of this range the resistance is infinitely high (infinity), for F is 0 (i.e., P/0 = infinity). The most valuable figure is the minimum resistance. Thus, the significant minimum resistance is averaged with the far less significant resistances at either end of the range. Although a correlation was found between the clinical picture and average resistance it was not as great as with minimum resistance.

The normal range for average urethral resistance (male and female) was found to be 2.49 - 3.53 R units. These figures are lower than those reported by Arbuckle and Paquin[19.] who studied normal females only (3.38 - 5.46 R units). These authors used a transurethral catheter to record the intravesical pressure. The subjects had to void around the catheter which would therefore partially obstruct the urethra and increase urethral resistance.

Lattimer and Gleason[17.] calculated minimum urethral resistance by dividing maximum voiding pressure by maximum flow rate. As previously noted, the maximum flow does not correspond to the maximum pressure. There is therefore no urethral resistance which corresponds to that calculated by using maximum pressure and maximum flow. As the pressure at maximum flow is usually lower than the maximum pressure the calculation of minimum resistance by dividing pressure at maximum flow by maximum flow will give a lower figure. Lattimer and Gleason used two separate tracings to determine pressure (Lewis cystometer) and flow (Kaufman flow-meter). It is obvious that both these parameters must be on the same tracing to determine pressure at maximum flow.

The normal range for minimum urethral resistance, (male and female), was found to be 1.33 - 2.00 R units. These figures again are lower than those given by Arbuckle and Paquin[19.] for minimum urethral resistance (2.08 - 3.38 R units). The same explanation as that for average resistance probably applies here as well.

Note is made of the fact that a normal male had a lower average and also minimum resistance than either of the two normal females tested. The resistance in the case of the male was calculated with a voided volume of 575 cc., whereas that in the two females was calculated with voided volumes of 160 and 210 cc. respectively.

The explanation of this opponent discrepancy may become obvious by referring to Graph I. This is a graph of the normal male in question and shows a decrease in both average and minimum resistance as the volume voided increases. It would seem reasonable to suppose that if a normal female were similarly tested by multiple voidings, a corresponding curve would be obtained, but at a lower level. Thus, the urethral resistance of the male when the volume voided is large would be less than that of the female with a considerably smaller voided volume. Only when the voided volume is kept constant would the resistance in the female be consistently lower than that in the male. Similarly, a lower curve might be expected for male children and an even lower one for female children.

It is obvious then that a proper evaluation of urethral resistance will require the testing of a number of normal adults and children, both male and female by multiple voidings at increasing volumes. Standard graphs would then be set up for the 4 groups. At the completion of a study on a patient, the calculated urethral resistance would have to be referred to the specific volume on the appropriate graph to determine whether or not it fell within the normal range.

SUMMARY

A technique and clinical study of the simultaneous measurement of various parameters of micturition is presented. These include intravesical pressure, rate of urine flow, intrarectal pressure and electromyography of the external urethral sphincter. From the first two, an attempt is made to calculate a fifth parameter, viz., urethral resistance.

A total of twenty-nine studies were performed on twenty-three patients. Five of the patients studied were felt to have normal lower urinary tracts. Of the remainder 12 had evidence of obstructive uropathy and 6 had neurogenic bladder dysfunction.

The intravesical pressure was measured by means of a suprapubic catheter. Bladder tone was evaluated as the bladder filled slowly by physiological means. Changes in pressure were recorded during and after voiding and the pressure curves compared in the three groups studied.

The recording of intrarectal pressure, by means of a small balloon inserted into the rectum, was found useful in distinguishing deflections in the vesical pressure curve that were due to straining from those due to involuntary bladder contractions. Intrarectal pressure was not measured, but changes in pressure were noted.

Rate of urine flow was measured by means of a flowmeter and the flow curves in the three groups compared.

Electromyographic pattern of the external urethral

sphincter in the male and the bulbo-cavernosus muscle in the female was recorded by means of a co-axial needle electrode. Perineal insertion of the electrode left the urethra free. Sphincteric activity or inactivity was recorded during bladder filling and then during and following voiding.

Both average urethral resistance and minimum urethral resistance on voiding were calculated. Minimum urethral resistance was found to show a closer correlation with the clinical picture. Urethral resistance was noted to vary with sex and age (adult vs. child) and also with volume of urine voided. It was therefore concluded that normal standards would have to be established according to these three variables. Only then could a given figure for urethral resistance be evaluated with respect to normality.

CONCLUSION

The study presented represents a significant addition to the means available for the evaluation of lower urinary tract dysfunction. In addition, analysis of the data derived from the study of normal individuals provides for elucidation of the physiology of micturition. The simultaneous recording of the various parameters of micturition will undoubtedly play an increasingly important role in the study of lower urinary tract dynamics in future years.

The technique, as presented in this paper, though contributing significantly to the study of micturition, may be improved upon. These improvements are well within the realm of possibility. One might, for example, devise a suprapubic tube for the measurement of intravesical pressure that would reduce to a minimum the amount of mucosal irritation. This could be done by attaching a small inflatable balloon close to the end of the tube, much like a Foley catheter. Following insertion of the tube the balloon could be inflated and the tube gently withdrawn until the balloon just touched the bladder wall. In this way a very short length of tube would remain in the bladder lumen without the danger of slipping out. Nor could the end of the tube pass into the prostatic urethra.

As well, more exact means of measurement of intra-abdominal pressure and rate of urine flow should be sought.

The importance of psychic inhibition is stressed and therefore the construction of a test chamber is indispensable if one is to obtain a normal or usual voiding pattern for evaluation. Such a chamber should be small to resemble a toilet. It should be sound-proof and contain the necessary equipment only, the remainder of the apparatus and the examiner being outside.

The study is considered incomplete pending the addition of cine-fluoroscopy of the lower urinary tract. Ideally, the cine-fluoroscopy should be synchronized with the pressure-flow recording, so that the fluoroscopic picture at any one time can be correlated with the pressure and flow at that time. Statements will then be possible with respect to pressure and flow at various stages of bladder neck opening, pressure at which vesico-ureteral reflux, if present, occurs etc.

It is obvious from the presentation and discussion of the results of this study that no single figure, for example, that for minimum urethral resistance, will give the full picture of lower urinary tract dysfunction in any one case. Rather, it will be necessary to know about the presence or absence of vesico-ureteral reflux, straining or residual and their degree, the pressure and flow pattern, the EMG pattern of the external urehtral sphincter and last but not least the minimum urethral resistance. The last figure will have to be compared with the normal range for the specific age, sex and volume voided.

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