# Clinical and Urodynamic Evaluation of Diabetic Patients Presented with Voiding Disorders

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## ABSTRACT:

**OBJECTIVES:**

To ascertain the relationship between diabetic voiding dysfunction and vesico-sphincteric behavior, and try to point out any clinical prediction to bladder cystopathy in diabetic patients.

**METHODS:**

Fifty-one diabetic patients complaining of voiding disorders were studied over two years (Oct/2000-Oct/2002), according to history, clinical examination and investigations especially full urodynamic assessment, patients were classified urodynamiclly as either impaired detrusor contractility, detrusor hyperreflexia , detrusor areflexia , and normal.

**RESULTS:**

Mean bladder capacity was 479±123.9ml. with a mean first sensation of filling of 241.1±61.2ml. of the 55 patients 23(45.3%) impaired detrusor contractility), 18 (35.2%) had detrusor hyperreflexia, 4 (7.8%) had detrusor areflexia, and 6 (11.7%) were normal. The presence of sacral neurological signs and infection were found statistically significant in predicting an abnormal urodynamic pattern, while neither age, sex, nor the type and severity of diabetes were found related.

**CONCLUSION:**

These data suggest that classical diabetic cystopathy is not the most common urodynamic findings in patients with diabetes mellitus and voiding dysfunction, and in fact these patients present with variable pathophysiological findings. These findings demonstrate the importance of urodynamic studies in diagnosing voiding dysfunction in diabetics before initiation of therapy.


***KEY WORDS:*** Diabetes mellitus, Diabetic cystopathy , Urodynamics

## INTRODUCTION:

Diabetic bladder dysfunction is among the most common and bothersome complications of diabetes mellitus. Studies suggest that up to 40% of diabetics will have some form of voiding dysfunction.[1]The bladder malfunction specifically associated with diabetes mellitus is called diabetic cystopathy (DCP) [ 2, 3, 4] Diabetic cystopathy (DCP) the term coined by Frimodt-Moller [5, 6, 7] It might be defined as a disturbance in the function of the urinary bladder in diabetic patients characterized by loss of sensation and increase bladder capacity without signs of mechanical outlet obstruction. [2, 5, 6, 7] The exact incidence of voiding dysfunction in diabetic patients is uncertain, as unselected patients generally don't complain of bladder symptoms. If specifically questioned up to 50% may report symptoms of voiding dysfunction. .[8, 10, 11, 12, 13] DCP begins with selective damage to autonomic afferent nerves, leaving motor function intact but impairing the sensation of bladder fullness. [14, 15, 16, 17] Chronic hyperglycemia is associated with loss of mylinated and unmylinated fibers, wallerian degeneration, and blunted nerve fiber reproduction. [6, 14] It has been reported that early changes in bladder function may be due to functional adaptation to bladder over distension And/or an increase in bladder work induced by diuresis caused by hyperglycemia. [8, 18] It has been suggested that nerve growth factor NGF has a role in DCP through its effect on the afferent dorsal root ganglion. [18, 19]

## MATERIALS AND METHODS:

Fifty-one diabetic patients complaining of voiding disorders were studied over two years (Oct/2000-Oct/2002), in the Urological Department of Al-Yarmuk Teaching Hospital. Those with abnormalities of the sacral reflex arc $S_2$-$S_4$ (absent lower limb and perineal sensation, lax anal sphincter tone, absent bulbocavernosus reflex, absent ankle jerk) were deemed sacral cord signs positive and those with intact sacral reflexes and sensations were deemed sacral cord signs negative. Investigations were done including urinalysis with culture, blood sugar, renal function tests, x-ray imaging and ultrasound studies. Flowmetery first test was done for all, excluding those who were unable to void in whom indwelling catheters have inserted. Then we perform cystometry test as following, Cystometry study filling phase in supine position, urethral catheterization was done with special 10 F. double lumen catheter introduced into the bladder. Here we have an opportunity to measure post void residual urine before starting cystometry. Isotonic saline was used for bladder

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filling at a rate of 30-100ml/minute. Intra-abdominal pressure was recorded via an 8F feeding tube introduced into the rectum. The electromyography's was recorded by using mainly concentric needle electrode inserted percutaneously in the perineal area near the anal verge in few patients we use anal plug electrod. All the system was calibrated at atmospheric pressure making the reference point at the level of superior edge of symphysis pubis. Before and during filling the individual was asked to cough for checking the correct position of the catheters as we noticed a rise in the abdominal pressure. At the end of filling phase we noticed the bladder capacity, compliance, and the presence of any uninhibited detrusor contraction. Voiding phase at this phase we ask the patient to attempt voiding on sitting position. Detrusor pressure was recorded, sometimes the patient unable to start voiding because of unusual circumstances in these situation we repeat the test again at another time. The results and graphs were electronically visualized on the monitor and were printed on thermal paper. Urethral pressure profile is started to be measured using a double lumen 10F catheter. A slow saline drip of 2ml/min used while the catheter slowly pulled out at a rate of 2mm/sec meanwhile the urethral pressure profile is recorded and intravesical pressure is measured simultaneously. Average EMG activity was recorded during the whole procedure through the needle electrode to detect detrusor sphincter dyssynergia (DSD). Patients were divided urodynamically according to their over all findings into:

*1-Hypoactive bladder group:* present with poor flow rate, increased compliance, and large bladder volume of more than 600 ml. They usually have weak voiding detrusor contraction below 30cm $H_2O$ and the expulsive force of voiding is usually supported by straining. [fig.1].

***2-****Detrusor hyperreflexia group:* Here the urinary flow rate may be within normal limits or less than 12 ml/sec, presence of uninhibited detrusor contractions. Some of them have detrusor sphincter dyssynergia. Voiding detrusor pressuzre is usually greater than 50 cm$H_2O$. [fig. 2,3,4,7].

***3-****Normal bladder function group:* Present with normal flow rate pattern and normal cystometric findings. [fig.5]. *4-Detrusor areflexia group:* Patients present with retention of urine, loss of sensation, and absence of detrusor contraction during the attempt to void. [fig.6].

Statistical evaluation**:** The significance between mean values was analyzed by Pearson Chi-square test and fissure exact probability index. Multiple groups comparison was made using analysis of variance (ANOVA). P value of<0.05 was considered as a significant relation for all data shown in our work.

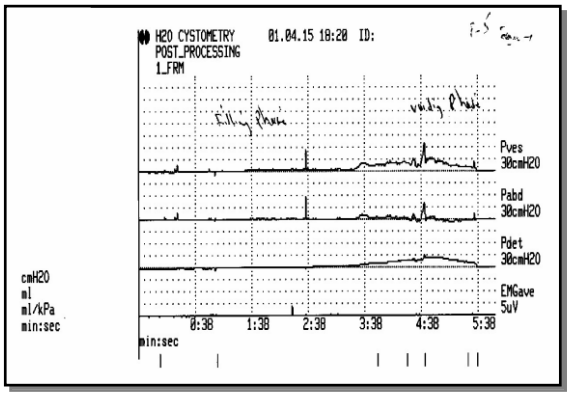


**Fig.1: Hypoactive contraction**

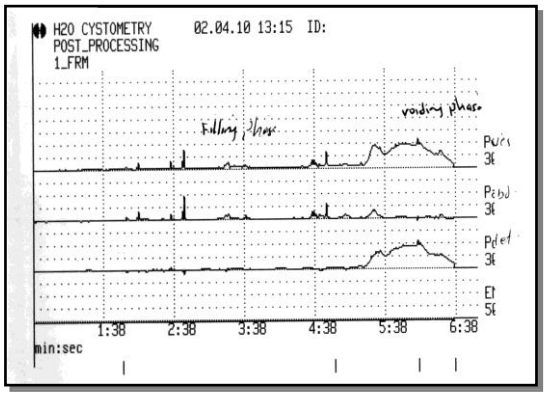


**Fig. 2: Detrusor hyperreflexia.**

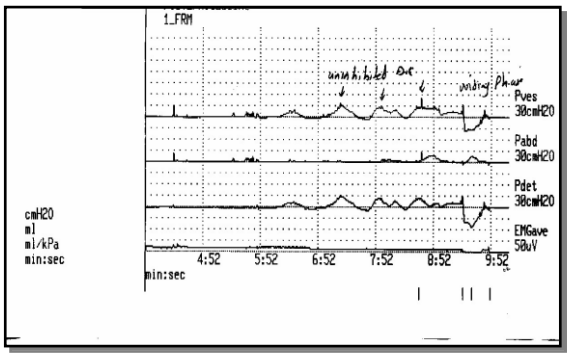


**Fig. 3: Detrusor Instability**

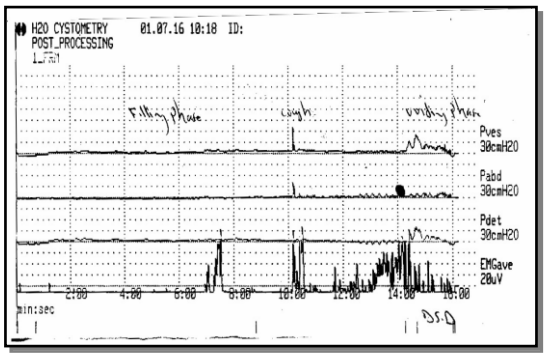


**Fig. 4: Detrusor sphincter dyssynergia**

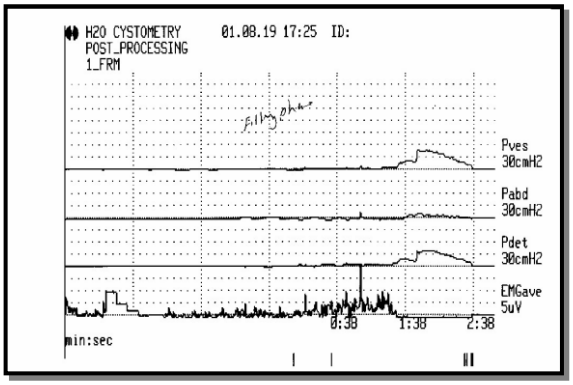


**Fig. 5: Normal voiding pattern.**

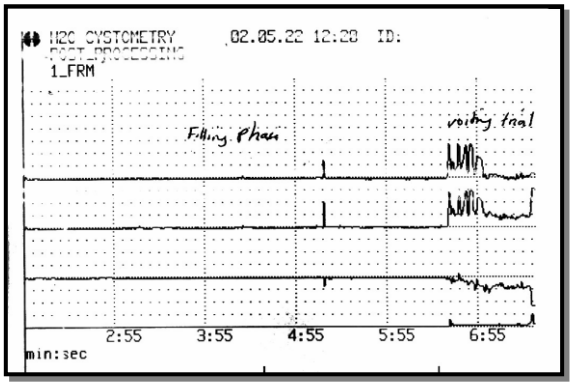


**Fig. 6: Detrusor areflexia**

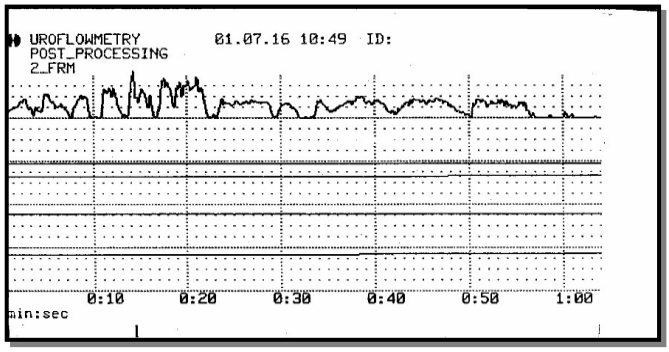


**Fig. 7: Intermittent pattern flow**

## RESULTS:

Fifty-one diabetic patients were studied with a median age of (61.5) years ranging between (24-82) years. The majority were males (76.5%) while (23.5%) were females. Neither the age nor the sex was found having significant impact on the urodynamic group patterns. P value 0.678. Although (88.2%) of the studied group were found having abnormal urodynamic pattern, only (7.8%) of them were having pure classical areflexia, while the commonest group is hypoactive detrusor contractility (45.3%).[fig.8] The mean duration of diabetes was (10.4) years the longer duration of diabetes (>10years or >15years) was not found significantly related to having abnormal urodynamic pattern nor was it related to any of the abnormal urodynamic group.P value 0.063 & 0.056 respectively. The most common presentation was urine retention in 22 patients (43.1%).

Three of them were females in the hypoactive group and 19 males 48.7%, they were as follow; four in the areflexic group, nine in the hypoactive group and six in hyperreflexic group.

Fifteen patients (29.4%) presented with irritative voiding symptoms representing most of the female population, while 14 patients (27.5%) presented with obstructive voiding symptoms.Clinically diagnosed peripheral neuropathy or sacral sign (SC) positive were seen in 47.1% of patients.

This was significantly related to urodynamic patterns. The entire normal group was SC negative. P value 0.026. [fig.9]. Twenty-four patients (47%) were having urinary tract infection. Those having infection found statistically more prone to develop abnormal urodynamic pattern mainly hypoactive detrusor contractility, while no growth was found in all the normal group cases. P value 0.032. Imaging X-ray studies and ultrasound revealed dilated upper tract in 11 patients (21%). Mean post voiding residual urine was 98ml (nil-440) ml. and mean prostatic size was 49ml (11-163ml). Among the hyperreflexic group the majority (38.9%) presented with irritative voiding symptoms, only 27.8% of patients in this group showed uninhibited detrusor contractions (detrusor instability) in their urodynamic study, and 16.7% showed detrusor sphincter dyssynergia.

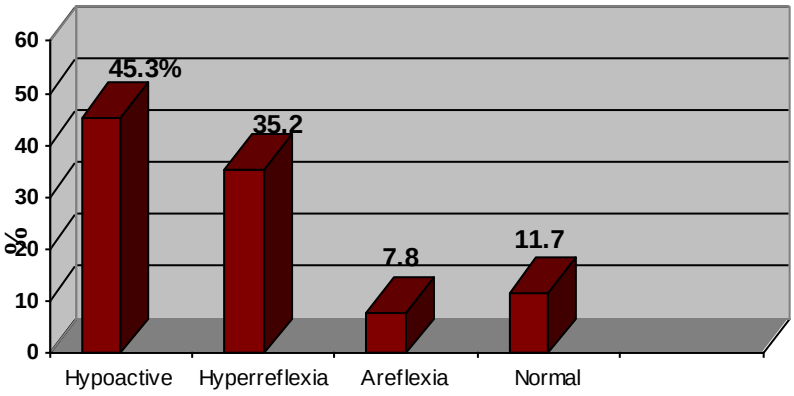


**Fig. 8: The urodynamic groups of diabetic patients.**

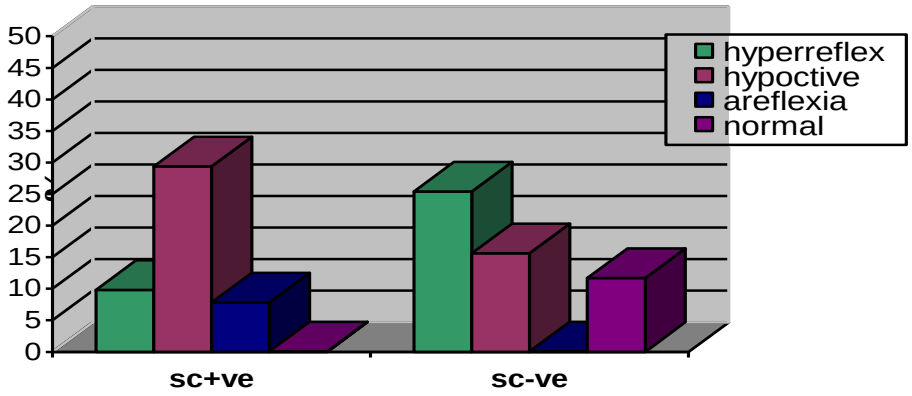


**Fig.9: Neurological findings in the urodynamic groups**

The over all urodynamic parameters found in studying our patients are summarized in table

**Table1: Mean values of urodynamic results**.

| Parameter | range | mean ± SD |
|---|---|---|
| First sensation of filling | 241.1±61.2ml | 99-456ml |
| Bladder capacity | 479±123.9ml | 290-691ml |
| Maximum detrusor pressure | 37.51±19 cm.$H_2O$ | 0-90cm. $H_2O$ |
| Maximum flow rate | 10.0 ± 10ml/sec | 0-30ml/sec |
| Average flow rate | 4.7 ± 5.0ml/sec | 0-17 ml/sec |
| Maximum urethral pressure | 65 ± 20cm. $H_2O$ | 33-121 cm. $H_2O$ |
| Urethral closure pressure | 56±19 cm. $H_2O$ | 28-106 cm. $H_2O$ |

## DISCUSSION:

In this study, the classical findings of Diabetic Cystopathy were not the most common urodynamic diagnosis. In our study neither the age nor the sex were found related to urodynamic groups and this is compatible with others, as Frimodit Moller who showed identical percentages of DCP among males and females.[5]

In our study 88.2% of diabetic patients presented with voiding symptoms were having abnormal urodynamic patterns, 45.3% were in the hypoactive group. In other studies the frequency of abnormal bladder function in diabetic patients, occur in 25 to 85% of the cases, depends on the selection pattern, diagnostic method used and duration of diabetes mellitus. [1, 10, 11, 13] Frimodt-Moller reported on 124 diabetic patients of whom 38% had DCP and 26% had BOO. [7, 13] Kitami et al performed urodynamic studies on 173 diabetics and demonstrated that diabetic patients showed varieties of vesicourethral dysfunction, such as over active bladder (14.5%), low compliance bladder (11.0%) and loss of detrusor- external sphincter coordination (31.7%).[8, 20, 21, 22] The recent fact is that classical hypoactive and areflexic bladder reported by Frimodt-Moller in 1976 is not the only urodynamic abnormality found in diabetic patients as shown in

the reports of more recent authors and was found as well in our work. It's unclear if there is an association between bladder dysfunction and the severity of diabetes or the type of diabetic therapy used. [1, 5] In our work we couldn't find a significant relation between duration of diabetes and having an abnormal urodynamic pattern, this may be attributed to the small number of normal urodynamic cases found in the studied group owing to the type of patient selection.

In histological study of the autonomic nerve fibers of the urinary bladder wall, the most marked alterations occurred found in subjects with poorly controlled diabetes [8] however, others believe that duration and severity of diabetes is not associated with bladder dysfunction. [1, 5, 23]

All the urodynamic abnormal patterns can present with retention as seen in our work, and in this concept the hyperreflexic group represent a dilemma in diagnosis due to the intermingle effect of BOO. In elderly male population with prostatic enlargement. [1, 7] By analysing our six-hyperreflexic males presented with retention, we found that in at least two of them, their abnormal urodynamic pattern was not related to prostatic obstruction as cleared from their neurological exam, urodynamic evaluation, and cystoscopy.

It has been documented that detrusor overactivity is a manifestation of diabetic patients.[7, 8, 13] However, its uncertain whether diabetes per se induces detrusor overacticity since other concomitant conditions, including suprasacral neurological lesion or BPH possibly cause the same condition.[1,7,8,14, 26] Recent study by Hong-Jeng et al, they found urethral outlet ( smooth and striated muscle dysfunctions ) during reflex bladder contractions in diabetic induced rats. This study my help to explain the mechanism of detrusor hyperreflexia in diabetic patients. [26]

We found that 8 out of the 15 male patients presented with retention in the hyperreflexic, hypoactive, and a reflexic groups whom ages were ≥ 60 years old with prostate more than 30 ml in size, will remain having retention or very poor evacuation of bladder after prostatectomy as predicted by full neurological exam, urodynamic evaluation, and cystoscopy done preoperatively thus their prostate was not the important cause of their retention. Stressing on the role of urodynamic studies in preventing unnecessary prostatectomies in diabetic males. The clinical finding of sacral sign positive was Statistically related in our study to the resulted urodynamic abnormality.

This is compatible with other studies. [5,7,8,25] Thus we can predict having DCP in sacral sign positive diabetic patients.

## CONCLUSION:

These data suggest that the classical diabetic cystopathy is not the only common urodynamic finding in patients with diabetes mellitus.

In our work the presented urodynamic pattern were hypoactive, hyperreflexia, a reflexia, and yet still some had normal urodynamic pattern.

We find there is strong correlation between peripheral neuropathy and infection with the resulted urodynamic groups mainly the hypoactive group, but neither the age nor the sex was found significantly correlated These findings demonstrate the importance of urodynamic studies in evaluating voiding dysfunction in diabetics before initiation of therapy.

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