Evaluation of Bulbocavernosus Reflex in the Investigation of Diabetic Impotence

Usama Al-Nasiri

ABSTRACT:

BACKGROUND:

The pathology of erectile dysfunction may be an autonomic neuropathy, vasculogenic, endocrine deficiency, psychological or a combination of these factors. This study was undertaken to determine the predictive value of Electrophysiological elicited Bulbocavernosus Reflex in discrimination between neurogenic and non- neurogenic impotence in diabetic patient.

METHODS:

Twenty five male diabetic patients were recruited from out-patients attending the urology department at AL-KADIMYA Teaching Hospital. Bulbocavernosus Reflex (BCR) was evaluated on all patients at AL- NAHRAIN College of Medicine department of physiology, using the methods described by Rushworth.

RESULTS:

Test sensitivity was 72%, while test specificity was 83.3%. An abnormal Bulbocavernosus reflex carries a high probability of neuropathology.

CONCLUSION:

A degree of objectivity in the evaluation of possible neurogenic impotence can be offered by testing the Bulbocavernosus Reflex.

KEY WORDS: Erectile dysfunction, Diabetes mellitus, Bulbocavernosus reflex.

INTRODUCTION:

National Institutes of Health defines erectile dysfunction (ED) as the consistent inability to achieve and maintain an erection sufficient for satisfactory sexual activity 1... The disorder is associated with age, with a 39% prevalence at the age of 40 years and a 67% prevalence at the age of 70 years. Massachusetts Male Aging Study reported on 52% of men aged 40-70 years having some degree of erectile difficulty. ^{2,3} Patients with ED undergo a preliminary assessment designed to address significant diseases such as diabetes, coronary artery disease (CAD) and hypertension before starting therapy, so non- or/and least invasive treatments are to be used first.4,5,6 most studies of diabetic males, erectile dysfunction has been a common complaint. The high prevalence of impotence in diabetics is due to presence of several mechanisms for this complaint, which are themselves, common complications of diabetes⁷. The chronic hyperglycemic condition is associated with long term damage, dysfunction and failure of various organs especially eyes, kidneys nerves heart and blood vessels 8. Complications of DM include acute complications that are generally a reflection of altered energy homeostasis either (diabetic hyperglycemia ketoacidosis hyperosmolar syndrome) or hypoglycemia and

Department of Urology, College of Medicine, Al-Nahrain University

Chronic complications consisting of retinopathy, neuropathy and angiopathy⁹. The incidence of impotence in diabetic men has been estimated at between 40 and 60% 10. The pathology may be an autonomic neuropathy, vasculogenic, endocrine deficiency, psychological or a combination of these factors¹¹. The incidences of neuropathy in diabetic impotence has been estimated at 55 to 85% in different studies ^{11, 12}. An endocrinological cause for impotence in diabetes mellitus can be effectively excluded⁹. A neuropathic vasculogenic pathogenesis should be considered in cases thought to have an organic cause¹³. Diabetic autonomic neuropathy is among the least recognized and understood complications of diabetes despite its significant negative impact on survival and quality of life in people with diabetes^{14, 15.} Several Different factors have been implicated in the pathogenic process.

Heperglyceamic activation of the polyol pathway leading to accumulation of sorbitol may cause direct neuronal damage and /or decreased nerve

Blood flow, so does activation of protein kinase C by inducing vasoconstriction and reducing neuronal blood flow¹⁶ The precise role of the BCR in the routine investigation of the impotent diabetic patient remains to be defined. This study was undertaken to address this issue and also to determine the predictive value of this test in discrimination between neurogenic and non-

neurogenic impotence in diabetic patient. **PATIENTS AND METHODS:**

This study is a prospective study carried out from December 2004 to November 2005 at AL-KADIMYA teaching hospital, linked to AL-NAHRAIN College of medicine department of physiology. Twenty five male diabetic patients (mean age 49.1 years, range 30-63) from outpatients attending the urology department at AL-KADIMYA Teaching Hospital presented with loss of potency following the onset of diabetes mellitus, were studied .Those with any other neurological disorders not directly attributed to diabetes mellitus were excluded. Standard history, general physical and neurological examination, routine laboratory, hormonal profiles, and psychological evaluation were done. Bulbocavernosus Reflex (BCR) was performed on all patients at AL- NAHRAIN College of medicine department of physiology, using the methods described by Rushworth ¹⁷.

The glans penis was stimulated with rectangular pulses of 0.1 and\ or 0.2 msec. duration at intensities beginning from "0" rising up to 200 v in a stepwise manner. The psycho-sensory threshold for sensation on the glans penis was estimated by such a progressive increase in stimulation at a rate of 1 sec, in each case with durations of stimulation of 0.1, 0.2 and sometimes 0.5, 1.0 up to 2.0 msec. The reflex threshold from the BC muscle was also determined by the same procedure. Finally, the glans penis was stimulated maximally. In all normal subjects, rectangular pulses with 0.2 msec. duration and 80-150 v intensity were sufficient to evoke very stable reflex response from the BC muscle. The maximum electrical stimuli used with all normal control males and with the majority of the patients investigated did not evoke any painful sensations in the patients. The sweep speed used for recording the reflex action potentials on the film was 2.5and 5.0 msec\mm. The reflex conduction time was measured from the beginning of the stimulus artifact to the onset of the first action potential of the reflex response. The BCR considered to be abnormal when absent or when

the onset of latency was more than 44 msec¹⁸. Twenty four age matched, potent, neurologically normal male were also studied by the same technique and result served as controls.

RESULTS:

The mean age of patients was 49.1 years (range: 30-63 yrs). The duration of the diabetes mellitus varied from 8 years to 24 years; (mean = 14.5 years, median 16years). The rectangular electrical shocks with 0.1—0.2 msec. duration applied on the glans penis were first perceived at about an intensity range of 80—150v mean 95v in all normal males. Although the psychosensory threshold of a given patient could vary for different trials, such differences were not significant so that approximate values could be assigned to threshold. Electrical shocks 1.5—2.5 times stronger than the reflex threshold produced stable and constant EMG discharges in the BC muscle.Fig1 shows a BC reflex response from a normal control males.

The mean latency of the reflex was 33.6+5.6 msec. and ranged from 21.5 to 44.3 msec in 24 normal males. The reflex had the shape of a single motor unit response .these results was similar to those reported by other workers 12, 19, 20. Four out of twenty four persons (16.6%) in control group had abnormal BCR latency. Out of twenty five diabetic impotent patients, the mean latency of the reflex was 49.2±9.7 and range from 28.5-67.4, which is statistically significantly prolonged in comparison with normal control values using t-test with (t=5.94, p=0.0001). Table1 summarizes the results of BCRL from the normal controls & the patient groups. There were 6 cases out of 25 diabetic impotent patients (24%) still within normal limits, one of them had grossly elevated sensory threshold (280v). Four out of 19 diabetic impotent patients with prolong latency showed elevated sensory thresholds i.e. more than 150v (21%) other 15 diabetic impotent patients had normal sensory thresholds (80-150 v). Fig.2 shows a BC reflex response from a diabetic impotent patient. Fig. 3 is a diagram show distribution latency period (L.P.) among control and patients Test sensitivity was 72%, while test specificity was 83.3%.

Group	Latency	
	Mean <u>+</u> SD	(Minimum-Maximum)
	msec.	msec.
Control	33.6 <u>+</u> 5.6	(21.4-44.3)
DM Impotence	49.2 <u>+</u> 9.7	(28.5-67.4)

Table: 1 summarizes the results of BCRL from the normal controls & the patient groups.

Control V DM with impotence, t=5.94, P=0.0001 (Significant)

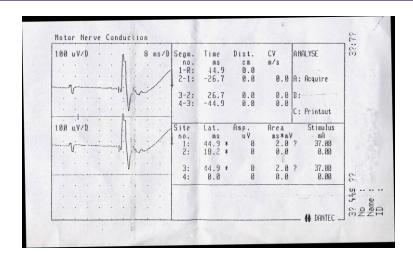


Fig. 1 shows a BC reflex response from a normal control subject.

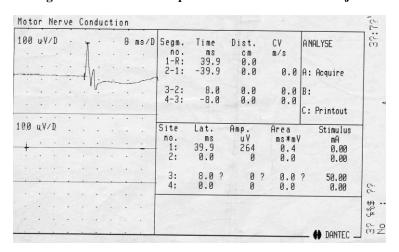


Fig.2 shows a BC reflex response from a diabetic impotent patient.



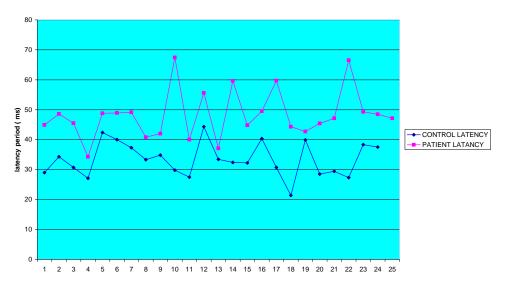


Fig.3 diagram show distribution of latency period (L.P.) among control & patients

DISCUSSION:

The mechanism of erection is an autonomic response predominantly a parasympathetic function, though there is histochemical evidence for a sympathetic role as well⁹.

The BCR latencies measure a somatic reflex pathway from the dorsal nerve of penis via sensory pudendal afferents to motor pudendal efferent and because the lateral branch of the pudendal plexus is a mixed autonomic and somatic nerve¹³, it would be rare for a patient to have solely an autonomic lesion without involvement of the somatic fibers¹⁰, therefore the BCR represent an indirect assessment of the autonomic pathway of erection .

The BCR latency in the entire control population was (33.6+5.6 msec). which is within the limits of normal healthy subjects recorded by other workers ^{19, 20}. In this study out of 25 diabetic impotent patients 19 (76%) patients has longer latency $(49.2\pm9.7 \text{ msec})$ i.e. abnormal results which refer to the presence of a neurological deficit, four of them showed elevated sensory thresholds with prolonged conduction latencies, this would indicate a peripheral neuropathy, while others had prolongation of BCR latencies with normal sensory threshold indicating lesion in the periphero-conus pathway. One patient with normal BCR latency had grossly elevated sensory threshold indicating a lesion in either the ascending tracts or cerebral cortex with a normal efferent pathway.

The percentage of diabetic impotent patients with neurogenic cause of there impotence (with abnormal BCR latency and\or elevated sensory thresholds) was 80% in this study; this suggests that figures of 85% estimated by other worker^{21,} are slightly overestimated. On the other hand the relationship between erectile dysfunction and metabolic pathologies has been researched mostly in diabetic patients as it progressed with neuropathy and microangiopathy. In 12-34 % of diabetic patients, prolonged BCR-L has been reported, relating to neuropathy²².

The 6 diabetic impotent patients with normal BCR latency (24%) may refer to other causes of impotence in diabetes mellitus (vasculogenic, endocrinologic, etc).

There is no explanation in this study for the four control persons with abnormal BCRL.

The BCR has accepted sensitivity &specificity (72%, 83.3%) respectively so it is a useful and accurate investigation in determining the cause of diabetic impotence. An abnormal bulbocavernosus reflex (BCR) latency time, defined as a value greater than 3 standard deviations above the mean (30-40 msec), carries a high probability of neuropathology²³.

CONCLUSION:

1-Incorporating assessment of BCR to the impotent patient, we can offer a degree of objectivity in the evaluation of possible neurogenic impotence.

2-In analyzing conduction latency studies, measurement of the sensory threshold in the neurophysiologic parameters should be included .This will increase diagnostic accuracy, identifying neurological lesions in impotent diabetic men, in the face of normal conduction latencies, and thus may be implicated in the etiology of impotence.

3- Although impotence is a multifactorial disorder, yet the present study revealed that the neurogenic affection is the major contributory factor. **REFERENCES:**

- 1. D Golijanin; E Singer; R Davis; S Bhatt; A Seftel; V Dogra Int J Impot Res. 2007;19:37-42.
- Johannes CB, Araujo AB, Feldman HA, Derby CA, Kleinmann KP, McKinlay JB. Incidence of erectile dysfunction in men 40-69 years old. Longitudinal Results from the Massachusetts Male Aging Study. J Urol 2000; 163: 460-463.
- 3. Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. J Urol 1994; 151: 54-61.
- **4.** Padma-Nathan H. Diagnostic and treatment strategies for erectile dysfunction: the 'process of care' model. Int J Impot Res 2000; 12(Suppl 4): S119-S121.
- 5. Gingell C, Wright P, Barnes T, Dean J, Dinsmore W, Eardley I et al. Guidance on the management of erectile dysfunction in primary care. Prescriber 1999; (Suppl 5): 1-15.
- 6. Jardin A, Wagner G, Khoury S. Recommendations of the 1st International Consultation on Erectile Dysfunction. In: Jardin A, Wagner G, Khoury S, Giuliano F, Padma-Nathan H, Rosen R (eds). Erectile Dysfunction. Plymbridge Distributors Ltd.: Plymouth, UK, 2000, pp 711-726.
- **7.** Kolodny, R.C., Kahan. Sexual dysfunction in diabetic men .diabetes, 1988; 23,306-309.
- **8.** Yousef WM, Omar AH, Morsy MD, Abd El-Wahed MM, Ghanayem. The role of calcium channel blockers in the treatment of diabetic nephropathy. Diabetes and metabolism. 2005; 13:68-75.
- 9. DCCT [Diabetic Control and Complication Trial]m Research Group: Retinopathy and nephropathy in patients with type 1 diabetes, four years after a trial of intensive therapy. N Engl J Med. 2000; 342381-89.

- Benet AE, Melman A: The epidemiology of erectile dysfunction. Urol Clin North Am Nov.1995; 22: 699-709.
- **11.** Sharlip, I.D.Clinical andrology .In general urology, Ed, Smith, D.R. Eleventh edition. 1984; chapter 35.pp. 622-626.
- **12.** Lehman, T.P. & Jacobs, J.A. Etiology of diabetic impotence. J.UROl.1983; 129: 291-294.
- Althof SE, Seftel AD: The evaluation and management of erectile dysfunction. Psychiatry Clin North Am Mar. 1995; 18: 171-92.
- **14.** Vinik AL, Erbas T. Recognizing and treating diabetic autonomic neuropathy. Cleve Clin J Med. 2001; 68:926-944.
- **15.** Freman R.The peiphral nervous system and diabetes.In Loslin's Diabetes Mellitus. Wwir G,Kahn GL, Eds.Philadelphia,lippincott,2002.
- **16.** Veves A, King GL. Can VEGF reverse diabetic neuropathy in human subjects? J Clin Invest.2001; 107:1215-1218
- 17. Rushworth, C. Diagnostic value of the electromyography study of reflex activity in man. In Recent advances in Clinical Neurophysiology, ed. Widen, L.1967; Suppl. No. 25 to Electroencephalogram.

- Neurophysiology Clin. pp. 65-73. Amsterdam: Elsevier.
- **18.** Vodusek DM, Janko M. The bulbocavernosus reflex. Brain.1990; 113:813-20 (1990)
- **19.** Rockswold.G. L. and Bradley. The use of evoked myographic responses in diagnosing lesions of the cauda equina. J. urol.1977; I18, 629—631.
- **20.** Yalla,S.V.,Di Bernadetto,M.,Blunt,K. , SETH.J.M & Fam,B.A. urethral striated sphincter responses to electro-bulbocavernosus stimulation .J.urol, .1978;119,406-409.
- 21. Vacek, J.and Lochman, M. Bulbocavernosus reflex in diabetics with erectile disorders, clinical and electromyographical studies cas. lek.Cesk. 1977; 116, 1014-1017. (1977).
- **22.** A. Ardicoglu, T. Ardicoglu, V. Yuzgec: The Role Of Bulbo Cavernosus Reflex Latency (Bcr- L) In The Etiology Of Patients With Erectile Dysfunction. *The Internet Journal of Urology*. 2006:3, 2.
- 23. Advanced Testing for ED• Male Sexual Dysfunction May 05, 2006. Cited from www.health.am