

THE BULBOCAVERNOSUS REFLEX IN DIABETIC IMPOTENCE

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SYNOPSIS

Diabetic impotence is a common and distressing problem among male diabetics whose libido remain intact. For management, it would be necessary to ascertain whether impotence is psychogenic or organic. We found that measuring the latency at onset of the electrical bulbocavernosus reflex response is a useful and simple method of documenting neurogenic impotence. Fourteen impotent diabetic males were compared with eighteen normal controls. Nine of the patients had significant delay in latency; two showed no response; two had a borderline delay; and only one patient had a latency response within normal limits. Abnormal bulbocavernosus reflex reflects closely lower parasympathetic dysfunction which presents as impotence. The absence of the reflex elicited clinically is also a useful bedside sign.

INTRODUCTION

Ellenberg (1) reported that the prevalence of impotence in diabetic men is as high as 50-60%. Since it is well known that psychological impotence is the commonest cause in the non-diabetic population, it is necessary to distinguish organic from psychogenic impotence even among diabetics. We report that the bulbocavernosus reflex (BCR) measured electrophysiologically and tested clinically is a reliable way of confirming a neurogenic cause. These findings support the reports by Vacek et Lachman (2) and Karacan (3).

PATIENTS AND METHODS

Normal values were established by studying a group of eighteen volunteers whose ages ranged from 32 to 66 years. All had no peripheral neuropathy and the BCR was present clinically. Fourteen male diabetics, ages 42 to 61 years, were selected. All had complained of impotence (failure of erection with loss of nocturnal emission and early morning erection) for more than six months duration. Libido remained intact. Other causes of impotence e.g. cauda equina compression, arteriosclerosis of aortic bifurcation, trauma and drugs were carefully ruled out. Doppler measurement of penile systolic pressure was not done. The sural, median motor, median sensory and posterior tibial nerves were studied by nerve conduction velocities and recording amplitude of the responses.

The bulbocavernosus reflex was clinically tested by brisk manual compression of the glandular penis and palpating for contraction of the bulbocavernosus and ischiocavernosus muscles at the perineum. Electrically the reflex was tested by a modification of the technique described by Ertekein (4). Briefly, a stable response is obtained by stimulating the glans penis with ring electrodes at 0.2 ms pulse duration and 60 volts or 1.5 times above threshold level. This intensity is non-nociceptive and diabetics may require stronger stimulation. The recording electrode is a tiny unipolar needle inserted into the corpus cavernosus through the perineum. The indifferent surface electrode is located at the lower sacrum. The patient lies on his side doing the procedure.

RESULTS

The normal response obtained on the oscilloscope is negative bifid wave. Latency at onset is measured. Ampli-

tude is widely variable and not reliable. Our normal value for onset latency is 38 ± 3.4 ms. Values above 45 m sec is considered abnormal. The repeatability and reproducibility of this test is demonstrated by the fact that other workers (4, 5) have similar normal values for latency.

Table 1 shows the latency values for the diabetics. Nine had significant delay ($> 3SD$); two showed absent responses; two had borderline values ($> 2SD < 3SD$); and only one showed a normal response ($< 2SD$). Peripheral neuropathy was confirmed in all patients electrophysiologically.

DISCUSSION

The BCR is probably a polysynaptic flexor cutaneous reflex and latency varies at minimal stimulation. However, the reflex is stable at stimulus intensity stated and the normal values given are similarly to that of other workers. The afferent limb of the reflex pathway is via the dorsal nerve of the penis and the pudental nerve. At the sacral segment probably both somatic and parasympathetic afferent pathways are via the pudental nerve and *nervi erigentes*. Even if the method tests purely somato-somato pathways as argued by Siroky et al (5), it, nevertheless, reflects autonomic lower parasympathetic involvement accurately from our data. The use of the reflex in detecting cauda equina lesions e.g. disc herniation, trauma and tumour is well established (6, 7).

Latency values of more than 80 milliseconds are occasionally found (Table) and Ertekein et al (7) have noted such values in disc herniation. They hypothesise that local segmental demyelination is the cause and probably a similar pathology exists in the BCR pathway of impotent diabetics. We observed that in diabetics, peripheral neuropathy is associated with impotence as a rule.

**TABLE 1
BULBOCAVERNOSUS REFLEX (BCR) RESPONSES IN 14 DIABETICS WITH THE COMPLAINT OF IMPOTENCE (AND NORMAL VALUES)**

Number of Patients	Age (years)	Latency at Onset of Responses (ms)	Clinical Bulbocavernosus Reflex
1	42	79.6	Absent
2	42	45.2	Present
3	47	50.8	Absent
4	48	51.0	Absent
5	49	79.2	Absent
6	49	47.2	Present
7	50	71.3	Absent
8	53	69.0	Absent
9	55	Absent	Absent
10	55	Absent	Absent
11	55	66.6	Absent
12	57	56.0	Absent
13	60	82.0	Absent
14	61	42.9	Present

Normal Values for BCR
(18 volunteers age range 32-66 years)
Mean of onset latency = $38.1 \text{ ms} \pm 3.4 \text{ SD}$
+ 2SD = 44.9 ms
+ 3SD = 48.3 ms

Since in psychological impotence, the BCR is normal (6), prolonged latency would be a useful way of confirming neurogenic impotence in a diabetic. Prior to the test, organic impotence can be established by nocturnal penile tumescence measurements and vascular causes ruled out by doppler measurement of penile systolic pressure (3). The clinical sign of an absent BCR is also helpful. The value of this electrophysiologic test in detecting early impotence needs further study.

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