Diabetic erectile dysfunction – is prevention possible?

Dear Sir,

We read with interest the article by Schiavi and co-workers [1]. Diabetic erectile dysfunction has been and remains an often neglected aspect of the overall care of diabetic men. The authors provide us with a reminder both of its high prevalence and the need for it to be addressed. There are a number of points which we would like to discuss which are pertinent to the present study.

The precise role of nocturnal penile tumescence recording in the evaluation of erectile dysfunction and the interpretation of this investigation remains much disputed and debated. The present study raises again some of the issues that were reviewed authoritatively by Morales et al. [2]. The basic premise is that patients with psychogenic impotence have unimpaired nocturnal penile tumescence, while those with an organic aetiology have impaired nocturnal tumescence. At best this represents an oversimplification of a complex behavioural phenomenon. However, although this intuitively appealing theory has lacked adequate standardisation, the use of nocturnal penile tumescence has gained widespread popularity. It may be argued that abnormal nocturnal penile tumescence only provides confirmation of the absence of normal erections during sleep and this may occur because sleep erections do not involve the neural pathways involved in subsuming erections in an erotic setting. Nocturnal penile tumescence recording should be used in conjunction with other tests of erectile function as the results in isolation often have the effect of forcing a perhaps spurious diagnostic dichotomy. Indeed, the assignment of a purely organic or psychogenic aetiology is perhaps something that should be resisted as we should remain cognizant of the fact that there may be a psychological component in many cases of organic aetiology and vice-versa.

In addition to the investigations performed, Schiavi et al. may have found it beneficial to further evaluate their subjects with normal and abnormal penile tumescence recordings. It would have been valuable to evaluate erectile function with RigiScan monitoring during audiovisual sexual stimulation. This would have enabled them to explore the relationship between abnormal nocturnal penile tumescence recordings and the more objective data on tumescence and rigidity obtained in an erotic (albeit still artificial) setting. The further addition of penile Doppler ultrasonography would have made their study yet more powerful. It may have then been possible for the investigators to confidently demonstrate that an abnormal nocturnal penile recording is related to impaired erectile function in another setting. In addition, a subclinical impairment in sexually non-dysfunctional men would be expected to be apparent in these other investigations. The ability to accurately identify patients with such

subclinical erectile impairment may have important therapeutic implications.

A recently published study has shed light on the relationship of lipid abnormalities to alterations of vascular reactivity [3]. Given the association of diabetes mellitus and abnormal lipid profiles it would be valuable to obtain such data for the study population. We suggest that a population of diabetic men with subclinical erectile impairment, or indeed mild erectile dysfunction, might benefit from a correction of lipid profiles were they found to be abnormal.

The relationship between glycaemic control and the development of diabetic complications was another subject addressed by Schiavi et al. Their finding that poor diabetic control is associated with diabetic complications, including erectile dysfunction, is in accordance with other published studies [4]. The important results of Reichard et al. [5] demonstrate that the development of neuropathy, retinopathy and nephropathy are retarded by intensified insulin treatment.

Taken together, the overall importance of recent findings is that it may be possible to identify diabetic men with subclinical erectile impairment and to prevent, if not to reverse, the progressive development of this often very distressing complication by: i) ensuring normoglycaemic diabetic control, ii) the correction of abnormal lipid profiles and, iii) attempting to halt the progression of diabetic neuropathy [6]. The adoption of such an approach may prove to be of major benefit not only to erectile performance but also to the other complications of diabetes.

Yours sincerely,

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Response from the authors

Dear Sir,

We appreciate the comments made by Miller et al. We agree that the occurrence of erectile problems is often neglected in the evaluation of diabetic men and that the tendency to view causation in dichotomous terms, i.e. organic vs. psychogenic, frequently leads to incorrect diagnostic conclusions and inappropriate interventions.

We believe that the monitoring of nocturnal tumescence in the sleep laboratory is a valuable procedure to assess degree of organic impairment in erectile capacity. However, as indicated in our article [1], interpretation of abnormal nocturnal penile tumescence findings should be made with caution. Abnormal nocturnal penile tumescence results need to be considered in the context of age-related normative information and should take into account i) emotional states such as clinical depression or excessive anxiety, ii) hypoactive sexual desire disorders, iii) hypogonadal conditions and, iv) sleep disorders [2]. We have found that the diabetic men had significantly higher prevalences of respiratory and periodic leg movement disturbances during sleep. Sleep-related respiratory abnormalities were associated with impaired sleep architecture and with diminished sexual arousal during wakefulness [3]. Independent assessment of sleep architecture is frequently neglected in the diagnostic use of nocturnal penile tumescence monitoring leading to inaccurate conclusions and contributing, in no small degree, to the controversy about the accuracy of this test.

Although little is known about the brain mechanisms that control sleep erection, there is evidence, as Miller et al. suggest,

that erotic and sleep-related erections may have different neuroendocrine substrates [3]. The evaluation of diabetic patients with Rigiscan monitoring during audiovisual sexual stimulation may have provided external validating evidence of erectile impairment in an erotic context. However, the diagnostic value of the visual sexual stimulation method has not been yet clearly established [4]. An important limiting factor is the difficulty that a substantial proportion of physically normal men, particularly those who are older, have in becoming aroused under laboratory conditions.

The value of nocturnal penile tumescence monitoring for the assessment of subclinical erectile impairment needs to be further evaluated. We fully agree with Miller et al. that, if the validity of these results is confirmed, it may become possible to identify diabetic patients at risk and to implement therapeutic strategies that may prevent or delay the clinical onset of erectile disorders.

Sincerely yours,

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