The Role of Nocturnal Penile Tumescence and Rigidity (NPTR) Monitoring in the Diagnosis of Psychogenic Erectile Dysfunction: A Review

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ABSTRACT

Introduction: Nocturnal penile tumescence and rigidity (NPTR) monitoring with RigiScan was considered one of the most reliable methods to differentiate psychogenic erectile dysfunction (pED) from organic ED. However, its reliability has been questioned because of some limitations in the practice.

Aim: To present contemporary views on the role of NPTR monitoring in the diagnosis of pED.

Method: We performed a comprehensive review of English-language literature on NPTR and pED by a PubMed search.

Main Outcome Measures: Studies were included if the mechanisms of pED and nocturnal erection and the practice of NPTR monitoring in ED were the main research contents.

Results: The pED results from not only psychosocial factors but also physiological changes containing central nervous abnormality. NPTR monitoring with RigiScan is still considered a useful method for the diagnosis of pED. A normal NPTR recording in a man with ED complaints probably suggests pED, whereas an abnormal recording may represent organic ED. Radial rigidity of no more than 60% is correlated well with axial rigidity, but, when it is more than 60%, the correlation between them is questioned. The consistency between NPTR and sex-stimulated erection is questionable, and the correlation of NPTR with different patient-reported outcome scoring systems is different. A normal NPTR recording in patients with ED does not necessarily mean pED, especially in patients with spinal cord injury. NPTR recordings can be influenced by depression, smoking, aging, negative dream content, and sleep disorders.

Conclusion: NPTR monitoring with the RigiScan is still considered a useful diagnostic tool for pED at the present stage. However, there are some disputes regarding the correlation between penile radial rigidity and axial rigidity and between NPTR and sex-related erection, as well as normative evaluation criteria for ED and the possibility of a false NPTR result, that need to be further studied. **Zou Z, Lin H, Zhang Y, et al. The Role of Nocturnal Penile Tumescence and Rigidity (NPTR) Monitoring in the Diagnosis of Psychogenic Erectile Dysfunction: A Review. Sex Med Rev 2019;7:442–454.**

Key Words: Penis; Erectile Dysfunction; Nocturnal Penile Tumescence and Rigidity; Diagnosis

INTRODUCTION

Erectile dysfunction (ED) is defined as the persistent or recurrent inability to attain and maintain an erection sufficient to perform sexual activity.[1] The cause of ED may be vasculogenic, neurogenic, anatomic, hormonal, drug-induced, or psychogenic. Although there is controversy regarding the differential diagnosis of different sexual dysfunctions,[2,3] the diagnostic practice between psychogenic and organic ED has been already included in the international guidelines and widely accepted.[4]

Psychogenic ED (pED) represents an erectile disorder associated with psychosocial health and has a negative impact on the quality of life of both sufferers and their partners. pED often

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occurs in young adults, accounting for 13−85.2% of patients under age 40 years with ED, whereas the incidence is about 40% in patients older than 40 years with ED.[5−8] With regard to all patients with ED, 45% of them have psychogenic problems.[9] Psychological factors play an important role in the development and maintenance of sexual dysfunction, and therefore psychological interventions are considered promising treatments for these disorders.[10] Although outcomes of psychological interventions alone for pED vary,[11,12] many studies have proved their effectiveness, such as group psychotherapy, in the treatment of pED, especially when they are used in combination with medical treatment.[10,13,14] Moreover, it was reported that 30−70% of patients with pED recovered even immediately after diagnosis of pED.[15,16] Formal cognitive-behavioral interventions by trained and experienced therapists are probably best used in patients with ED with a predominantly psychogenic cause, but such interventions are less likely to be beneficial in those with predominantly organic alterations.[17] Therefore the differentiation of pED from organic ED is necessary.

The diagnosis of pED is one of exclusion, and other etiologic disorders should be excluded first. In addition, a thorough psychosocial history is critical to identify risk factors for patients with suspected pED. Clinical features suggesting pED include sudden-onset, good-quality self-stimulated or spontaneous erections (eg, morning and nocturnal erections), previous psychological problems, or major life events. Conversely, gradual onset, lack of erections, and normal libido are more suggestive of an organic ED.[18] Nocturnal penile tumescence (NPT) is a physiological and spontaneous phenomenon, and NPT rigidity (NPTR) monitoring by the RigiScan device is considered an objective method for recording the spontaneous erections to differentiate psychogenic and organic ED. However, use of NPTR testing with RigiScan in routing practice is controversial. This review will provide current information and explore the role of NPTR monitoring in the diagnosis of pED.

METHODS

A PubMed search of English-language literature published before June 2018 was performed with the following search terms: "NPT," "NPTR," "nocturnal erection," "nocturnal tumescence," "nocturnal penile tumescence and rigidity," "RigiScan," "sleep-related erection," "psychological erectile dysfunction," and "psychogenic erectile dysfunction." Original articles and reviews about the mechanisms of pED and nocturnal penile erection and the practice of NPTR monitoring in ED were reviewed. Reference lists of relevant reviews were searched for potential eligible literature, which were also reviewed. Subsequently, we summarized the literature as the chapter of "psychological factors in pED," "physiological basis of pED," "nocturnal penile tumescence and rigidity," "NPTR monitoring using the RigiScan," and "audiovisual sexual stimulation (AVSS) and RigiScan monitoring."

Psychological Factors in pED

pED resulted predominantly or exclusively from psychological or interpersonal factors such as anxiety, depression, loss of self-esteem, previous traumatic sexual experiences, suspicions in sexual roles, physical disorders in spouses and lack of attraction, sexual myths or socioeconomical factors (eg, job stress).[19] These influence factors are usually classified as predisposing, precipitating, and maintaining factors, which are related to the development of pED.[20,21] It is realized that pED is the expression of a complex psychological discomfort associated with relational dynamics.[22,23] In addition, pED sometimes accompanies pediatric cancers[24] and many chronic diseases including gout,[25] irritable bowel syndrome,[26] and migraine[27] because of mental stresses of a cancer diagnosis and long-term treatment, depression, and anxiety.

ED with no sexual intercourse is a new concept defined as the subjective inability to have enough erection hardness and duration and thereby lacking enough confidence to attempt sexual intercourse, accounting for 7% of patients with ED.[28] Lack of confidence in erectile function (EF) and seeking sexual intercourse is considered the mainly cause.[29]

Physiological Basis of pED

Emerging evidence indicates that pED is organic and results from central nervous system alterations, which may inhibit the spinal erection center suprasacrally and not only be "psychogenic." With the help of structural and functional magnetic resonance imaging (MRI), the role of the central nervous system in pED is being revealed gradually (Table 1). It is proven that pED is associated with the alterations of cerebral activities and structures that are involved in sexual arousal and cognitive and emotional processes. Aberrant cortical morphometry and network organization,[35,36] microstructural alterations of cerebral white matter,[37] and gray matter atrophy in subcortical structures (eg, nucleus accumbens)[40] play a significant role in patients with pED. Recently, it was realized in a resting state that pED (diagnostic criteria included impaired self-reported erection, normal nocturnal erection, and negative emotion) might be related to the less-efficient connectivity in the prefrontal-amygdala pathway in the left hemisphere.[31] During visual erotic stimulation, activation of the left superior parietal lobe[39] and abnormal responses of brain networks including default-mode network and salience network, possibly play an inhibitory effect on EF in the patient group.[38] In addition, in the study by Sakamoto et al[41] of a putative rat model for post-traumatic stress disorder, the spinal reproductive center, a gastrin-releasing peptide system at the L3-L6 level, was proven to be involved in stress-related male sexual dysfunction.

In addition, pED is found to be associated with peripheral catecholamine. The serum norepinephrine level was higher in patients with pED when compared with healthy control subjects and patients with vasculogenic ED.[42] An elevated peripheral

Table 1. Summary of studies about central nervous alterations in patients with pED using magnetic resonance imaging

Reference	No. of patients	The diagnosis criteria of pED	Erectile function of pED	Duration of pED	Sexual stimulation*	Central nervous alterations in pED
Chen et al[30]	25 pED vs 26 healthy control subjects	1. Absence of organic ED; 2. Normal morning erections, and normal NPT rated by the RigiScan; 3. Other psychiatric disorders were excluded.	9.44 ± 5.12 (IIEF-5)	2.68 ± 2.1 y	No	1. Reduced connectivity strength in several regions of the left prefrontal and limbic cortex; 2. An aberrant hub distribution of the brain structural network.
Chen et al[31]	21 pED vs 24 healthy control subjects	1. Normal morning and nocturnal erections rated by the RigiScan; 2. Normal penile hemodynamics evaluated by DDU and ICI.	9.71 ± 5.71 (IIEF-5)	NA	No	1. Impaired connectivity in the left prefrontal-amygdala pathway; 2. Lower leftward asymmetry in the inferior frontal gyrus; 3. More hub regions and fewer pivotal connections; 4. The degree of the left amygdala was negatively correlated with the self-reported erection.
Wang et al[32]	27 pED vs 27 healthy control subjects	1. Absence of organic ED evaluated by DDU and RigiScan test; 2. Other psychiatric disorders were excluded.	31.38 ± 12.14 (IIEF-total)	More than 6 mo	No	1. The disrupted homogeneity within the right aINS; 2. Aberrant connection patterns between the right aINS and the right dorsolateral prefrontal cortex, as well as the right aINS and the right temporoparietal junction.
Jin et al[33]	26 pED vs 26 healthy control subjects	1. Absence of organic ED; 2. Normal morning erections and nocturnal erections rated by the RigiScan.	30.9 ± 8.7 (IIEF-total)	More than 6 mo	No	Lower baseline brain activity in the right anterior insula and the right orbitofrontal cortex.

(continued)

Table 1. Continued

Reference	No. of patients	The diagnosis criteria of pED	Erectile function of pED	Duration of pED	Sexual stimulation*	Central nervous alterations in pED
Chen et al[34]	24 pED vs 26 healthy control subjects	1. Absence of organic ED; 2. Normal morning erections, and normal NPT rated by the RigiScan; 3. Other psychiatric disorders were excluded.	9.08 ± 5.25 (IIEF-5)	2.75 ± 1.94 y	No	1. Brain networks exhibited higher small-worldness and more modules. 2. Nodal metrics were profoundly affected at frontoparietal network and prefrontal-limbic circuit. 3. The altered small-worldness and strength of right parahippocampal gyrus were related to the duration and IIEF-5 of pED.
Zhao et al[35,36]	40 pED vs 39 healthy control subjects	1. Absence of organic ED evaluated by DDU; 2. Normal NPT using RigiScan test; 3. Other psychiatric disorders were excluded.	34.82 ± 11.90 (IIEF-total)	More than 6 mo	No	1. A less optimal global topologic organization of structural cortical networks with reduced global and increased local efficiencies; 2. Decreased CTh in widespread cortical regions; 3. Abnormalities in interregional morphologic correlations.
Zhang et al[37]	27 pED vs 27 healthy control subjects	Absence of organic ED	13.56 ± 3.61 (IIEF-5); 2.70 ± 0.54 (EHS)	32.44 ± 27.43 mo	No	1. Multiple white matter regional alterations; 2. The alterations of the splenium of the cingulate cortex were correlated with symptom severity.
Cera et al[38]	16 pED vs 19 healthy control subjects	1. Absence of organic ED; 2. Normal morning erections, and normal NPT rated by the RigiScan; 3. Other psychiatric disorders were excluded.	14.5 ± 5.5 (IIEF-5)	9.2 ± 5.1 mo	Yes	1. Decreased connectivity values in the inferior parietal lobes, posterior cingulate cortex and medial prefrontal cortex; 2. Increased connectivity in the right insula and in the anterior cingulate cortex.

(continued)

Table 1. Continued

Reference	No. of patients	The diagnosis criteria of pED	Erectile function of pED	Duration of pED	Sexual stimulation*	Central nervous alterations in pED
Cera et al[39]	17 pED vs 19 healthy control subjects	1. Absence of organic ED; 2. Normal morning erections, and normal NPT rated by the RigiScan; 3. Other psychiatric disorders were excluded.	14.4 ± 5.4 (IIEF-5)	9.2 ± 2.1 mo	Yes	1. Larger activation in the left superior parietal lobe, ventromedial prefrontal cortex, and posterior cingulate cortex, but lower activation in the right middle insula and dorsal anterior cingulate cortex and hippocampus. 2. Larger activation in the left superior parietal lobe especially during the later stage of sexual response.
Cera et al[40]	17 pED vs 25 healthy control subjects	1. Absence of organic ED; 2. Normal morning and nocturnal erections rated by the RigiScan; 3. Other psychiatric disorders were excluded.	NA	NA	No	A significant gray matter atrophy of both left and right nucleus accumbens and left hypothalamus.

aINS = anterior insula; CTh = cortical thickness; DDU = duplex doppler ultrasonography; EHS = erection hardness score; ICI = intracavernous injection; IIEF = International Index of Erectile Function; NA = not available; NPT = nocturnal penile tumescence; pED = psychogenic erectile dysfunction.
*Whether patients were sexually stimulated, when imaging examination was performed.

Table 2. Normal criteria for potential normal erectile function using the RigiScan device to differentiate psychogenic form organic ED

Ref.	No. of subjects	Sessions	Methods	Referenced method	Criteria	Diagnostic values
Wang et al[66]	1,078 ED patients	1078	AVSS + PDE5I + RigiScan test	A battery of tests and evaluations	1. Average event rigidity of tip was 43.5%. 2. Duration of erectile episodes over 60% was 8.75 min. 3. Average event rigidity of base was 50.5%.	Sensitivity was 93.8, 92.6, and 93.8%, respectively. Specificity was 84.0%, 84.8%, and 81.8%, respectively.
Elhanbly, et al[67]	416 ED patients	639	NPTR monitoring	NPTR monitoring curves	1. For the single best event, RAU and TAU at the tip were 23 and 11, and at the base were 23 and 13. 2. For the total night, RAU and TAU at the tip were 57 and 31, and at the base were 74 and 41. 3. Normalized units, standardized units, summated units and R/T ratio	1. Sensitivity, specificity, and accuracy were 77.7-81.2%, 53.1-65.3%, and 67.8-73.7% for the single best event. 2. Sensitivity, specificity, and accuracy were 75.1-84.6%, 54.6-71.4%, and 66.7-75.4%, respectively. 3. When using the newly computed units, the highest diagnostic accuracy did not exceed 75.9%.
Hatzichristou et al[68]	12 healthy young men	108	NPTR monitoring	No	For the best event: 1. Tip penile rigidity ≥60% and duration ≥10 min; 2. Tip penile rigidity ≥60% and duration ≥15 min; 3. Tip penile rigidity ≥70% and duration ≥10 min; 4. Tip penile rigidity ≥70% and duration ≥15 min.	1. Accuracy was 83.33—100%. 2. Accuracy was 83.33—100%. 3. Accuracy was 66.66—83.33%. 4. Accuracy was 50—83.33%.
Karadeniz et al[55]	64 ED patients	NA	NPTR monitoring	A multidisciplinary approach	Penile rigidity ≥70% and duration ≥10 min.	Sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were 81%, 82%, 89%, 69%, and 81%.

(continued)

Table 2. Continued

Ref.	No. of subjects	Sessions	Methods	Referenced method	Criteria	Diagnostic values
Benet et al[69]	40 ED patients	80	NPTR monitoring	A battery of tests and evaluations	1. The single best event with 70% tip rigidity for 5 min. 2. For the single best event, RAU and TAU at the tip were 9.5 and 6.5, and at the base were 11.5 and 8.0. 3. For 2 nights, RAU and TAU at the tip were 9.5 and 8.5, and at the base were 18.5 and 9.5.	1. Sensitivity, specificity, accuracy were 85%, 100%, and 92.5%. 2. Sensitivity, specificity, and accuracy were 85—90%, 85—95%, and 87.5—90 % for the single best event. 3. Sensitivity, specificity, and accuracy were 85—90%, 90—95%, and 87.5—92.5%.
Licht et al[59]	28 men	56	NPTR monitoring	Clinical observation	For a single recorded event: 1. Base rigidity $\geq$55%; 2. Tip rigidity $\geq$70%; 3. Base rigidity $\geq$70%.	1. Sensitivity, specificity, and accuracy were 85%, 91%, and 88.1%. 2. Sensitivity, specificity, and accuracy were 38.5%, 93.9%, and 69.5%. 3. Sensitivity, specificity, and accuracy were 50%, 97%, and 76.3%.
Ogrinc et al[70]	153 ED patients	752	ICI + RigiScan test	Clinical observation	1. An erectile episode of tip or base penile rigidity $\geq$60% and 10 min in duration; 2. An erectile episode of tip or base penile rigidity $\geq$70% and 10 min in duration.	1. Sensitivity, specificity, accuracy were 70.8%, 85%, and 80.3%. 2. Sensitivity, specificity, accuracy were 53.8%, 92.9%, and 80.1%.

AVSS = audiovisual sexual stimulation; ICI = intracavernous injection; NPTR = nocturnal penile tumescence and rigidity; PDE5I = phosphodiesterase 5 inhibitor.

catecholamine level may increase penile smooth muscle tonus and then inhibit smooth muscle relaxation.

Nocturnal Penile Tumescence and Rigidity

Halverson et al[43] first recorded spontaneous penile erection in infants during sleeping in the scientific literature in 1940, and Ohlmeyer et al[44] and van Driel [45] recorded the phenomenon in healthy men in 1944. Afterward, Fisher et al[46] reported that spontaneous erections were usually in conjunction with the rapid eye movement phase. Normal NPTR includes 3−6 tumescence periods, with at least 1 erectile event having tip rigidity >60% lasting an average of 10−15 minutes during 8 hours of monitoring.[47] NPT are present in healthy men throughout their whole life and represent an intrinsic mechanism to protect the morphologic and dynamic integrity of the corpora cavernosa by regulating oxygen-required biologic processes.[48] The definite regulation mechanisms of NPT are not clear, but there is some evidence indicating that NPT is associated with neurovascular mechanisms and hormonal control.[47] In addition, spinal regulation has been proven important for nocturnal erectile activity. Schmid et al[49] found that nocturnal erections of normal quality required preservation of thoracolumbar and sacral neuronal control, as well as partially intact connections between the spinal erection centers and brain areas responsible for sexual arousal. Suh et al[50] found that the isolated cervical cord was more critical than the isolated thoracic cord in maintaining NPT.

Normal NPTR indicates that vascular and neural supplies of the penis, as well as the penile structures, are intact. In the study by Yilmaz et al[51] with the immunohistochemical method, it was found that the content of penile smooth muscle cells (pSMC) in patients with ED and with normal NPTR was equal to the content of pSMC in men with normal EF but was significantly more than the content of pSMC in patients with ED and with abnormal NPTR. Since its introduction in 1970,[52] the measurement of NPTR has been used as a diagnostic approach in the evaluation of ED, especially in the differentiation of causes. The general belief is that a normal NPTR recording in a man with ED complaints may suggest pED, whereas an abnormal pattern recording is indicative of organic ED.

NPTR Monitoring Using the RigiScan

Clinically, NPTR measurement is mainly conducted by 7 methods and their modifications, including sleep laboratory testing, the mercury strain gauge, the stamp test, the erectometer, the snap gauge, nocturnal electrobioimpedance volumetric assessment, and the RigiScan.[53] The RigiScan device, first introduced by Bradley et al[54] in 1985, is now the most widely accepted method . The instrument is designed to measure both penile circumference and radial rigidity continuously and quantitatively and is used in real-time or ambulatory mode.

The advantages of RigiScan include (i) non-invasiveness; (ii) a synthetic measurement device that is capable of simultaneously recording sleep-related erection duration, tumescence, and rigidity; (iii) the most objective recording of penile erectile activity; (iv) real-time and continuous recording, providing descriptive details of erectile performance such as duration and degree of rigidity, which cannot be offered by other devices; and (v) home use that can avoid patient anxiety caused by hospital circumstance and manipulations.

In the past, monitoring NPTR with the RigiScan device was considered one of the most reliable tools to differentiate pED from organic cases on the basis of the assumption that psychological factors do not interfere with nocturnal erectile activity. Karadeniz et al,[55] studying 64 randomized patients with a complaint of ED, reported that sensitivity, specificity, positive predictive value (abnormal NPTR meant positive results), negative predictive value, and accuracy of NPTR monitoring by the RigiScan in differentiating organic ED from pED with respect to a multidisciplinary diagnostic approach using sophisticated techniques were 81%, 82%, 89%, 69%, and 81%, respectively. However, the reliability of NPTR measurement using the RigiScan has been questioned because some limitations have been noted as follows.

Axial Rigidity Measurement

Axial rigidity is the best physical parameter that determines the capacity of the erect penis to resist vaginal compressive forces during vaginal penetration and pelvic thrusting. However, the RigiScan device only measures radial rigidity but not axial rigidity. Axial rigidity of the penis is dependent on intracavernosal pressure, cavernosal erectile tissue properties, and penile geometry, whereas radial rigidity is dependent on intracavernosal pressure and tunical surface wall tension. Intracavernosal pressure is a common dependency shared by axial and radial rigidity, but other physical determinants are unique for each other. Although the 2 types of rigidity are different parameters, a good correlation between them has been widely demonstrated. Udelson et al,[56] calculating 161 data about axial buckling force and RigiScan radial rigidity with correlation coefficient of 0.58, concluded that a correlation existed between axial and radial rigidity in a large population. Their result was similar to other relevant studies, in which the correlation coefficients between axial and radial rigidity values were 0.59,[57] 0.78,[58] 0.66,[59] and 0.70,[60] with 25, 29, 59 and 18 data points, respectively. Whether radial rigidity >60% is still well correlated with axial rigidity is questioned. It was found by some researchers that when tip and base rigidity were >60%, the correlation was poor because the 2 parameters had different dependent variables.[57] However, Ku et al[61] evaluated the relationship between radial rigidity and resistance index, which was only influenced by intracorporeal pressure, and believed that radial rigidity might have a good correlation with the intracorporeal pressure in circumstances when it exceeded 60% and could reflect erectile capacity efficiently. Udelson et al,[56] however, considered that the axial buckling measurement was not predicted by the RigiScan radial measurement in an individual and that this was no better than flipping a coin.

The Extent to Which NPTR Can Reflect Sex-Related Erection

The extent to which NPTR can reflect erectile performance during sexual intercourse is still unknown because the physiological mechanisms are not completely clear and are even considered different. However, much of literature advocated that NPTR revealed resident EF in some patients with ED caused by trauma or surgery. The study by Peng et al[62] of 31 patients with ED caused by pelvic fracture urethral disruption suggested that NPTR recordings reflected potential EF in patients with traumatic ED and with a 76.9% pharmacologic response rate when tip rigidity was >40%, but only 22.2% when tip rigidity was <20%. Bannowsky et al[63] found that NPTR recordings during the acute phase after nerve-sparing radical prostatectomy could show residual EF as early as the first night after catheter removal.

The mechanisms of NPTR and sex-stimulated erection are not completely the same, and the consistency between them is questionable. Patient-reported outcome scoring systems for EF can subjectively reflect the patient's EF during sexual intercourse. The correlations of NPTR with different patient-reported outcome scoring systems are different. Nocturnal maximal penile circumferential change is correlated well with the erection hardness score. Maximal penile circumferential change >20 mm can differentiate a patient with an erection hardness score ≥3 from that ≤2 ($P = .013$).[64] However, International Index of Erectile Function erectile domain scores and NPTR measurements are weakly associated, and the clinical utility of NPTR to predict the former is limited.[65]

Normative Evaluation Criteria

Normative evaluation criteria of the RigiScan test for NPTR are variable in different studies (Table 2). The best erectile event has been suggested as a parameter to predict the diagnosis of ED. Presence of ≥1 erectile event with >70% penile tip rigidity and >10 minutes in duration was accepted as the most common criterion to define normal EF. Hatzichristou et al[68] suggested that this criterion was too strict and NPTR with at least 1 erectile episode of tip penile rigidity >60% and 10 minutes in duration, detected by RigiScan, was probably associated with normal EF. With regard to radial rigidity measured by the RigiScan system, values <30−40% correspond to an erection inadequate for vaginal penetration, above 60−70% indicate unbuckleable penis, and the middle values provide enough rigidity so that vaginal penetration could be achieved with assistance.[55] At least 2 consecutive nights of recording, suggested by Hatzichristou et al[68] and Levine and Lenting[71] were necessary to assess NPTR recordings considering possible patient discomfort from wearing the device and its interference with normal sleep (the so-called "first night effect"). Similarly, Greenstein et al[72] suggested that patients with normal erections during the first night could be saved from second-night testing, whereas consecutive night recording should be reserved for those with abnormal NPTR during the first night. These criteria are widely accepted in current clinical practice.

Rigidity activity units (RAU) and tumescence activity units (TAU) are integrated parameters introduced in 1994. RAU represent the product of elapsed time during a detectable erectile event multiplied by the corresponding rigidity, with rigidity expressed as a fraction between 0 and 1, and resultant values ranging from 0 to 120, whereas TAU represent the product of elapsed event time multiplied by the increase in tumescence over baseline tumescence with resulting values ranging from 0 to 120.[69] Benet et al[69] studied 80 sessions of 40 patients and obtained cutoff values (the values of RAU and TAU at the base were 18.5 and 9.5, respectively; at the tip were 9.5 and 8.5, respectively) with good diagnostic accuracy of 87.5−92.5%, but the outcomes were not repeated by the study of Elhanbly et al[67] of a larger population. In the latter study of 639 RigiScan night records of 416 patients with ED, it was demonstrated that TAU cutoff values of 11 and 13 at the tip and base, and RAU of 23 at both sites had the highest diagnostic accuracy of 67.8−73.7%.

NPTR monitoring is a reliable method for discriminating organic ED from pED in most patients, but the parameters used are not disease-specific. In addition, patients with ED and with normal NPTR recordings do not necessarily mean pED, especially in patients with spinal cord injury-related ED. In the study by Huang et al[3] of 191 patients with ED, the brachial artery flow−mediated dilation values in the pED group were significantly lower than in those in the organic ED group ($P = .020$), indicating that underlying endothelial dysfunction could also present in pED with normal NPTR. Perhaps it is one of the reasons why the combination of psychological intervention and medical treatment is superior to psychological intervention alone in the treatment of pED. When evaluating NPTR in patients with spinal cord injury-related ED, normal recordings should be considered cautiously because nocturnal erectile activity is present in some patients with spinal cord injuries with significantly impaired volitional EF.[50] Young men with ED caused by multiple sclerosis may also have normal nocturnal erectile activity.[73]

False Abnormal Results of NPTR Detected by the RigiScan

Abnormal NPTR recordings during 2 consecutive nights commonly indicate organic erectile disorder. However, depression, negative dream content, sleep disorders, and smoking sometimes produce false abnormal results.[74−76] These influence factors should be noticed when explaining abnormal recordings of NPTR. In addition, Yaman et al,[77] studying 455 patients (ages 20−71 years) with initial complaints of ED, reported that aging negatively influenced the quality of NPTR, especially after age 50 years, and suggested that age was required to be taken into account in the diagnostic interpretation of NPTR testing. Non-organic factors affecting NPTR values were summarized in Table 3.

AVSS and RigiScan Monitoring

The erectile physiology during AVSS is similar to that during sexual intercourse but different from nocturnal erectile activity.

Table 3. Effects of non-organic factors on the NPTR result

Non-organic factors	Effects	Possible mechanisms	References
Smoking	Reduction of NPTR values	Dysfunction of penile veno-occlusive mechanisms	Elhanbly et al[78]
Depression	Reduction of NPTR values	A neurophysiological association with depression	Nofzinger et al,[79] Steiger et al,[80] Thase et al[81]
Aging	NPTR values decreased with age reduced, especially after 50 y.	1. Reflect normal physiological changes or pathologic conditions. 2. Aging was associated with decreased sleep efficiency.	Yaman,[77] Ware,[82] Schiavi,[83] Reynolds,[84] Karacan[85]
Sleeping	Poor quality of sleeping adversely affects NPT.	Disturbed REM sleep	Hirshkowitz and Schmidt,[86] Fisher et al[46]
Medications	Adversely affect NPT	1. Antiandrogens reduce testosterone. 2. Tricyclic antidepressants suppress REM sleep. 3. Some antihypertensive drugs can adversely affect EF and lower testosterone.	Hirshkowitz and Schmidt,[86] Rosen et al,[87] Wincze et al[88]
Trazodone	Increase NPT duration	α-Adrenoceptor blocking properties to interference with the sympathetic control of penile detumescence.	Saenz de Tejada et al[89]
Sleep apnea	NPT decrement	Decreased androgen or recurrent episodic hypoxia	Hirshkowitz et al,[90] Schmidt and Wise[91]
Periodic limb movement disorder	NPT decrement	Unknown	Schmidt and Wise[91]

EF = erectile function; NPT = nocturnal penile tumescence; NPTR = nocturnal penile tumescence and rigidity; REM = rapid eye movement.

RigiScan monitoring with AVSS was recently conducted for the diagnosis of pED. RigiScan-monitored AVSS can confirm the clinical diagnosis of pED with 71% sensitivity and 96% specificity.[92] In the study by Wang et al[66] of 1,169 patients with ED, AVSS-RigiScan testing with administration of phosphodiesterase-5 inhibitor discriminated pED from organic ED with a sensitivity and specificity of 87.7% and 93.4%, respectively. It was suggested that basal rigidity >60% sustained for ≥8.75 minutes, average event rigidity of tip ≥43.5%, and base ≥50.5% would be the new normative Chinese evaluation criteria for sex-related tumescence and rigidity of the penis.[66] In addition, RigiScan-monitored AVSS is an expeditious diagnostic method and is less time-consuming than NPTR monitoring. However, AVSS-RigiScan testing also has some limitations, including susceptibility to psychological factors (eg, erotic excitement inhibition[93]), which are possible bias factors and cannot be avoided completely in all clinical manipulations.

CONCLUSION

Although the organic basis of pED, mainly regarding the central nervous alterations, has been gradually discovered with the help of functional and structural MRI, neither specific biomarkers nor biologic characteristics are available in clinical practice for the diagnosis of pED. Specific examination methods including MRI for the purpose still require further exploration. The diagnostic approach for pED should be multidisciplinary and individualized. A comprehensive assessment of EF is the goal, and RigiScan for NPTR monitoring is still considered a useful diagnostic tool for pED at the present stage. In the future, the correlation between NPTR and spontaneously sex-related EF needs to be further explored. The question about to what extent of NPTR reflects normal EF should be answered. How to combine NPTR with other novel modalities, such as endothelial function tests, to improve the diagnostic accuracy of pED and find underlying organic alterations, still needs to be studied. Developing a novel device, which can directly and accurately measure the axial rigidity, will further enable NPTR to live up to its potential as a diagnostic approach for pED. With normal NPTR at least indicating no significant penile structure abnormality, the aims of the research on pED may be mainly focused on the upstream of erectile-regulating pathway.

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