

USE OF NOCTURNAL PENILE TUMESCENCE AND RIGIDITY IN THE EVALUATION OF MALE ERECTILE DYSFUNCTION

Laurence A. Levine, MD, FACS, and Eric L. Lenting, MD

As an objective, noninvasive measure of erectile activity, nocturnal penile tumescence (NPT) has evolved into what many in the field of erectile dysfunction consider the "gold standard" study to differentiate between psychogenic and organic impotence. The increased understanding of the physiology of normal erectile function as well as insights into the cause of impotence have enhanced the benefits of NPT monitoring. But the evaluation of the impotent male remains a difficult task which cannot be made by any single test. For the practicing physician, NPT remains one of the fundamental tools for the evaluation and consequently appropriate management of patients. It has also become an essential research tool for the objective assessment of various pharmacologic therapies and the evaluation of technologic advances in the analysis of the impotent male.

Nocturnal penile tumescence was first described in infants by Haiverson in 1940.²¹ In 1944, Ohlmever and associates²² characterized a cycle of sleep erections in men 20 to 40 years old. Aserinsky and Kleitman²³ first described rapid eye movement (REM) sleep in 1953 and later recognized that nocturnal erections seemed to correspond with REM periods during normal sleep.²⁴ It is on the shoulders of the

work of Karacan, Fisher²⁵⁻²⁸⁻³⁴ and their associates that NPT monitoring has become a valuable resource in the assessment of the male with erectile dysfunction. Fisher and coworkers²⁵ reported in 1965 that in the normal male, three to five erections occur each night and account for up to 40% of sleep time. In 1975, Karacan and associates^{31,34} reported that healthy males between the ages of 3 and 79 years had NPT during normal sleep and that the amount of NPT was age related.

Karacan²⁵ further hypothesized that NPT testing might provide a useful method for differentiating between organic and psychogenic impotence. The basic assumption is that the relevant psychologic factors which may inhibit a sexually induced erection while awake would be inoperative during sleep. Therefore, in the impotent male without organic impotence, NPT would be present during sleep. In men with impotence due to neurologic or vascular factors, however, the mechanisms responsible for the erectile dysfunction would remain operational during sleep and NPT would be absent or diminished, thereby providing an objective measure to discriminate between these two groups of patients.

A second assumption made in NPT evaluation is that the same physiologic mechanism is

From the Department of Urology, Rush Medical College, and Male Sexual Function and Fertility Program (LAL), Rush-Presbyterian-St. Luke's Medical Center (ELL), Chicago, Illinois

responsible for both a nocturnal penile erection and a sexually stimulated erection. Although the validity of this assumption has been questioned, it has been suggested that the physiologic mechanisms responsible for the development of nocturnal erections are similar to those operative during sexual stimulation.¹¹ On the other hand, other groups caution that NPT may not always be a true indication of erectile potential in erotic and sexual circumstances.

NPT AND NPTR TESTING

Formal NPT testing, as it was initially introduced and still practiced in many places today, is performed in a sleep laboratory and includes sleep monitoring by electroencephalogram (EEG), electro-oculogram (EOG), or electromyogram (EMG), penile circumference measurements, and repeated measurements of axial rigidity at or near the time of maximum tumescence.

Before the value of routine penile rigidity testing was recognized, it was arbitrarily determined that a change in penile circumference of 20 mm represented a full erection and that a change of 16 mm in circumference or 80% of a full erection would be equivalent to the penile rigidity necessary for intromission.¹² Wein and associates¹³ demonstrated that significant changes in penile circumference occurred in 23% of normal control patients without sufficient increases in penile rigidity for vaginal penetration. Furthermore, recent studies by Earls and associates¹⁴ demonstrated the value of direct rigidity testing. They found a significant delay between adequate rigidity based on NPT recorded changes in circumference and the subject's perception of erectile sufficiency for intromission during visual sexual stimulation.

Due to large interindividual differences in the increase of circumference associated with full erections and the recognition that maximal increases in shaft circumference did not indicate adequate rigidity, direct measurements were needed to determine the stiffness of an erection. This involved waking the patient when an erection exceeded 80% of a full erection to measure rigidity as a function of the buckling pressure of the penis with an externally applied device. Karacan and co-workers¹⁵ found that a buckling pressure of 100 mm Hg would be adequate for intromission and that a buckling pressure of less than 60 mm Hg would be inadequate for vaginal penetration. Buckling force testing with standard NPT measurement as advocated to assess rigidity is lim-

ited because this technique occurs as a single isolated measurement of rigidity, which is subject to observer error and process of detumescence. In addition, although several criteria for adequate rigidity have been suggested, none have been universally accepted and they remain difficult to test scientifically as the concept of adequate rigidity pertains to any given subject's erection with a particular partner and situation (i.e., vaginal size and lubrication, partner receptivity, etc.). Overall NPT with intermittent rigidity testing is costly, time-consuming, and unnatural, thereby increasing the possibility of anxiety interfering with results.

Consequently, other methods to evaluate NPT have been developed. In 1980, Barry and associates¹⁶ developed a method of using stamps placed around the base of the penis to measure tumescent activity during sleep. Significant problems associated with this test include a lack of standardization, false-negative results due to slippage, and false-positive results due to incidental tearing of the stamps without erection.¹⁷ These problems led to the development of the Snap Gauge Band (Dacomed Corporation, Minneapolis, MN) in 1982 which consists of three preset snap-release fasteners with release force constants of 8, 12, and 16 ounces. This test was further modified to improve its reliability by replacing the fasteners with a serial arrangement of three plastic elements designed to break at 10, 15, and 20 ounces.¹⁸ The Snap Gauge Band was shown to correlate well with NPT testing in several studies.^{11,19} Although each of these methods can be used as a screening tool for NPT, none provide information regarding details of erectile performance such as the frequency, duration, or degree of rigidity of erectile events.

Ultimately, concerns over accurate rigidity monitoring, increased cost of a sleep laboratory assessment, and disruption of normal sleep led to the development of small portable monitors which could measure the rigidity of the erect penis continuously as well as record the number and duration of tumescent episodes.

In 1985, Bradley and Timm²⁰ described the Rigiscan monitoring device (Dacomed Corporation, Minneapolis, MN) used by patients at home to provide continuous recording of penile tumescence and rigidity (NPTR). The Rigiscan consists of two major components: (1) An NPTR data logging unit and (2) a micro-computer and printer to process and report the recorded data (Fig 1). The logging unit is strapped to the patient's thigh and consists of



Figure 1. Rigiscan ambulatory penile tumescence and rigidity monitoring device.

two loops placed around the base and tip of the penis proximal to the coronal sulcus. At 15-second intervals, the circumference of the penis is measured and compared with the subject's baseline measurement. The device also performs measurements of penile rigidity by applying a force of 2.8N to each loop every 3 minutes. When an increase of greater than 10 mm in circumference is detected in the base loop, rigidity measures are increased to every 30 seconds. Once the change in the circumference is less than 10 mm, the device returns to taking rigidity measurements every 3 minutes. The Rigiscan can collect data for three 10-hour monitoring sessions, during which the rigidity, tumescence, and duration of each event are recorded. The data can then be downloaded from the ambulatory logging unit into the microprocessor and the results printed in graphic display providing a composite pattern of rigidity and tumescence (Fig. 2).

The Rigiscan expresses penile rigidity as a function of displacement when the loop is tightened around the penis. A rigidity of 100% represents no linear displacement and for each 0.5 mm of loop shortening the rigidity measure is reduced by 2%. The device therefore defines rigidity in terms of penile stiffness as determined by cross-sectional response to radial compression. Previous measures of penile rigidity were expressed as measures of axial rigidity. Froneb and associates¹⁰ compared measurements of axial and radial rigidity at constant corporal body pressures. They found that these measures of axial and radial rigidity were functionally related and that these measures correlated with intracavernous pressure, which is generally regarded as the physiologic event responsible for penile rigidity.

A recent version of Rigiscan Summary Analysis software is described by Levine and Carroll¹¹ in a study of normal potent men eval-

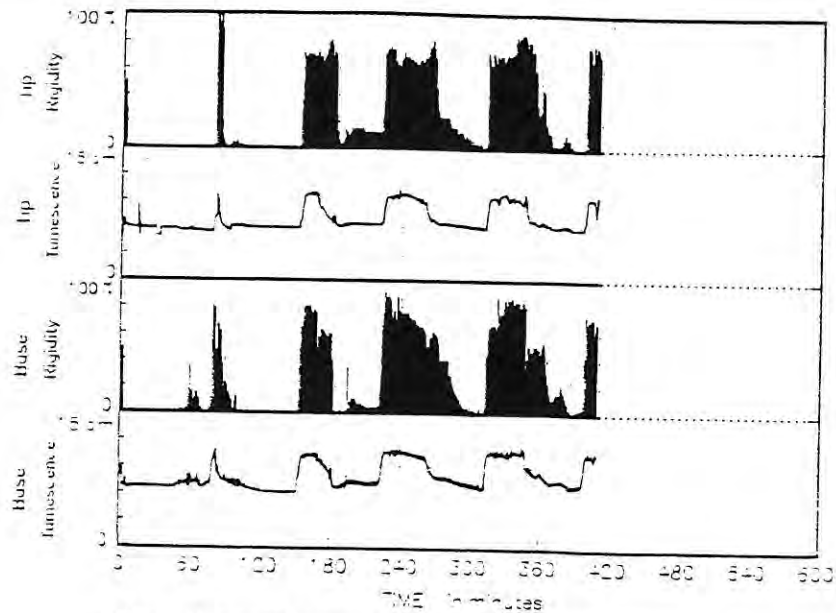


Figure 2. Rigiscan graphic printout of a 40-year-old potent man.

uated with NPTR. The software is used to scan the graphic data, and convert and display rigidity and tumescence information in numeric form to provide a quantitative analysis of the data. The software recognizes erectile activity as an event if there has been a 20% increase in the base loop circumference persisting for at least 3 minutes. Shorter duration erectile events (less than 3 minutes) would be displayed on the graphic printout but would not be counted by the software. This version (3.00) describes erectile events in a somewhat different manner than the previously released versions and contains additional statistical features not previously available. Summary statistics provided by the software include the number of detected events, event durations (cumulative time that tumescence was greater than 20% of baseline tumescence), average tumescence and rigidity readings during events, and integrated time intensity area measures of tumescence (tumescence activity units, TAU) and rigidity (rigidity activity units, RAU).

These two units of measurement, RAU and TAU, were developed to facilitate the interpretation of the time dependent nature of rigidity and tumescence. RAU represents the product of the minutes spent at a given rigidity level and the rigidity level expressed in decimal form. This value is calculated on a point-by-point basis and summarized for the entire erectile event. Similarly, TAU represents the time of

duration of an erectile event multiplied by the percentage increase of circumference (expressed as a decimal) over the estimated baseline tumescence. RAU and TAU for both tip and base measurements are calculated and evaluated separately. These summary parameters can easily be compared with normal cumulative distributions (Fig. 3).

LIMITATIONS OF NPT

NPTR is a useful, noninvasive, and relatively inexpensive objective measure of erectile activity (i.e., erectile behavior over time) as well as erectile capacity (i.e., ability to have a sufficient erection). The usefulness of NPT measurement, however, has been questioned by various authors. Wasserman and associates³⁷ questioned the basic assumption of NPT. Although they agreed that NPT monitoring is a useful aid in differentiating organic from psychogenic impotence, they stressed that it has never been validated in patients shown to have psychogenic or organic impotence independent of the NPT measurements themselves. Furthermore, the value of NPTR in the evaluation of the impotent male has yet to be clearly defined. By definition, the goal of NPTR is to monitor nocturnal erectile activity, but it has not yet been proven that nocturnal erections are the same as sexually induced erections. Likewise,

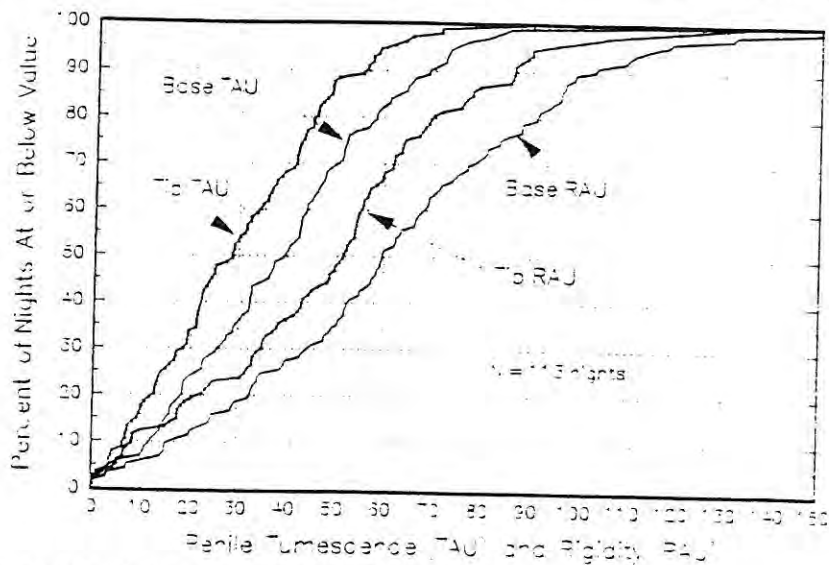


Figure 3. Cumulative distribution of penile tumescence activity (TAU) and rigidity activity units (RAU) from 14 potent men. Useful for comparison of NPTR results from impotent subjects. (From Lavigne LA, Carrion RA: Nocturnal penile tumescence and rigidity in men without complaints of erectile dysfunction using a new quantitative analysis software. *J Urol* 152:1103, 1994; with permission.)

the presence of nocturnal erections has not been proven to clearly indicate the capacity to have an awake erection which is sufficiently rigid for vaginal penetration.

Other concerns are that psychologic factors may in fact affect NPT in the patient with psychogenic impotence. Previous studies by Karacan^{22,33} have shown that 15% to 20% of patients with no identifiable evidence of organic impotence have abnormal NPT results. He suggested that subtle, undetectable physiologic factors may be operating in these patients. He also found that NPT was impaired in normal men with dreams of anxiety content.²⁹ Fisher²⁵ also noted impaired NPT in men having dreams containing anxiety, aggression, and other negative content. Depression has also been shown to adversely affect NPT. Roose and associates²⁹ and Thase and associates^{33,34} have reported on patients with major depression exhibiting a reversible loss of NPT which was restored when the depression was successfully treated. Jovanovic²³ reported a decreased number of tumescence episodes, total tumescence time, and decreased quality of erections on the first night of sleep laboratory testing, giving rise to the concept of a "first night effect."²³ Although wearing the NPT device is unnatural and could presumably interfere with normal sleep resulting in false NPT readings, two recent studies of normal, healthy volun-

teers did not reveal a first night effect or user complaints due to discomfort when NPTR was measured with the Rigiscan device.^{43,44}

Another drawback of these devices is the inability to assess the adequacy of sleep. As described by Pressman and co-workers,^{35,36} conditions such as sleep apnea, periodic leg movements, and nocturnal myoclonus can negatively affect NPT. A diminished NPT measurement can be directly caused by these sleep disorders and is not indicative of abnormal erectile function. Bradley⁷ has suggested that disturbed sleep impairs the appearance of a spontaneous erectile event. On the other hand, Schiavi²⁰ has demonstrated that a full erection may not occur in normal potent males despite a normal sleep pattern.

INTERPRETING NPTR RESULTS

Normal patterns of tumescence and rigidity as measured by the Rigiscan have evolved over the past 10 years. The first and most regularly used normal criteria were provided by the Dacomed Corporation based on over 500 NPTR studies performed at the Uro-Center of San Diego.⁴⁵ These include a change in circumference of at least 3 cm at the base of the penis and at least 2 cm at the tip of the penis. A rigidity measurement of greater than 70% repre-

sents a nonbuckling erection and a rigidity of less than 40% represents a flaccid penis. Measures between 40% and 70% represent varying degrees of penile stiffness. The number of erections considered normal is 3 to 6 per 3-hour session and lasting an average of 10 to 15 minutes.

For most physicians using the Rigiscan, interpretation of data is usually done as a visual review of the graphic printout. Kaneko and Bradley²⁷ presented a schema for further identifying abnormal Rigiscan results. They recognized several new NPTR patterns associated with impotent men. These include (1) dissociation of rigidity between the tip and base of the penis, (2) uncoupling between rigidity and tumescence, (3) shortened episodes of rigidity, (4) low amplitude rigidity, and (5) no episodes of rigidity and tumescence. This approach allows pattern recognition, which contributes to the visual gestalt approach to Rigiscan graphic analysis but does not provide useful baseline data obtained from normal potent controls.

In an effort to make Rigiscan analysis more objective, Kirkeby and associates²⁸ developed a computerized program for Rigiscan data analysis from 15 healthy subjects and compared the results with both the Dacomed criteria and the findings of Kaneko and Bradley. They observed that most of the nocturnal erections fluctuated in rigidity and that a substantial number of erections only sporadically obtained full rigidity (i.e., greater than 70%). At least half of the total number of erections recorded were considered insufficient for vaginal penetration based on visual analysis. In another study, Kirkeby and co-workers²⁹ noted that nocturnal erectile rigidity was normal in 11 of 26 patients with a well-established neurologic disorder known to affect erectile function (i.e., multiple sclerosis). They recommended that NPTR be used with the following reservations: absence of sufficient rigidity for vaginal intromission during nocturnal erections is found frequently in normal males, even if performed for several sessions, the presence of NPTR sufficient for vaginal intromission proves waking erectile capability only in the absence of neurological disorders.

In 1989, Burris and associates⁷ developed a quantitative rather than qualitative (visual) approach to Rigiscan data analysis in 47 normal potent men. They used area-under-the-curve (AUC) as a integrated measure of erectile amplitude and duration in hopes of reducing overinterpretation of single peaks as occur

with maximum rigidity. Using this approach, they found a highly reproducible method to quantify tumescence and rigidity. There was also significant correlations found between tumescence and rigidity ($P < .001$) and between tip and base measurements ($P < .001$).

In a 1994 study of normal subjects, Levine and Carroll³⁰ examined 44 potent subjects in an attempt to develop additional normative NPTR data as well as to determine the level of variability that could be expected in this population. They found that, like Burris and colleagues,⁷ although there was a high degree of uniformity in the results for the study population as a whole, regardless of subject age, there was considerable variability in individual responses during the three nights of monitoring (Fig. 4). Six of the 44 men displayed little or no tip rigidity on at least one of the 3 nights of monitoring, demonstrating that at least 2 nights are needed to characterize a subject adequately. They concluded, however, that evidence of significant erectile activity during a single night may be sufficient to demonstrate the potential for normal functioning.

They also found no simple criteria for a normal Rigiscan evaluation such as minimum number of events, duration, or percent rigidity as has been previously described. Rather, a cumulative distribution of the two new time-intensity measures of tumescence (TAU) and rigidity (RAU) were developed into a nomogram format to permit rapid comparison of NPTR findings (Fig. 3). The high correlation in the Levine and Carroll study supports similar findings for the AUC data for tumescence and rigidity for the normal populations of men studied. Furthermore, they provide an indication of both the reproducibility of these parameters as measures of erectile function and the consistency of the relationship between tumescence and rigidity in normal men.

The approach that seems most helpful in evaluating the NPTR of an impotent man is to determine the percentile rank of the tip RAU for the night of NPTR monitoring with the highest Rigiscan measures. For the three examples described below the graphic printout, the nomogram and the highest tip and base RAU and TAU data are shown.

Case 1 (Fig. 5) presents the NPTR results for a potent 62-year-old male with six obvious erectile events of normal duration. RAU and TAU data are transposed to the cumulative distribution curve. Using the above format, this patient falls in the 92nd percentile consistent with normal erectile activity.

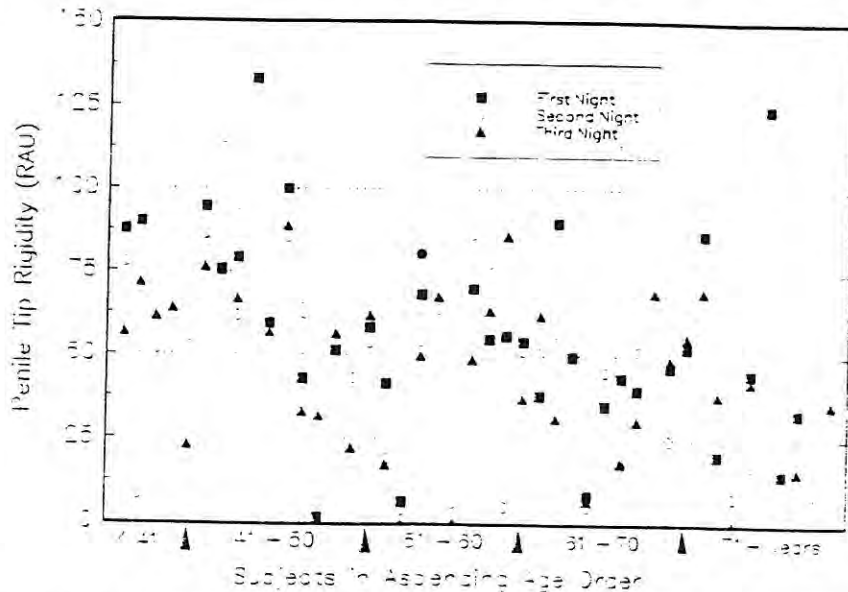


Figure 4. Penile tip rigidity measured by rigidity activity units (RAU) arranged by age in 44 potent men. Note intraindividual variation on a nightly basis but only a slight downward trend of rigidity with increasing age.

Case 2 (Fig. 6) presents the study results of a 71-year-old potent male whose graphic print-out is more difficult to assess. There are multiple short-duration erectile events with variable rigidity. Evaluation of tip RAU data demonstrate that this individual falls in the 30th percentile compared with the potent population of men studied. Although this man's NPTR data are not as straightforward to define as in Case 1, it is important to recognize that his awake erectile capacity is satisfactory for sexual function despite the borderline nocturnal rigidity measurements. A similar result for a younger impotent male, for instance, may indicate suboptimal nocturnal erectile activity consistent with organic erectile insufficiency. This latter patient may benefit from further investigation.

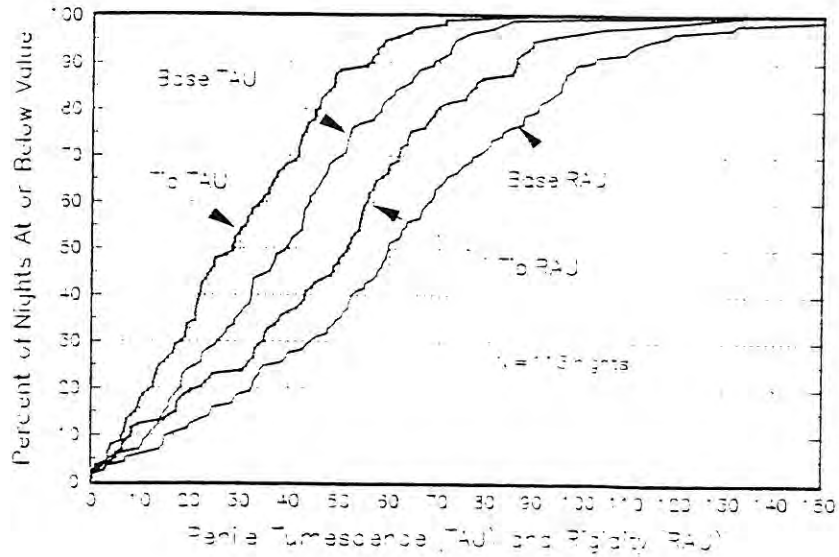
Case 3 (Fig. 7) demonstrates the NPTR results of a 60-year-old male with erectile failure following radical prostatectomy. His tip RAU reading falls in the 3rd percentile which clearly suggests poor nocturnal erectile activity consistent with an organic cause.

Further studies of an impotent population of men using new RAU and TAU measures of NPTR will be necessary to validate the use of the cumulative distribution curve presented in Figure 3.

Bain and Guay⁵ have shown that NPTR results from Rigiscan monitoring are highly reproducible when repeatedly studied in the same patient over time. In 15 of 17 patients, the

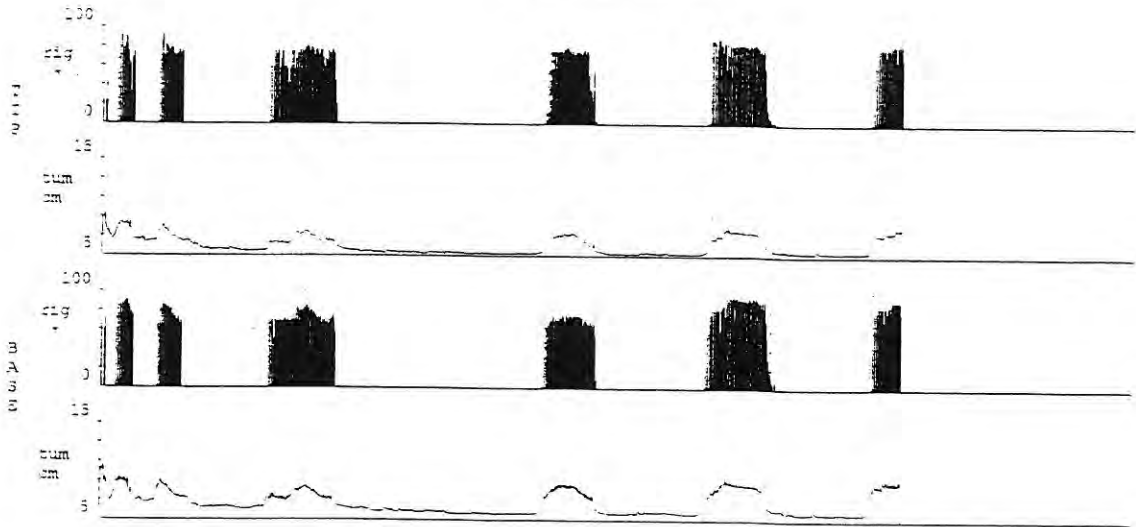
initial NPTR pattern was reproduced, and in the two patients whose patterns were not reproduced, this was explained by febrile illness or alcohol ingestion during the period monitored.

Several studies have been conducted to determine the relationship between erectile activity and aging. Karacan and associates¹³ have shown that total sleep time is constant from 20 to 50 years of age and that total REM sleep remains rather stable during this time period. They also demonstrated that total tumescence time and the average number of erectile episodes decreases with age.^{30,31} Kahn and Fisher,^{25,29} Jovanovic,²⁴ and Reynolds and associates¹⁷ also report a decrease in the number of tumescence episodes and duration of each episode with age in separate studies of potent men. On the other hand, Reynolds and associates¹⁷ found that measured buckling forces remained stable during the 3rd to 6th decades of life. Burris and coworkers⁷ studied 47 potent males and also found that total tumescence time decreased with advancing age; however, they did not note a significant change in the number of erectile episodes or penile rigidity. Recently, Levine and Carroll³⁸ also studied NPT in potent males, and their observations demonstrated an increase in the number of NPT events per evening with increasing age; however, the duration of each event appeared to decrease with age. This appears to be in contrast to previous studies, but



RigiScan Plus Session Graph

Data for Session 1
 Sampling Mode: Nocturnal
 Session Date: 05/04/90



Start Time: 0:00

Screen Width: 9 hrs

Patient Name:

Version: 4.0

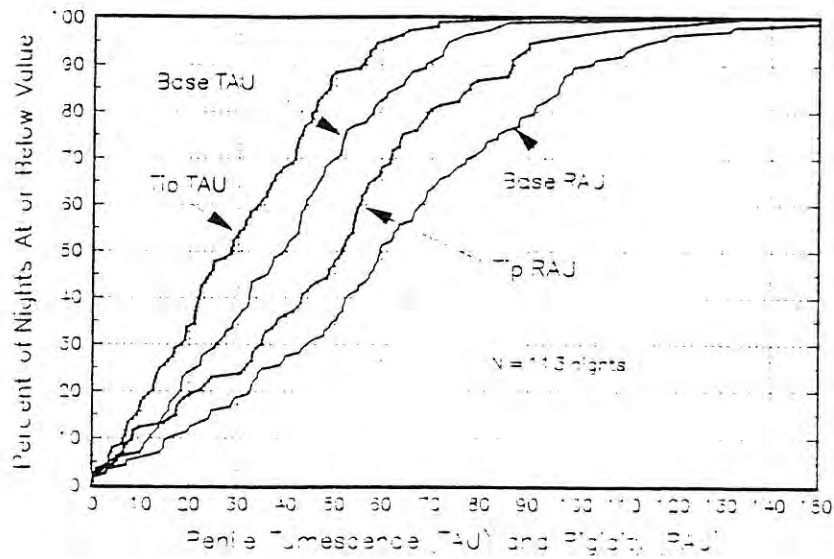
Patient ID#:

Print Date: 06/27/95

CASE 1: NORMAL MAN, 62 Years

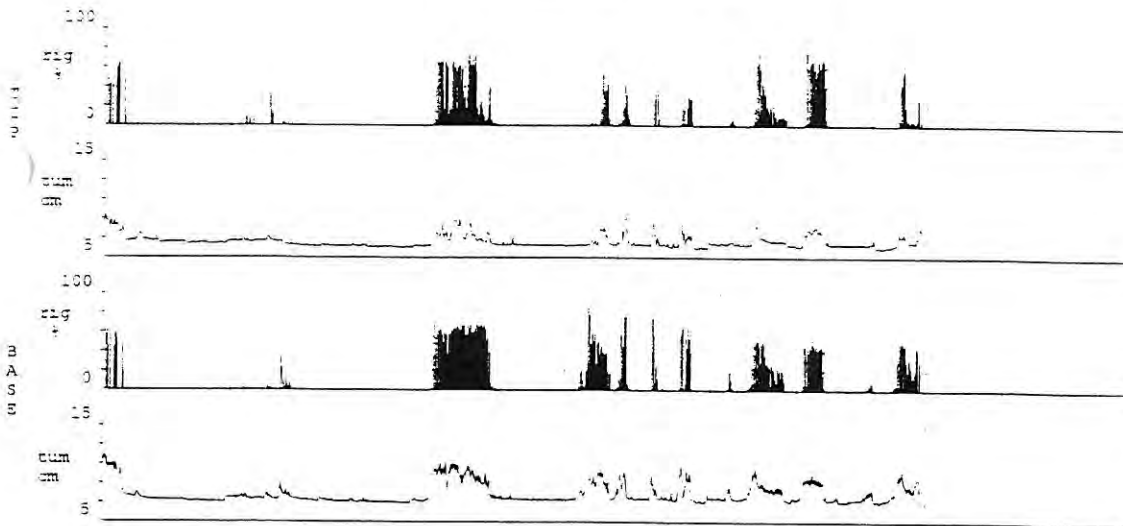
	RAU	TAU
Tip	89	46
Base	96	61

Figure 5. Case 1. Comparison of graphic printout and cumulative distribution curve ranking using tip RAU data places this 62-year-old potent man in the 92nd percentile.



RigiScan Plus Session Graph

Data for Session 2
 Sampling Mode: Nocturnal
 Session Date: 05/23/97



Start Time: 0:00

Screen Width: 3 hrs

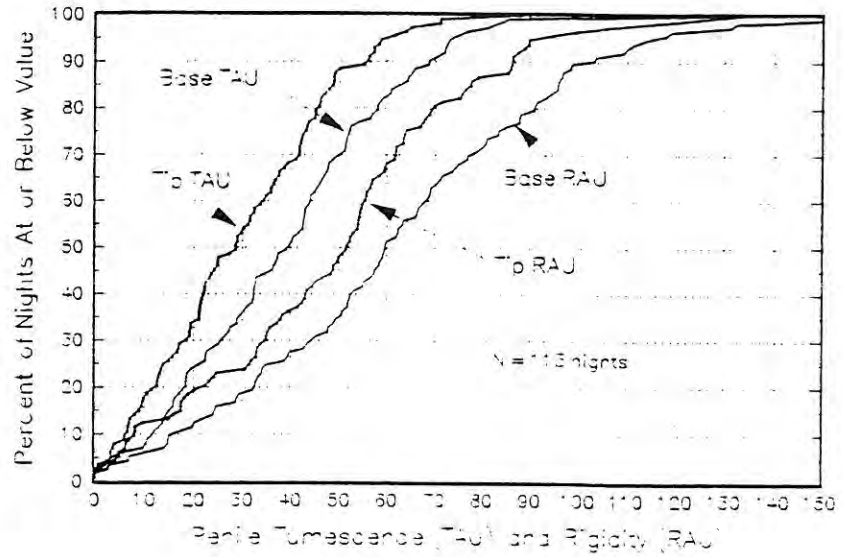
Patient Name:
 Patient ID#:

Version: 4.0
 Print Date: 06/27/95

**CASE 2: NORMAL MAN, 71 Years
 Difficult to Assess**

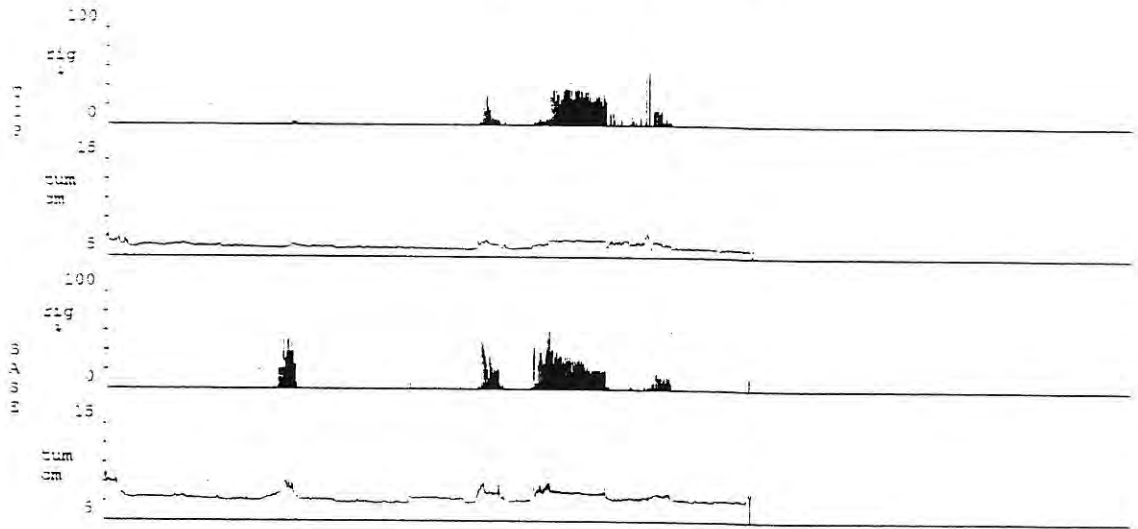
	RAU	TAU
Tip	34	20
Base	42	31

Figure 6. Case 2. Comparison of graphic printout and cumulative distribution curve ranking using tip RAU data places this 71-year-old potent man in the 30th percentile.



RigiScan Plus Session Graph

Data for Session 3
 Sampling Mode: Nocturnal
 Session Date: 05/29/90



Start Time: 0:00 Screen Width: 9 hrs
 Patient Name: Version: 4.0
 Patient ID#: Print Date: 06/27/95

**CASE 3: IMPOTENT MAN, 60 Years
 Radical Retropubic Prostatectomy,
 Impotent 5 Years**

	RAU	TAU
Tip	2	2
Base	5	5

Figure 7. Case 3. Comparison of graphic printout and cumulative distribution curve ranking using tip RAU data places this 60-year-old impotent man in the 3rd percentile. This is clearly abnormal and consistent with organic disease.

This difference may be explained by the fact that they defined an erectile episode as an event lasting for a minimum of 3 minutes whereas other studies have used a 5-minute criterion to define an erectile episode. They also demonstrated a weak but significant downward trend of tip rigidity with increasing age, although considerable variability was observed in subject responses (see Fig. 4). Although several different patterns of NPT responses have been described, it seems that there is an overall negative trend with increasing age. Perhaps further studies will help to clarify the exact nature of the effects of aging on NPT.

CURRENT TRENDS IN THE USE OF NPTR

The question that frequently arises and should be addressed in this article is "Does NPTR need to be used in the evaluation of all men with a complaint of erectile dysfunction?" Although the answer has certainly been debated and is subject to personal belief, the authors' opinion is probably not. Most men with significant organic factors associated with impotence such as diabetes mellitus, hypertension, history of smoking, elevated triglycerides or cholesterol, known vascular disease, all probably have primary organic erectile dysfunction. What needs to be remembered, however, is that most, if not all, of these men will also have a significant psychogenic component which is likely to exacerbate the effects of the underlying organic factors. On the other hand, it has been shown that men with diseases known to affect erectile dysfunction can also have normal NPT as well as adequate sexual function.³⁸

Clearly, the greatest benefit of NPTR is for the patient with no known neurovascular risk factors who presents with a history that seems suspicious for a psychogenic cause. In this patient, an objective measure of erectile function with NPTR should confirm the diagnosis and aid the physician to guide that patient to the appropriate therapy.

Hospital or sleep laboratory NPTR assessment is suggested where a compromise in patient compliance may occur as in the following circumstances: When there are concerns about (1) manual dexterity issues preventing the patient from properly applying the Rigiscan unit, (2) malingering, (3) dementia, and (4) validity in an outpatient, unobserved setting particularly in medicolegal testing. In this situation,

NPTR results are most valuable when normal because an abnormal result may indicate organic or undetectable factors interfering with non-REM or REM sleep. Full sleep laboratory evaluation should also be considered when there is a history or suspicion of sleep disturbance.

Although formal NPT monitoring provides valuable information in the evaluation of male erectile dysfunction, it is a costly and inconvenient test for the patient. Several studies have addressed this issue by investigating the usefulness of penile tumescence monitoring during daytime naps. Gordon and Carey³⁹ studied seven healthy male subjects who were deprived of at least half of their normal amount of sleep the evening before the study and then performed NPT testing during a 3-hour morning nap. They found that the subjects were able to fall asleep rapidly, slept well, and experienced REM sleep. More recently, Morales and associates⁴² also studied this technique. They examined 18 impotent men who also underwent formal NPT testing during a morning nap session. Sixteen of the 18 patients (88%) experienced REM sleep, and 4 patients who did not experience erections on two separate sessions of nocturnal sleep recording also did not experience tumescence during the nap. Of the 12 patients with documented erections at night, 9 also exhibited erectile episodes during napping. Although both of these studies are small, further investigation may indicate that this technique may provide a more convenient and less costly method than 2 to 3 nights of formal sleep laboratory NPT monitoring.

The causes of erectile dysfunction are often complex and impotent men frequently exhibit combinations of organic and psychogenic causes. Several authors have attempted to use NPTR testing to unravel these complex issues to better define the specific subtypes of erectile dysfunction.

Shabsigh and colleagues⁴¹ studied 50 patients with erectile dysfunction in an effort to compare the results of penile duplex ultrasound with intracavernous injection of vasoactive drugs to those of NPT monitoring in a sleep laboratory. They demonstrated that although NPT monitoring shows abnormal findings in vasculogenic impotence, it cannot distinguish arterial from venous causes. On the other hand, it seems that penile duplex ultrasonography with intracavernous injection of vasoactive agents may be able to detect the nature of penile vascular insufficiency, especially in patients with the pelvic steal syndrome in

whom NPT results are often normal. Conversely, they note that penile duplex ultrasonography with papaverine injection yields normal findings in patients with neurogenic erectile dysfunction demonstrating that NPT monitoring is more sensitive in the evaluation of neurogenic impotence. Montague and associates¹¹ also studied 50 men with erectile dysfunction comparing the results of infusion pharmacocavernosometry (IPC) with NPTR monitoring as measured by Rigiscan. Using traditional criteria for IPC, they found a poor correlation between the two. When modified criteria were established using logistic regression techniques, however, a much stronger correlation between the two was observed. They concluded, however, that the correlation is not strong enough to suggest that clinicians should rely on the results of either study alone in making specific diagnosis of erectile dysfunction.

An additional use for NPTR is to provide real-time objective measures of erectile response to various pharmacologic treatments or as a comparative research tool to developing techniques of assessing erectile response (i.e., duplex penile ultrasound, cavernosometry). Unfortunately, psychogenic factors that may inhibit erectile response to pharmacologic erection therapy and visual sexual stimulation¹² must be taken into consideration in the subject being tested with a new treatment regimen and having erectile response assessed with NPTR.

Several studies have been done to compare NPT with the results of intracorporeal pharmacological (ICP) erection testing. Allen and Brendler² studied 37 impotent patients who underwent a formal NPT evaluation and correlated those results with subsequent ICP testing. They concluded that the response to ICP testing does not accurately distinguish psychogenic from organic impotence, but that the response does correlate well with the degree of physiologic impairment of erectile function and that a thorough NPT evaluation provides an excellent prediction of the ICP erection testing response but that the reverse is not true. They maintain that NPT should be performed before ICP erection testing to avoid unnecessary and inappropriate treatment of psychogenic impotence. In his comments on this study, Lue agreed that ICP testing is not adequate to differentiate psychogenic from organic impotence.² He contends that in patients with suspected vasculogenic impotence, routine NPT testing is not indicated.

Allen and coworkers² studied 40 impotent men comparing the results of duplex ultra-

sound evaluation to a formal NPT evaluation. Their results demonstrated that in men with a history of psychogenic illness, duplex ultrasound is unreliable. They believed that anxiety and increased sympathetic stimulation resulted in abnormal responses to pharmacologic stimulation and subsequent inaccurate duplex ultrasound measurements. They concluded that in patients believed to have a psychogenic cause of impotence, NPT testing should be carried out for appropriate treatment recommendations.

Our personal approach is to obtain a complete medical history, including medications, and a focused physical examination, including a neurovascular assessment. When a clear psychogenic cause is suggested by the history or because of absence of obvious neurovascular risk factors, then NPTR is recommended to confirm the diagnosis. In those patients who present with a complex history, which does not clearly indicate either an organic or psychogenic etiology, then penile duplex ultrasound with pharmacologic stimulation is performed. Patients who present with a recently identified duplex ultrasound pattern, including suboptimal peak systolic velocity (less than 30 cm/sec), normal end diastolic flow velocity (less than 5.0 cm/sec), and a full erectile response to pharmacologic stimulation, suggests the possibility of excessive adrenergic tone as the potential cause for the diminished arterial inflow manifested by the subnormal peak systolic flow velocity. These patients would benefit from further evaluation with NPTR to confirm a psychogenic origin associated with excessive adrenergic tone.^{13,33} In this circumstance, the cause of the erectile insufficiency is not clear but seems to indicate a psychogenic one. Therefore, NPTR is far less invasive and less expensive than phalloarteriography or infusion cavernosometry and will offer more objective data to support the physician's recommendation for sexual rehabilitation therapy.

Several authors have suggested selecting the "best erection" recorded during NPT(R) monitoring for quantitative classification of erectile function.^{7,15,32,36} Most recently, Sohn and colleagues³² reintroduced this concept as a time-sparing and examiner-independent method to evaluate NPTR recordings. They defined the best erection as the erectile event with the highest rigidity and the greatest tumescence. Using normal criteria established by Dacomed³⁸ and the observations of Kaneko and Bradley,³⁷ no pathognomonic patterns of neurogenic or vasculogenic impotence could be

defined by this technique. Although statistical differences in severity of erectile dysfunction between various subgroups were noted, the specific cause of impotence was unable to be defined from NPTR recordings alone.

We continue to believe that the use of "best erection" criteria addresses nocturnal erectile capacity for full erection rather than the entire nocturnal erectile behavior as recorded on 1 or more nights of study. Nocturnal erectile capacity may prove to aid in the diagnostic assessment of impotence with further study. On the other hand, using the cumulative distribution curve approach with TAU and RAU data as suggested by Levine and Carroil³⁵ allows one to compare cumulative data of one individual with a range of NPTR experience in a potent population rather than a set of unconfirmed criteria.

We agree with Sohn and co-workers³² and Condra and co-workers¹¹ that no single test exists to date which enables the physician to qualify the origin and degree of impotence. Despite its intrinsic weaknesses and limitations, however, NPTR is a valuable resource in differentiating between psychogenic and organic impotence. Ultimately, for the practicing urologist, the goal of evaluating erectile dysfunction is to provide useful information so as to direct the patient to the most appropriate therapeutic options. For many patients, NPTR provides this information in a rather noninvasive and relatively inexpensive manner.

References

- Allen J, Ellis D, Carroil JL, et al: Snap gauge band versus multidisciplinary evaluation in impotence assessment. *Urology* 34:197, 1989
- Allen RP, Brendler CB: Nocturnal penile tumescence predicting response to intracorporeal pharmacological erection testing. *J Urol* 140:518, 1988
- Allen RP, Engel RM, Smolev JK, et al: Comparison of duplex ultrasonography and nocturnal penile tumescence in evaluation of impotence. *J Urol* 151:1525, 1994
- Aserinsky E, Kleitman N: Regularly occurring periods of eye motility and concomitant phenomena during sleep. *Science* 118:273, 1953
- Bain CL, Guay AW: Reproducibility in monitoring nocturnal penile tumescence and rigidity. *J Urol* 148:811, 1991
- Barry IM, Blank B, Boileau M: Nocturnal penile tumescence monitoring with stamps. *Urology* 15:171, 1980
- Bradley WE: New techniques in evaluation of impotence. *Urology* 29:383, 1987
- Bradley WE, Timm GW, Gallagher JM, et al: New method for continuous measurement of nocturnal penile tumescence and rigidity. *Urology* 26:4, 1985
- Burris AS, Banks SM, Sherins RJ: Quantitative assessment of nocturnal penile tumescence and rigidity in normal men using a home monitor. *J Androl* 10:492, 1989
- Chung WS, Choi HK: Erotic erection versus nocturnal erection. *J Urol* 143:294, 1990
- Condra M, Fenemore J, Reid K, et al: Screening assessment of penile tumescence and rigidity: Clinical test or snap gauge. *Urology* 29:254, 1987
- Earris CM, Morales A, Marshall W: Penile insufficiency: An operational definition. *J Urol* 139:536, 1988
- Ek A, Bradley WE, Krane RJ: Nocturnal penile rigidity measured by the snap-gauge band. *J Urol* 129:964, 1983
- Ellis DJ, Doghirami K, Bagley DH: Snap-gauge band versus penile rigidity in impotence assessment. *J Urol* 140:61, 1988
- Fisher C: Dreaming and sexuality. In Loewenstein RM, Newman LM, Schur M, et al (eds): *Psychoanalysis—A General Psychology*. New York, International University Press, 1966, p 537
- Fisher C, Gross J, Zuck R: Cycle of penile erections synchronous with dreaming (REM) sleep. *Arch Gen Psychiatry* 12:29, 1965
- Fisher C, Schiavi RC, Edwards A, et al: Evaluation of nocturnal penile tumescence in the differential diagnosis of sexual impotence. *Arch Gen Psychiatry* 36:431, 1979
- Fisher C, Schiavi R, Lear H: The assessment of nocturnal REM erection in a differential diagnosis of sexual impotence. *J Sex Marital Ther* 1:277, 1975
- Fronth DA, Goldstein I, Peyton TR, et al: Characterization of penile erectile states using external computer-based monitoring. *Journal of Biomechanical Engineering* 109:110, 1987
- Gordon CM, Carey MP: Penile tumescence monitoring during morning naps: A pilot investigation of a cost-effective alternative to full night sleep studies in the assessment of male erectile disorder. *Behav Res Ther* 31:503, 1993
- Halverson HM: Genital and sphincter behavior of the male infant. *J Gen Psychol* 56:95, 1940
- Impotence: Psyche vs. Soma. *Med World News* 17:28, 1976
- Jovanovic UJ: Der Effekt der ersten Untersuchungsnacht auf die Erektionen im Schlaf. *Psychother Psychosom* 17:295, 1969
- Jovanovic UJ: Sexuelle Reaktionen und Schlarperiodik bei Menschen. Ergebnisse Experimenteller Untersuchungen. Stuttgart, Ferdinand Enke Verlag, 1972
- Kahn E, Fisher C: Amount of REM sleep erection in the healthy aged. *Psychophysiology* 5:226, 1968
- Kahn E, Fisher C: Amount of REM sleep and sexuality in the aged. *J Geriatr Psychiatry* 2:181, 1969
- Kaneko S, Bradley WE: Evaluation of erectile dysfunction with continuous monitoring of penile rigidity. *J Urol* 136:1026, 1986
- Karacan I: Clinical value of nocturnal erection in the prognosis and diagnosis of impotence. *Medical Aspects of Human Sexuality* 4:27, 1970
- Karacan I, Goodenough DR, Chapiro A, et al: Erection cycle during sleep in relation to dream anxiety. *Arch Gen Psychiatry* 15:183, 1986
- Karacan I, Hirsch CI, Williams RL: Some characteristics of nocturnal penile tumescence in elderly males. *J Gerontol* 27:39, 1972
- Karacan I, Salis PL, Thornby JL, et al: The ontogeny of nocturnal penile tumescence. *Waking and Sleeping* 1:27, 1976
- Karacan I, Salis PL, Ware JC, et al: Nocturnal penile tumescence and diagnosis in diabetic impotence. *Am J Psychiatry* 135:191, 1978

33. Karacan I, Salis PJ, Williams RL. The role of the sleep laboratory in the diagnosis and treatment of impotence. *In*: Williams RL, Karacan I, Frazier SH (eds): *Sleep Disorders, Diagnosis and Treatment*. New York: John Wiley and Sons, 1978.
34. Karacan I, Williams RL, Thornby D, et al: Sleep related penile tumescence as a function of age. *Am J Psychiatry* 132:932, 1975.
35. Kirkeby HJ, Andersen M, Poulsen EU: Nocturnal penile tumescence and rigidity: Translation of data obtained from normal males. *Int J Impot Res* 1:115, 1989.
36. Kirkeby HJ, Poulsen EU, Pererssen T, et al: Erectile dysfunction in multiple sclerosis. *Neurology* 38:1366, 1988.
37. Krane RJ: Sexual function and dysfunction. *In*: Walsh PC, Gittes RJ, Perlmutter AD, et al (eds): *Campbell's Urology*, ed 5. Philadelphia, WB Saunders, 1986, p 719.
38. Levine LA, Carroll RA: Nocturnal penile tumescence and rigidity in men without complaints of erectile dysfunction using a new quantitative analysis software. *J Urol* 152:1103, 1994.
39. Levine LA, Carroll RA, Chapman TN: Identification of a new penile duplex ultrasound vascular flow pattern. *J Urol* 153:331A, 1995.
40. Marshall P, Earls C, Morales A, et al: Nocturnal penile tumescence recording with stamps: A validity study. *J Urol* 128:946, 1982.
41. Montague DK, Lakin MM, Medendorp S, et al: Intrusion pharmacocavernosometry and nocturnal penile tumescence findings in men with erectile dysfunction. *J Urol* 143:768, 1991.
42. Morales A, Condra M, Heaton JP, et al: Diurnal penile tumescence recording in the etiological diagnosis of erectile dysfunction. *J Urol* 152:1111, 1994.
43. Morales A, Condra M, Reid K: The role of nocturnal penile tumescence monitoring in the diagnosis of impotence: A review. *J Urol* 143:441, 1990.
44. Ohlmeyer P, Brilmayer H, Hullstrung H: Periodische Vorgänge im Schlaf. *Pflügers Arch* 248:559, 1944.
45. Pressman MR, Fry JM, DiPhillipo MA, et al: Avoiding false positive findings in measuring nocturnal penile tumescence. *Urology* 34:297, 1989.
46. Pressman MR, Fry JM, DiPhillipo MA, et al: Problems in the interpretation of nocturnal penile tumescence studies: Disruption by occult sleep disorders. *J Urol* 136:595, 1986.
47. Reynolds CF, Thase ME, Jennings JR, et al: Nocturnal penile tumescence in healthy 20 to 59 year-olds: A re-visit. *Sleep* 12:368, 1989.
48. Rigiscan: Ambulatory Rigidity and Tumescence System Document No. 750-156-0486. Dacomed Corporation, Minneapolis, MN.
49. Roose SP, Glassman AH, Walsh BT, et al: Reversible loss of nocturnal penile tumescence during depression: A preliminary report. *Neuropsychobiology* 5:284, 1982.
50. Schiavi RC: Luteinizing hormone and testosterone during nocturnal sleep: Relation to nocturnal penile tumescent cycle. *Arch Sex Behav* 6:97, 1977.
51. Shabsigh R, Fishman I, Shotland Y, et al: Comparison of penile duplex ultrasonography with nocturnal penile tumescence monitoring for the evaluation of erectile impotence. *J Urol* 143:924, 1990.
52. Sohn MH, Seeger L, Sikora R, et al: Criteria for examiner-independent nocturnal penile tumescence and rigidity monitoring (NPTR): Correlations to invasive diagnostic methods. *Int J Impot Res* 5:59, 1993.
53. Thase ME, Reynolds CF, Glang LM, et al: Nocturnal penile tumescence in depressed men. *Am J Psychiatry* 144:89, 1987.
54. Thase ME, Reynolds CF, Jennings JR, et al: Diagnostic performance of nocturnal penile tumescence studies in healthy, dysfunctional (impotent), and depressed men. *Psychiatry Res* 26:79, 1988.
55. Tomaszecski CS, Carroll RA, Levine LA: Psychogenic impotence evaluated by penile duplex ultrasonography. *In*: Proceedings of the 9th Annual Western Section American Urological Association Meeting, Palm Desert, California, November 1995.
56. Wasserman MD, Pollack CP, Spieiman AJ, et al: The differential diagnosis of impotence. *JAMA* 243:2038, 1980.
57. Wasserman MD, Pollack CP, Spieiman AJ, et al: Theoretical and technical problems in the measurement of nocturnal penile tumescence for the differential diagnosis of impotence. *Psychol Med* 42:575, 1980.
58. Wein AJ, Fishkin R, Carpiello VL, et al: Expansion without significant rigidity during penile tumescence testing: A potential source of misinterpretation. *J Urol* 126:343, 1981.

Address reprint requests to

Laurence A. Levine, MD
 Department of Urology
 Rush University
 1653 W. Congress Parkway
 Chicago, IL 60612-3833