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Anal Sphincter Electromyogram for Dysfunction of Lower Urinary Tract and Pelvic Floor

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1. Introduction

The electromyogram (EMG) is a diagnostic tool that measures and records the electrical activity produced by skeletal muscles. The anal sphincter and urethral external sphincter are skeletal muscle in nature. There are two kinds of EMG in widespread use in the field of urodynamic investigation: surface (patch) and intramuscular (fine-wire) EMG. In our laboratory, the fine-wire electrode is introduced by aid of a needle, we called it needle-guided wire electrode, or simply as wire electrode (Xu et al, 2007). Sphincter EMG studies the bioelectric potentials generated in the distal striated sphincter mechanism. Such studies are performed at two different levels of sophistication, each with distinct goals and requiring different instrumentation. The first, termed kinesiologic studies, are commonly performed in the urodynamic laboratory and simply examine sphincter activity during bladder filling and voiding. The second are neurophysiologic tests, which require considerable expertise and elaborate equipment and are designed to examine the integrity of innervation of the muscle. However, clinically the most important information obtained from sphincter EMG is whether there is coordination or discoordination between the external sphincter and the bladder. Surface patch electrodes are placed on the skin/mucosa overlying the muscle of interest and thus pick up the potentials produced by various muscles in the vicinity. Wire electrodes are preferable because they are placed directly into the muscle of interest, allowing for the detection of activity in individual motor units. EMG of the anal sphincter derived from transdermal route, being combined with simultaneous recording of uroflow rate, and bladder, abdominal pressure and then detrusor pressure (=bladder pressure–abdominal pressure) during both bladder storage and voiding phases, can give rise to a general essential data about functional states of the main elements (i.e. detrusor and sphincter) of lower urinary tract and the pelvic floor.

Sphincter dysfunction, either overactive or underactive, may occur in patients with either neurogenic or non-neurogenic lower urinary tract dysfunctions (LUTD). Recently, the European Association of Urology published its guideline (2008 version) on neurogenic LUTD, which is categorized into detrusor and sphincter levels, and the location of lesions is no longer emphasized (Stöhrer et al, 2009). Although several classification systems have been proposed for neurogenic LUTD, the recommendations for a functional classification for...
motor function are based on urodynamic and clinical findings. According to the functional state of detrusor and sphincter (whether it is overactive, normo-active or underactive), they are permuted and combined following the principle of mathematics, and eight groups of neurogenic LUTD are classified thereafter (Table 1): overactive detrusor (3 groups, combined with overactive, normo-active, or underactive sphincter, respectively); underactive detrusor (3 groups, combined with overactive, normo-active, or underactive sphincter, respectively); and normo-active detrusor (2 groups, combined with only overactive, or underactive sphincter, respectively).

| Type No | States of the functional elements of the lower urinary tract | Location of the lesion |
|---------|---------------------------------------------------------------|-----------------------|
|         | Detrusor           | Sphincter             |                       |
| 1       | Overactive         | Overactive            | Spinal               |
| 2       | Overactive         | Normo-active           | Suprapontine         |
| 3       | Overactive         | Underactive            | Lumbosacral          |
| 4       | Underactive        | Overactive            | Lumbosacral          |
| 5       | Underactive        | Normo-active           | Lumbosacral          |
| 6       | Underactive        | Underactive            | Subsacral            |
| 7       | Normo-active       | Overactive            | Sphincter only       |
| 8       | Normo-active       | Underactive            | Sphincter only       |

Table 1. The European Association of Urology (EAU)–Madersbacher classification system of neurogenic LUTD.

In both congenital and acquired neurogenic LUTD, early diagnosis and treatment are essential because irreversible changes within the lower urinary tract may occur, even when the related neuropathologic signs are normal. Additionally, neurogenic LUTD can, by itself, be the presenting feature of neurologic pathology. The classification of the disease cannot be defined purely by site of the neurogenic lesion. Typically, the detrusor should be overactive for those with suprasacral lesions or upper motor neuron lesions, and be underactive for those with sacral or hypsacral, or lower motor neuron lesions. However, most patients may present not typically with a combination of both storage and voiding symptoms, which may be due to multiple spinal cord injury, incomplete spinal cord injury or non-traumatic spinal cord lesion. A comprehensive urodynamic investigation protocol including EMG is mandatory to reveal the dominant character. We have also found some cases who presented with clinical, urodynamic and EMG abnormality, whereas their MRI and surgical exploration were negative. The neurogenic lesion developed at follow-up, and the lesion usually was unresectable at that time.

In the absence of neurologic disease, one cannot use the term detrusor-sphincter dyssynergia to describe EMG finding of sphincter overactivity. Instead, the term non-neurogenic neurogenic bladder, pelvic floor hypertonicity or dysfunctional voiding is used (Hinman, 1986; Deindl et al, 1998; Carlson et al, 2001). Sphincter functional state represents the state of the pelvic floor because of its ability of phasic functional change. Generally, sphincter overactivity is a behavioral problem and is often seen in children with voiding dysfunction and in neurologically normal women with voiding problems (Carlson et al, 2001; Goldman & Zimmern, 2006; Kuo, 2010). We have identified this phenomenon in Chinese women with urinary frequency and poor-weak flow, and the prevalence of this
disorder is more than 30%. Most of those women had a history of such behavioral problem as voluntary withholding of urination in individuals who work long hours, or voiding in an unnatural position, i.e., with her haunch hanged up for avoidance of touching commode. Furthermore, sphincter overactivity not only implies some degree of functional obstruction, but also co-exists with overactive bladder (OAB) or stress urinary incontinence (SUI). The intrinsic mechanism may be related with guarding or continence reflex of the sacral spinal cord (Kamo et al, 2004). When the bladder is filled to near capacity or following a sudden increase in intravesical pressure, pudendal motoneuron firing occurs. Urodynamic and EMG data indicate that a rise in maximal urethral pressure corresponds to increased EMG activity of the striated muscles of the pelvic floor. This phenomenon has been termed the guarding or continence reflex and represents a somatic mechanism for increasing urethral resistance. The firing of the pudendal motoneuron may also be induced by detrusor overactivity, more leaked urine into the proximal urethra in female patients with SUI, inhibited by more urine into the lower part of the ureter or acute anal stretch (Akkad et al, 2007; Chen et al, 2007; Chen et al, 2008; Xu et al, 2010). Voluntary overactivity of this reflex can result in obstruction of the lower urinary tract and differentiating voluntary contraction of this sphincter from detrusor sphincter dyssynergia can be difficult. Close examination of the onset of sphincter activity relative to the rise in intravesical pressure can help distinguish a guarding reflex from true detrusor sphincter dyssynergia (Rudy & Woodside, 1991). There were 3 characteristic urodynamic features in this patient population that were distinct from those seen in patients with true detrusor-external sphincter dyssynergia: (1) quieting of the external sphincter EMG immediately prior to the onset of a detrusor contraction, (2) quieting of the EMG during the upslope of a detrusor contraction, and (3) augmented EMG activity during the downslope of the detrusor contraction. Although both involve incoordinate bladder and pelvic floor/external sphincter activity, a simple cystometrogram (CMG) with EMG may allow reliable differentiation between true detrusor-external sphincter dyssynergia and non-neurogenic neurogenic bladder.

Differentiating voluntary contraction of the sphincter from detrusor sphincter dyssynergia or dysfunctional voiding is not as important clinically as someone declared previously. If the recipient is a healthy volunteer, the finding may be voluntary contraction of the sphincter. If the recipient is a patient and he or she has symptoms related to lower urinary tract, we would rather consider it as dysfunctional voiding in neurological intact ones, or neurogenic detrusor sphincter dyssynergia in patients whose neurogenic system is involved. The real intention of the guarding reflex is beneficial for the subject. It is associated with unbalance between the detrusor and sphincter, and perhaps it is already a pathologic change when the reflex is fortified too much, such as in patients complained of OAB or SUI. The most important question that urodynamic personnel should answer is whether the finding is an artifact or abnormal sign.

EMG artifacts were so common in adults that EMG signals should be interpreted with great caution previously, and they could be very frustrating and difficult to deal with especially when EMG studies was performed using surface patch electrodes. At present, especially when wire electrode is widely used in practice in this institution, the situation is not as what they addressed and sphincter state whether normal or overactive, could be displayed very distinctly (see below).

This Chapter deals with the utilization of the EMG signal (with needle electrodes) from the anal sphincter as an indicator of dysfunction of the lower urinary tract. The major impact is that anal sphincter EMG may serve to present the functional state of the urethral sphincter.
At the following description text, we are going to express our thinking and measures about this subject.

2. Consideration

Urodynamic study (Life-Tech Urovision Janus and MMS Solar) involving more than 7000 patients with LUTD have been conducted with wire electrode EMG and gained better results since 2002 in this institution. Other equipments (Laborie, Nidoc, Dantec and Duet) have also been used since 1985. The aetiology and causes underlying various LUTD were explored, and then allow management decisions to be made. Apart from the guidelines the company proposed, some modifications were undertaken. The surface patch electrode was forsaked from the very beginning. The main reason was that it cannot be pasted on the perianal skin firmly. The wire of the electrode was first exerted out from inside of the needle hole (Fig. 1,b), and in our modification it was inserted into the needle hole from outside inside (Fig. 1 c, d). The most brittle part of wire is the site of reflexion, and if this happened, the debris will remain in the needle tube, not fall into the surrounding tissue.

2.1 Technological consideration: Electrocircuit system as a whole

EMG artifacts may be caused by multiple sources including the patient and the urodynamics suite itself. Electrical signals from other electrical equipment such as lights, transformers, electrocautery units, or the fluoroscopy equipment can all contribute. Other sources of artifact include improper grounding, defective insulation, and movement of the patient (Webster & Guralnick, 2002). Apart from the usually mentioned factors leading to weak signals or artifacts, including electrodes being placed too far from the sphincter, becoming wet, having too low gain or falling off the patient (Blaivas et al, 2007), other factors associated with technical consideration should be more important thereafter. From the point of electrology, the EMG recording system of the equipment, the cables, the wires and the muscles between two electrode tips compose an electrocircuit system. The internal or external factors of the system, such as its integrity, consistency, environmental temperature, humidity, the bio-electric resistance of the tissues tested in between and distance length between the electrodes, may influence the tracing outcomes. External moving force against the needle or breaking off of the closed circuit of the system should also be considered. The static bio-electric resistance between the two poles are dependent on the medium in between. In our series of 152 patients measured at a radian of 1.0 \( \pi \) between the two poles, resistances were 6.30±2.10 kΩ (3.0~12.0), 7.74±2.97 kΩ (1.5~16.0), 2150.66±1733.58 kΩ (100~8000) for saline, muscle and skin respectively (Cui et al., 2009).

Apparently, the resistance of muscle was higher than saline and lower than skin (P=0.001). Most EMG recording artifacts are secondary to a recorded potential that does not arise from the depolarization of the striated muscle to be studied. EMG raw signals, expressed in micro-volts, were obtained directly from the electronic circuitry and were not normalized. For example, when surface patch is used, the potential may come from the levator ani muscles or other pelvic floor muscles. If these factors are all OK and within normal limits, abnormal findings on the screen may imply pathological significance, and cannot be blamed ad libitum as artifacts.

From our experience, nearly 70%-80% so-called “artifacts” or bad recordings of EMG came actually from an open circuit, not from a closed circuit, of the electrocircuit system (Fig. 2). Open circuit means some thing wrong, usually break or bad conduct, occurred in the electrocircuit system. Closed circuit means an intact electrocircuit system, The normal EMG
tracings are continuous and consecutive, strengthening in the storage phase and relaxing in the voiding phase. It had better response to cough or other physiological actions. At the beginning of CMG, before bladder filling begins, the patient is asked to demonstrate volitional control of the sphincter by actively contracting and relaxing it. The ability to do this implies intact pyramidal tracts. Next, the bladder control reflex is tested by squeezing the glans penis or clitoris or pulling on the Foley catheter. A burst of EMG activity is a positive result and implies an intact sacral arc. Bladder filling then begins; and, as it proceeds, there is a progressive recruitment of sphincter activity, demonstrated by increased amplitude and frequency of firing. Just before the onset of voiding, sphincter activity ceases and remains so for the duration of micturition. Once the bladder is empty, sphincter EMG activity resumes.

Fig. 1. Needle-guided wire electrodes and its modification: the original design with wire inserted into needle from needle tail then inside outside (b), modified design with wire inserted into needle head from outside inside (c, d), and the modified needles puncturing into the anal skin at 3, 9 and 10:30 o’clock (a).
When needle-guided wire electrodes are used, the raw signals of the anal sphincter coming from half circle of the anus are expressed on the urodynamic screen in a condensed fashion (20 sec/grid). Their potential amplitude could be measured: the more sphincter it includes, the higher the amplitude. Concentric electrodes and oscilloscope were used to measure motor unit potentials (MUP), and signals came from only a granule-size sphincter were displayed on the oscilloscope in a faster velocity (20 ms/grid). So the former was a continuous trace and latter was wave-like or ECG-like. However, due to changes in location, orientation or other factors, it is not possible to point the difference. The electrocircuit was secured making sure that the equipment was in good condition, the wires in unbroken and no tarnishing state, the inserted needles free from outside interference and so on. Otherwise, the trace would be abnormal and referred to as “artifact”. The crenate-like EMG trace in Fig 2a came from the screen before the wire was inserted into the anus with the wire dispersed. The lower amplitude trace in Fig 2b came from the screen before the wire was inserted into the anus with the wires linked together. The intermittent, crenate-like, sawtooth-like, or wave-like EMGs in Fig. 2c and d show that something went wrong (wire is broken or the needle guided the wire is compressed by lateral force) in the circuit during examination. By re-doing or changing of the wire electrode, the trace became normal.

Fig. 2. Urodynamic and EMG trace recordings displaying anal sphincter EMG: the electrocircuit system is in open circuit (a) or in closed circuit (b), without insertion of the wire or urodynamic investigation. Bad recordings of EMG during urodynamic investigation: intermittent trace from beginning to end as dashed arrows indicated (c), or in the intermediate phase due to external depressing force toward the needle, which turned normal (as solid arrows indicated) after revision of the needle direction (d).
2.1.1 Wire or patch electrodes

Compared with the perineal surface patch electrodes which are widely used in most labs in Europe and United States, the only shortcoming of the needle-guided wire electrode is a slight pain associated with needle puncturing. The surface electrode is easy to fall off, having low sensitivity and poor reliability. Although patients have a greater tolerance for surface EMG electrodes, the reliability of the latter when compared to needle-guided wire electrode or even concentric needle electrode (CNE) is doubtful. Volume conduction results in the compounding of motor unit signals from all the muscles of the perineal body, makes signals from an individual muscle difficult to identify. Given the differing innervation of the levator ani muscles and the external urethral sphincter it is unlikely that EMG activity of the levator ani muscles recorded with surface electrode adequately reflects motor unit activity of the external urethral sphincter. As the ways of expression are concerned, CNE inserted into the rhabdosphincter close to the urethral orifice are superior to surface electrodes patched close to the anus for EMG documentation of urethral sphincter relaxation during voiding (Mahajan et al, 2006). A study was conducted to compare interpretations of EMG recordings from surface electrodes with those from urethral CNEs during voiding. Consenting women underwent urodynamic testing with a 30 G, 3.8 cm CNE at the 12 o’clock position in the striated urethral sphincter muscle, and with surface electrodes placed at the 2 and 10 o’clock positions around the anus. Pressure-flow studies (PFS) were conducted with simultaneous input from both EMG electrodes. Representative, de-identified paper copies of EMG signals were assembled by chronology and electrode type. Six examiners unaware of the patient details were asked to determine if the tracings were interpretable and whether there was quiescence of the urethral sphincter motor unit during voiding. The consistency between the interpretations of each tracing was assessed using McNemar and κ statistics. Twenty-two women undergoing urodynamic testing for incontinence (16), voiding dysfunction (two) or urinary retention (four) participated in the study. CNE tracings were consistently more interpretable than surface electrode tracings (mean 89% vs 67%). When tracings were interpretable, a significantly higher percentage of CNE EMG tracings (mean 79%) had urethral sphincter motor unit quiescence than surface electrode EMG tracings (mean 28%). The κ values for agreement among the reviewers’ interpretations were highly variable and none were statistically significant. Reviewers unanimously agreed on only 12 of the 44 tracings, and 11 of these showed quiescence when using a CNE. CNEs were more often interpretable than surface electrodes for determining motor unit quiescence during voiding. CNE EMG appeared to have greater clinical utility for central reading than surface electrodes (Mahajan et al, 2006).

2.1.2 Radian and its influence on the amplitude of potentials

Surface electrodes are placed at the 2 and 10 o’clock positions around the anus, and the needle-guided wire electrodes we used, representing two poles of the circuit, are placed at the 3 and 9 o’clock position around the anus. CNE has two poles indeed too, but the distance of them is almost neglectable and it describes less myoelectric information than those with longer distance. Two wire electrodes at radian distance of $0.25\pi$, $0.75\pi$, or $1.0\pi$ prevail over CNE in potential amplitude and the potential increased in accordance with the radian as our experiment displayed. This experiment was conducted in this institution on patients suffered from LUTD to study the role of radian distance of the wire-electrodes and its impact on the amplitude and quality of EMG (Cui et al., 2009). Between September 2008 and January 2009, a total of 152 patients with LUTD underwent comprehensive urodynamic
study. Each underwent three sessions of CMG, EMG and PFS with one of the 3 radians when EMG was conducted. The electrodes were randomly placed at radians of 0.25 π (9-10:30 o’clock), 0.75 π (10:30-3 o’clock), or 1.0 π (3-9 o’clock) (Fig. 3. a). The electro-potentials during storage and voiding phases were measured separately, recorded and analyzed according to their disclosed radians. Mean electro-potentials during storage phase were 13.6±6.2, 23.2±11.8, 30.6±14.2 μV at 0.25π, 0.75π, or 1.0π respectively (F=86.94, P=0.000) as to the whole group. When the data were further analyzed according to their gender, age or original disease, this gradually increased trend still remained (Table 2). The result was that the potentials of the storage phase were influenced by the radians of the electrodes either in total, or in different genders, age subgroups or original diseases. There was a positive relation between them, either in total or divided by gender, age or original diseases. It could be ratiocinated that wire electrodes with three radian distances (0.25, 0.75 or 1.0 π) should be better than the CNE. These data suggest that the use of either 1.0, 0.75 or 0.25 π for use as an EMG standardization criterion will do and is warranted, and that the 1.0 π is the best. Whereas there were no significant differences during voiding phase in the mean electro-potentials between the patients with or without sphincter overactivity (Table 3). It seems

Fig. 3. The design of the experiment: an EMG cable with three interfaces (black, green and red, the green interface for grounding), three electrode adapters and three end needle-guided wires directly acupunctured into the anal sphincter at 3, 9, and 10:30 o’clock positions around anus (a), the EMG tracings during storage phase with radian of 1.0 π (b), 0.75 π (c), and 0.25 π (d) in a patient.
that the sphincters during the voiding phase are relaxed as loosely in patients with intact detrusor-sphincter relation and contracted as tightly in patients with sphincter overactivity as possible irrespective of distance or radian between the two electrodes (Fig. 4). Using Solar from MMS (Netherlands), we can also record simultaneously detrusor sphincter synergia or dyssynergia as Janus from Life-tech (USA) in same patients (Fig 4).

Table 2. Comparison of electro-potentials of anal sphincter at storage phase among different location radians of the electrodes μV, mean±SD (range)

| Variables          | Data of electro-potentials at different orientations (expressed in radians and degrees in parentheses) | F value | P value |
|--------------------|---------------------------------------------------------------------------------------------------|---------|---------|
|                    | 0.25 π (45°)                                                                                     |         |         |
|                    | 0.75 π (135°)                                                                                    |         |         |
|                    | 1.0 π (180°)                                                                                     |         |         |
| In general (n=152) |                                                                                                  |         |         |
| Data               | 13.60±6.20 (4.60-33.00)                                                                          | 23.20±11.82 (5.20-85.00) | 30.57±14.18 (6.40-127.6) | 86.94 <0.001 |
| Influence factor analysis | Sex                                                                                          |         |         |
| Male (n=79)        | 14.86±6.90 (6.20-33.0)                                                                          | 25.84±14.01 (9.8-85.00) | 32.49±16.61 (9.80-12.76) | 36.14 <0.001 |
| Female (n=73)      | 12.24±5.06 (4.60-24.2)                                                                           | 20.35±8.04 (5.20-45.00) | 28.48±10.70 (6.40-127.6) | 70.49 <0.001 |
| Age                |                                                                                                  |         |         |
| <30 (n=18)         | 14.36±4.58 (8.8-24.2)                                                                           | 26.96±11.54 (14.2-61.4) | 35.74±14.62 (20.2-79.6) | 16.95 <0.001 |
| 30~ (n=15)         | 12.43±6.77 (5.4-30.4)                                                                           | 22.32±10.22 (8.2-52.4) | 26.89±13.57 (9.8-52.8) | 7.36 <0.001 |
| 40~(n=22)          | 13.42±6.18 (7.0-33.00)                                                                          | 20.31±8.19 (8.8-41.4) | 29.37±12.48 (11.6-53.0) | 16.19 <0.001 |
| 50~(n=36)          | 13.81±6.24 (4.6-28.6)                                                                           | 24.05±15.34 (5.2-85.0) | 31.21±11.41 (6.4-55.0) | 20.42 <0.001 |
| 60~(n=20)          | 13.25±6.46 (5.0-28.2)                                                                           | 23.19±9.60 (7.8-39.2) | 28.46±8.14 (16.0-41.8) | 17.90 <0.001 |
| 70~(n=41)          | 13.79±6.75 (5.0-32.4)                                                                           | 22.69±11.72 (6.0-73.0) | 30.74±18.88 (13.2-127.6) | 16.38 <0.001 |
| Original disease   |                                                                                                  |         |         |
| BOO(n=52)          | 14.10±6.15 (6.2-32.4)                                                                           | 24.27±14.09 (9.8-85.0) | 31.93±17.18 (13.4-127.6) | 34.19 <0.001 |
| DV(n=57)           | 12.77±6.17 (5.0-33.0)                                                                           | 22.11±11.12 (5.2-61.4) | 28.86±12.83 (6.4-79.6) | 16.42 <0.001 |
| UAB(n=30)          | 14.54±6.65 (6.0-28.6)                                                                           | 22.82±10.53 (8.8-57.4) | 28.97±11.50 (9.8-57.8) | 24.24 <0.001 |
| SUI(n=13)          | 13.09±5.72 (4.6-21.4)                                                                           | 24.62±7.55 (12.4-37.6) | 36.26±11.23 (20.0-53.0) | 24.24 <0.001 |

BOO, bladder outlet obstruction; DV, dysfunctional voiding; SUI, stress urinary incontinence; UAB, underactive bladder.
### Table 3. Comparison of electro-potentials of anal sphincter at voiding phase among different location radians of the electrodes (μV, mean ± SD (range))

| Variables | Data of electro-potentials at different orientations (expressed in radians and degrees in parentheses) | \( F \) value | \( P \) value |
|-----------|-----------------------------------------------------------------------------------------------------------------|----------|----------|
| 0.25 \( \pi \) \((45^\circ)\) | 14.54±6.78 (4.0-34.0) | 16.91±6.93 (4.0-34.0) | 1.76 | 0.17 |
| 0.75 \( \pi \) \((135^\circ)\) | 15.55±7.90 (5.0-40.0) | 15.75±6.57 (4.0-31.0) | 1.85 | 0.16 |
| 1.0 \( \pi \) \((180^\circ)\) | 16.91±6.93 (4.0-34.0) | 21.54±6.57 (13.0-34.0) | 0.51 | 0.60 |

**Influence factor analysis (n=122)**

**Original disease**

| BOO (n=52) | 13.48±6.20 (4.0-30.0) | 15.75±6.57 (4.0-31.0) | 1.85 | 0.16 |
| SUI (n=13) | 18.77±7.57 (8.0-34.0) | 21.54±6.57 (13.0-34.0) | 0.51 | 0.60 |
| Sphincter overactivity DV (n=57) | 87.18±66.31 (7.0-300.0) | 102.79±80.55 (11.0-400.0) | 0.66 | 0.52 |

BOO, bladder outlet obstruction; DV, dysfunctional voiding; SUI, stress urinary incontinence; UAB, underactive bladder.

2.1.3 Use of TL value

To compare the values of EMG potential at different time, for example, before start of voiding or during maximal flow rate (i.e., \( Q_{\text{max}} \)), a derived parameter, TL value, was introduced (Xu, et al., 2007, 2010). TL value is a derived parameter used to measure the degree of detrusor-sphincter synergia. In digitalization terms, sphincter overactivity or dysfunctional voiding was expressed with a quantitative analysis of the potentials using the parameter of tense/loose (TL) value. TL value is derived from sphincter EMG as a format of \( \lg [\text{potentials before voiding}/\text{at } Q_{\text{max}}] \) with a positive number (>0) as normal level and a negative number (<0) indicating sphincter overactivity during voiding. By this parameter, information of sphincter EMG could be utilized maximally. However, this parameter is possible only after consecutive and satisfactory recording of EMG tracing alongside with CMG and PFS recording during monitoring of the storage and voiding process. In laboratories where EMG signals are recorded poorly or given up, this value is not available. Indirect parameters, such as \( P_{\text{det}}Q_{\text{max}} \) (≥20 cmH2O), \( Q_{\text{max}} \) (<12 mL/s) and increased EMG were used in the diagnosis of dysfunctional voiding (Wang & Chen, 2003). \( P_{\text{det}}Q_{\text{max}} \) means the detrusor pressure at maximum flow rate in the voiding phase. The diagnosis of dysfunctional voiding was made when there was an increased external sphincter activity during voluntary voiding in neurologically intact people, as evidenced by EMG tracing and/or fluoroscopy, with a sustained detrusor contraction (Carlson et al, 2001, Chuang & Kuo, 2007, Kuo, 2010). If the TL value is measured in these figures, a minus value will be obtained too.
Another special type of dysfunctional voiding is Fowler’s syndrome in women, which has three characteristic features: sphincter overactivity, detrusor underactivity, and polycystic ovary (Fowler et al., 1988; Fowler, 2003). In Fowler’s syndrome urinary retention is associated with a primary abnormality of the striated urethral sphincter, characterized by an abnormal sphincter EMG signal displaying ‘complex repetitive discharges’ and elevated maximum urethral closure pressure (MUCP). Many of the young women with this disorder manifest features of polycystic ovaries, suggesting that it is due to a hormonally sensitive channelopathy. A normal mechanism for suppression of incontinence involving the striated urethral sphincter becomes exaggerated in Fowler’s syndrome and prevents voiding. Reports of increased urethral pressure and sphincter volume corroborate the concept that inappropriate sphincter contraction causes the urinary retention, leading to detrusor failure with loss of bladder sensation (Wiseman et al., 2001). Six female patients with Fowler’s syndrome in whom sacral neuromodulation restored their ability to void underwent functional brain magnetic resonance imaging immediately after neuromodulation and when untreated (Kavia et al, 2010). The most striking results were the widespread negative responses to bladder infusion, which were quite different from the activations seen in ‘normal’ individuals. The function of visceral sensation in the right insula representing brain responses to bladder filling was abnormal (negative) when the bladders in the patients with Fowler’s syndrome were empty. Increase of bladder volume and neuromodulation treatment reduced the extent of negative neural responses, and normal (positive) responses in the periaqueductal grey strengthened, whether used alone or together. They concluded
that the primary abnormality of the syndrome is an overactive urethra that generates abnormally strong inhibitory afferent signals, so effectively blocking bladder afferent activity at the sacral level and deactivating the periaqueductal grey and higher centres (Kavia et al, 2010). This central reflex and sacral guarding reflex have the same nature. The constriction of the pelvic floor-external sphincter complex is a normal response to control urgent urination and results in a reflex inhibition of the detrusor. When this action becomes habitual with time or exaggerated, as in patients with dysfunctional voiding or Fowler’s syndrome, abnormal dyscoordination carries over to voluntary voiding, resulting in sphincter overactivity, intermittent urinary stream and post-void residual urine (McGuire, 1984, as cited in Carlson et al, 2001). We think a negative TL value is also a criteria for the diagnosis of Fowler’s syndrome. Abnormal EMG findings may imply neural, psychological, or behavioural dysfunction from central, peripheral, regional or reflexual origin. It certainly provides the directions for further research.

2.1.4 EMG, MUP and other neurophysiologic examination
Failure of the sphincter to relax or stay completely relaxed during micturition is abnormal. The inappropriate sphincter activity during voiding has a variety of patterns, ranging from crescendo contraction to failure of relaxation. In neurologic conditions, abnormal EMG waveforms, in addition to detrusor-sphincter dyssynergia, are seen. These include fibrillation potentials, complex polyphasic potentials, and complex repetitive discharges, but they require more specialized equipment for their demonstration (Aminoff, 2007; Nandedkar et al, 1986). Due to the limited utility of clinical examination (Agur et al, 2009; Warren et al, 2011), urodynamics and imaging studies to demonstrate a neurogenic etiology of the LUTD, the role of clinical neurophysiologic methods remains important. For evaluation of patients with neurogenic LUTD a number of neurophysiologic methods have been used, including motor unit potential (MUP), interference pattern, nerve conduction, evoked potential, and sacral reflex analysis (Finsterer, 2001; Podnar, 2007).

Nerve conduction studies are performed by the stimulation of a peripheral nerve and the monitoring of the time taken for a response to occur in its innervated muscle. The time from stimulation to response is termed the latency. Nerve conduction studies are tests of the integrity of a reflex arc and can be relatively sensitive indicators of the presence of neurologic disease. In urologic practice, these studies are most often performed as bladder-cortex reflex latency determinations. They require elaborate instrumentation and careful user interpretation. Abnormal responses occur in a variety of situations and are particularly diagnostic in patients with diabetes and peripheral neuropathies. In patients with conus medullaris or cauda equina lesions, normal, prolonged, or absent latencies may be found, and asymmetrical responses are not uncommon. Patients with suprasacral lesions may have normal or low latencies (26 to 30 msec) because of loss of inhibitory neural pathways from higher centers (Webster & Guralnick, 2002).

Evoked response studies are means to assess afferent neuronal pathways by applying a stimulus to a peripheral nerve (e.g., dorsal penile nerve/pudendal nerve) and recording the cortical response. As such, they are used to test the integrity of peripheral, spinal, and central nervous system pathways. They also require sophisticated instrumentation using averaging techniques, and their performance is confined to specialized centers (Aminoff, 2007).
Sphincter EMG derived from concentric needle electrodes comes from muscle fibres within 0.5 mm of tip of the needle. The analysis of the EMG data using this type of electrode shown on an oscillograph with a speed of 10 ms/div (much faster as compared with a speed of 10-20 s/div in urodynamic study) is known as MUP (Podnar et al, 2000; Podnar, 2007). Those from two wire electrodes inserted in the same position as the anal surface electrode come from half of the sphincter muscle fibres as whole. So the EMG trace is smooth and constant with strain or coughing-induced strengthening. If a MUP means electronic activity of a small group of motor units of the sphincter, then sphincter EMG gained from needle guided-wire electrodes means highly concentrated MUP. The former is just like a piece of roasted mutton, and the latter is a long string of roasted mutton. Three quantitative MUP analysis techniques (manual-MUP, single-MUP and multi-MUP), with similar sensitivities for detecting reinnervation changes, are available (Podnar et al, 2000; Podnar, 2007).

Normally, potentials generated during sphincter activity may be recorded with a specialized concentric needle electrode inserted directly into the muscle to be tested, and the MUP recorded from the distal urethral sphincter muscle has a biphasic or triphasic waveform with an amplitude of 50 to 300 mV and a firing frequency of 10 to 100 discharges per second. Simplistically, when the motoneuron or nerve to a muscle is damaged, those muscle fibers that have lost their innervation become reinnervated by adjacent healthy nerves. The resultant MUP changes from a simple waveform to one that is larger in amplitude, complexity, and duration; these are termed polyphasic potentials. At least five deflections on the tracing must be present for a MUP to be called polyphasic (Abrams et al, 1988). These are thought to represent the increased number of muscle fibers per motor unit that follows reinnervation. Normal muscle may have up to 15% of its activity in the form of such polyphasic potentials; however, when the amount of polyphasic activity is significantly greater than this, neuropathy is implied. Other findings that suggest neuropathy include fibrillation potentials, which are spontaneous, low-amplitude potentials of short duration, and positive sharp waves, which are biphasic potentials. Neurophysiologic studies require more sophisticated instrumentation and investigator expertise and are designed to actually diagnose and characterize the presence of neuropathy or myopathy. MUPs in health and disease differ, and, within certain limitations, the expert observer may use these studies to determine whether neuropathy is present. Neurophysiologic studies are beyond the expertise of most urodynamic laboratories and are uncommonly indicated. Their role is in diagnosis of occult neuropathy or myopathy. In the patient with overt neurologic findings who has bladder dysfunction, neurogenic bladder dysfunction can be deduced without further study. In such cases, a kinesiologic study to identify the pattern of dysfunction is all that is indicated. MUP studies find their role in the evaluation of the patient with bladder dysfunction of unknown cause in whom neuropathy is suspected. They are also used in medicolegal situations in an attempt to correlate voiding symptoms and sexual dysfunction with prior injuries.

Furthermore, MUP may be preferable for ongoing study on underactivity of detrusor or sphincter (Takahashi et al, 2000; Jiang et al, 2009a, 2009b, 2010). Today, this abnormality is associated with intrinsic sphincter deficiency (ISD) in patients with SUI, intermittent or constant urinary incontinence after spinal shock in patients suffered from spinal cord injury, multiple system atrophy (MSA) or multiple sclerosis. Unilateral needle EMG of the subcutaneous external anal sphincter muscle, including quantitative MUP analysis is clearly indicated in patients with suspected MSA, particularly in its early stages when the diagnosis is unclear. If the test is normal, but the diagnosis remains unclear, it might be of value to
repeat the test later (Libelius & Johansson, 2000, as cited in Podnar, 2007). The main urodynamic finding is lowered MUCP or Valsalvour leak point pressure in patients with SUI. There would be subtle myogenic injuries occurred in skeletal sphincter as Takahash found in patients with type 3 SUI (ISD) (Takahash et al, 2000). In order to determine the EMG features of the striated urethral sphincter in patients with type 3 SUI, Takahash et al performed EMG and MUP analysis of the striated urethral sphincter muscle and urodynamic studies in a total of 51 women, 41 female patients with type 3 SUI and 10 women with normal urinary control. MUP of SUI patients showed significantly shorter duration, lower amplitude, and larger number of phases compared with those in the control group. Thirty (73%) of the SUI patients showed an obvious low amplitude (less than 350 mV)/short duration (less than 4.5 milliseconds)/polyphasic pattern and early recruitment of interference activity with low amplitude at voluntary contraction of the striated sphincter, both indicating existence of myogenic damages. These patients showing myogenic damages had significantly lower Valsalvour leak point pressure and more leakage in the pad-weigh test compared with the SUI patients without myogenic damage findings. These results suggested that myogenic-dominant damages of the striated urethral sphincter may contribute to the etiology of ISD in most patients with type 3 SUI (Takahash et al, 2000).

2.2 Clinical consideration and EMG significance

It is very important to check the functions of detrusor-sphincter and their relationship with patients with LUTS. If the patients have both detrusor and sphincter intact and in harmony, treatment for other organic or functional disorder, for example, outlet obstruction and lower compliance, is then feasible. From this point of view, excellent measurement of EMG widens the scope of LUTS.

Kinesiologic or EMG studies do not diagnose neuropathy but may characterize its effects. They are indicated in any patient in whom there is a suspicion of discoordination between the sphincter and the bladder. Thus, patients with spinal cord injury, with neurologic disorders (e.g., Parkinson’s disease, spinal dysraphism, multiple sclerosis, and MSA), or with voiding dysfunction after radical pelvic surgery or spinal surgery; children with voiding dysfunction and upper tract changes; and young women with urinary retention are appropriate candidates. In this last group, there is evidence that involuntary muscle fiber activity preventing sphincter relaxation may have a hormonal etiology associated with polycystic ovarian disease (Fowler, 1988; Fowler et al., 2003). However, EMG should be interpreted in light of the patient’s symptoms, physical findings, and urologic and urodynamic investigations (Abrams et al., 1988; Goldman, 2006; Griffiths et al., 1997). Except in patients with an unusual voiding history, a standard neurologic evaluation of the perineum and lower extremities is usually adequate to exclude most neurologic problems (O’Donnell et al., 1998, as cited in Webster & Guralnick, 2002). Thus, there is a limited role for EMG or kinesiologic studies in the routine urodynamic evaluation of incontinent or obstructed patients in whom neuropathy is not suggested by other clinical findings. Furthermore, often video-urodynamic studies provide as much information as is needed in determining the coordination between bladder and sphincter (which can be viewed fluoroscopically) and allow management decisions to be made (Shah, 1994; McGuire et al, 1996, as cited in Webster & Guralnick, 2002). However, if detrusor is relevant to sphincter urodynamically, it is very helpful to use standard urodynamics to evaluate synergy or dyssynergia, in coordination or discoordination. We think video-urodynamics should be used for more sophisticated cases.
2.2.1 Baclofen as a useful agents for patients with dysfunctional voiding
In patients who complained of symptoms of frequency or urge may actually suffer from sphincter overactivity or dysfunctional voiding, to which baclofen (a GABA-ergic receptor agonist) may be administered as a rational option and obtain good response in this case. It was quantitatively analyzed using the TL value, which was successfully applied in a series of assessment. We had conducted a randomized double-blind placebo-controlled crossover trial in 60 women with dysfunctional voiding and LUTD from January 2003 to January 2006; patients were randomly assigned either baclofen 10 mg three times daily, then matching placebo for 4 weeks, or matching placebo then baclofen 10 mg three times daily for 4 weeks, separated by a 2-week washout period. Voiding diaries and multichannel urodynamics (at baseline, 4 and 10 weeks) were used to record the changes of voids/24 h and urodynamic variables. The efficacy analysis of the treatment showed that baclofen was associated with significantly fewer voids/24 h than placebo (mean difference from baseline 5.53 vs 2.70; \( P=0.001 \)) and a significant increase in TL value (mean difference from baseline, \(-1.78 \text{ vs } 0.01, \ P=0.001\)). A 4-week course of baclofen significantly reduced the number of voids/24 h and increased the TL value in women with dysfunctional voiding confirmed by anal sphincter EMG with wire electrode. Female patient with dysfunctional voiding (Fig. 5 a) received 4-week course of baclofen and her dysfunctional voiding disappeared at follow-up urodynamics (Fig. 5 b). These encouraging results suggest that baclofen could be used to treat dysfunctional voiding in women (Xu et al., 2007).

Fig. 5. A female patients aged 42 years old, who complained of urinary frequency for 3 years, was diagnosed as having dysfunctional voiding with a TL value of \(-0.65\) as indicated by dashed arrows (a), and her symptoms improved significantly with a TL value of 0.67 as indicated by solid arrows after 4 weeks of treatment with baclofen during the follow-up urodynamic evaluation (b).

2.2.2 Dysfunctional voiding associated with SUI in female patients with shortened urethral functional profile length and its impact on outcome of tension-free vaginal tape procedure
In order to determine the prevalence of dysfunctional voiding in female SUI and its modification after tension-free vaginal tape (TVT) procedure, three hundred and sixty
women with SUI were enrolled and underwent urodynamics from 2002 to 2008. Dysfunctional voiding was determined when non-neurogenic detrusor-sphincter dyssynergia occurred during voluntary voiding (Fig 6 b). It was further quantitatively analyzed using the tense/loose value, a parameter derived from external anal sphincter EMG. The distribution of other urodynamic variables was also evaluated. One hundred and fifty patients underwent the TVT procedure and forty of them were studied with urodynamics after anti-incontinence surgery of TVT during follow up. Overall, dysfunctional voiding was diagnosed in ninety-nine patients (Fig 6 b), with a prevalence of 27.5%. The functional profile length in SUI women with dysfunctional voiding was significantly shorter than that in SUI women without dysfunctional voiding (Fig 6 a) (3.13±0.76 vs 3.32±0.65, P = 0.017). After the TVT procedure, the recovery of SUI between cases with and without dysfunctional voiding showed no significant difference. The rate of dysfunctional voiding state change after the surgery, namely from with to without dysfunctional voiding or from without to with dysfunctional voiding, significantly differed between the female patients with and without dysfunctional voiding (66.7% vs 3.6%, P < 0.05) during follow up. The dysfunctional voiding improved after the surgery in SUI women with dysfunctional voiding. Dysfunctional voiding might represent a coexistent finding in women with SUI. The main difference of women with SUI and dysfunctional voiding, as compared with those without dysfunctional voiding, was a shortened functional profile length. In such cases, TVT procedure could improve dysfunctional voiding along with the treatment of SUI (Xu et al., 2010).

Fig. 6. Urodynamic studies on a female patient with stress urinary incontinence (SUI) without dysfunctional voiding as indicated by solid arrows (DV) (a) and another female patient with SUI and DV as indicated by dashed arrows (b) before the tension-free vaginal tape (TVT) procedure.
2.2.3 Timely selection of necessary surgical intervention for obstructed patients with decreased bladder compliance and intact detrusor contractility

Men older than 45 years old used to select conservative treatments (α-blockade, or 5α-reductase inhibitant, etc.) to treat their lower urinary tract obstructive symptoms, such as frequency, dysuria, and even nocturnal diuresis. If they complained of daily or nocturnal enuresis, the symptoms may be worse than before. This is a critical time point for them. Surgical intervention may be mandatory. Although the voiding reflex remains or detrusor is intact, the detrusor function has declined gradually since incontinence occurs. Between May 2002 and March 2009, a total of 4500 patients underwent urodynamic examination (Life-Tech Urovision Janus V) in this institute. Among them, 1500 male patients older than 45 years with obstructive symptoms were analysed. Among them, there were 15 patients with lower compliance and intact detrusor, and 15 patients with lower compliance and detrusor underactivity enrolled into this trial. Their urodynamics and surgical outcome were compared between the two groups. Routine endoscopic surgeries were carried out for them and those with lower compliance and intact detrusor gained better recovery thereafter as compared with those with detrusor underactivity (Xu et al., 2009) (Fig 7). The main improvement was disappearance of enuresis and increased peak flow rates during follow-up. The patients with detrusor underactivity improved slowly and 33% of them received self-catheterization or urodiversion operation at last. Male patients older than 45 years old complained of obstructive symptoms and nocturnal enuresis should routinely take comprehensive urodynamics. If they were diagnosed with decreased bladder compliance and intact detrusor, surgical intervention is a mandatory option. If they accept the surgical option, their prognosis is very well as compared with those with detrusor underactivity.

2.2.4 Lower compliance at second-half storage phase as main cause of hydroureteronephrosis in patients with diabetes insipidus

Nephrogenic diabetes insipidus with dilatation of upper urinary tract and bladder is rarely reported. Urinary tract dilatation and bladder dysfunction, usually in the form of a large, atonic bladder, are commonly believed to be secondary to high urine output. Low bladder compliance means an abnormal volume and pressure relationship, and an incremental rise in bladder pressure during the bladder filling. It is well known that at the time bladder capacity decreases, intravesical pressure increases, and the risk of upper deterioration increases. Hypocompliance is usually thought to be the range from 1.0 to 20.0 mL/cmH2O. Though the exact cause of hypocompliance is not known, it may be caused by changes in the elastic and viscoelastic properties of the bladder, changes in detrusor muscle tone, or combinations of the two (Park, 2010). The lower bladder compliance patterns could be classified into three groups (Cho et al., 2009). Group A (gradual increase) had the highest correlation with the presence of spinal cord injury. Group B (terminal increase) patients had a history of direct pelvic treatment such as radical prostatectomy and pelvic irradiation. Group C (abrupt increase and plateau) was positively correlated with the presence of detrusor overactivity and nocturnal enuresis. We found that children with polyuria, nocturnal enuresis and MRI-confirmed pituitary abnormality (hypointensities on T1-weighted MRI) and diabetes insipidus usually had hydroureteronephrosis, enlarged bladder capacity and lower bladder compliance at second-half storage phase. Their bladder compliance pattern belonged to terminal increase type as classified by Cho et al. Their detrusor and sphincter function had to be evaluated carefully as the first procedure. If the detrusor could contract and sphincter could relax during the voiding phase, the prognosis is good (Fig. 8), and vice versa. Che et al described 5 patients with nephrogenic diabetes
insipidus whose first presentation was bilateral hydroureteronephrosis and chronic renal insufficiency (Che et al, 2009). Between May 2005 and March 2009, 5 boys came to our clinic with complaint of polydipsia and polyuria (4), bilateral flank pain (2), and fatigue (2). Ultrasonic scan found bilateral hydroureteronephrosis in all 5 patients and blood creatinine test showed renal insufficiency. Fluid deprivation test were performed and according to the results they were diagnosed as nephrogenic diabetes insipidus. All patients were catheterized for 7 to 18 days till blood creatinine level decreased as normal. Urodynamics showed that the mean values of the bladder capacity, detrusor pressure at the mid and end of filling, maximum flow rate (Qmax), and PVR were 760 ml, 15.0 and 40.5 cm H2O, 30.8 ml/s, and 436.3 ml, respectively. These results reminded us all these patients had a lower compliance at second-half storage phase. Given that the detrusor and sphincter function well as displayed on EMG, operation or desmopressin are rational option for patients with

![Image](https://via.placeholder.com/150)

Fig. 7. A male patient aged 65 years old, who complained of poor-weak flow and urinary frequency for 5 years and nocturnal enuresis for the last 5 months, was confirmed as having bladder outlet obstruction and lower bladder compliance as indicated by dashed arrow, however, his detrusor and sphincter function was still intact with detrusor-sphincter synergia as indicated by solid arrows (a, b), and his symptoms at follow-up recovered 6 months after a successful TURP with a normal flow rate and compliance thereafter as indicated by solid arrow (c, d).
obstruction or diabetes insipidus. The patients had taken desmopressin acetate (0.1 mg, 1/d), emphatic and timed voiding for three months, and then all symptoms were improved slightly. And after a follow of 4 to 12 months, renal function remained normal and PVR was reduced. Nephrogenic diabetes insipidus should be considered in patients with dilatation of the urinary tract and polyuria. A lower compliance at second-half storage phase may contribute to the dilatation of urinary tract. Normal detrusor contractility with large PVR is a unique manifestation of this condition.

Fig. 8. A male patient aged 15 years old, complaining of pelvic pain and polyuria for more than 5 years, was found with hydroureteronephrosis and atrophy of the posterior lobe of the pituitary gland (hypointensities on T1-weighted MRI of the pituitary gland). His functional bladder capacity reached 800 ml, and lower bladder compliance was terminal at a bladder volume of 400 ml as indicated by dashed arrow, whereas his detrusor contracted and sphincter relaxed normally when he initiated a voiding reflex as indicated by solid arrows.

2.2.5 Validation of somatic-autonomic nerve anastomosis or artificial somatic-autonomic reflex pathway for the purpose of re-establishing bladder function
In the field of neurogenic LUTD, abnormal changes of EMG should not be neglected as well. So far different opinions exist about the so-called “artificial somatic-autonomic reflex pathway” for patients complained of dysuria and incontinence after spinal cord injury or with spina bifida. Whether the operation succeeds or not depends upon exhibition of detrusor contraction and still remaining of detrusor and external sphincter dyssynergia. Theoretically, this type of neuro-anastomosis could not reverse dyssynergia. There were three kinds of neuro-anastomosis for patients with neurogenic LUTD due to spinal cord
injury in China (with intercostals nerve: one author; with ventral roots of the lumbar or sacral nerves: two authors) (Fig. 9). They are: intercostal nerve anastomosed to pudendal nerve (Zhang & Zhao, 1993), T10 or T11 ventral root above the lesion anastomosed to the S2 ventral roots for complete conus medullaris injury with atonia bladder or detrusor underactivity; or S1 ventral root under the lesion anastomosed to the S2 or S3 ventral roots for hyperreflexic neurogenic bladder and external sphincter dyssynergia caused by complete suprasacral SCI (Lin et al., 2008a, 2008b, 2009, 2010); L5 ventral root under the lesion anastomosed to S2 or S3 ventral roots for hyperreflexic neurogenic bladder and external sphincter dyssynergia (Xiao et al., 2005; Xiao, 2006).

![Diagram](image)

**Fig. 9.** Creation of microsurgical anastomoses between T12 and S2 ventral roots (a). Drawing showing creation of microsurgical anastomoses between S-1 and S-2 ventral roots (b). Reproduced from Lin and Hou (2010, Neurosurgery 66: 948-952; 2009, Journal of Neurosurgery of Spine 10: 452-457) with permission.

We have shown the detrusor contraction and sphincter overactivity in some patients suffered from spinal cord injury who received a successful procedure of artificial somatic-autonomic reflex pathway (T10 anastomosed to S2) for bladder control in this institution. One of them with neurogenic detrusor underactivity and sphincter overactivity due to L1 fracture received operation of neuroanastomosis nine years ago. Her spontaneous voiding pattern recovered 1 year later and continued to be normal thereafter. Urodynamic follow-up data showed clearly detrusor contraction and some degree of external sphincter dyssynergia (Fig 10). Whereas in the papers of other authors, who used surface patch electrode, which was inferior to CNE for EMG documentation of urethral sphincter relaxation during voiding phase (Mahajan et al., 2006), the so-called “satisfactory voluntary voiding with detrusor contraction” could still be debated and improved (Xiao, 2006).
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Fig. 10. A female patient aged 46 years old, complained of paraplegia due to fracture of L1 for 9 years, urodynamic study before the procedure (a), 5 years (b) and 9 years (c) after successful artificial somatic-autonomic reflex pathway procedure showed that detrusor was underactive before the operation (as dashed arrows indicated), and after the procedure, detrusor became contractile and detrusor-sphincter dyssynergia still remained (as solid arrows indicated). Voluntary voiding and bladder emptying was satisfactory with detrusor contraction and without abdominal straining. EMG, electromyogram; I. Vol, infused volume; Pabd, abdominal pressure; Pdet, detrusor pressure; Pves, vesical pressure; Q, flow rate; Qvol, uro volume.

2.2.6 Detrusor underactivity and symptomless detrusor underactivity

Detrusor underactivity (DUA) is recently known as underactive bladder, which is a diagnosis made on a simultaneous PFS and EMG (Chancellor & Kaufman, 2008; Thomas, et al., 2005). Such condition is characterized by a low sustained or wave-like contraction and is associated with poor flow or no flow at all. The ICS defines DUA as “a detrusor contraction of inadequate magnitude and/or duration to effect complete bladder emptying in the absence of urethral obstruction” (Griffiths et al., 1997). During conventional urodynamic measurements, adequate emptying of the bladder, without a detrusor contraction is possible in two ways. Firstly, in females who can have a very good relaxation of the pelvic floor; in this case, hardly any detrusor contraction is needed for complete voiding (Fig. 11a). These patients are considered to be ‘normal’, or belong to symptomless detrusor underactivity. Secondly, in patients using abdominal straining without simultaneous sphincter relaxation to empty their bladder (Fig. 11b). These cases are considered to be ‘abnormal’. (van Koeveringe et al., 2009). In a comparative study, ambulatory urodynamic measurements/monitoring was conducted in 25 patients categorized as suspected acontractile bladder by conventional urodynamic measurements. Of the 25 patients with a suspected acontractile detrusor based on the conventional urodynamic measurements data, 21 patients had multiple detrusor contractions during voiding attempts on ambulatory urodynamic measurements during normal daily life activities. Four patients even showed overactive detrusor contractions on their ambulatory urodynamic measurements. This implies that the symptoms of these patients must have been due to other factors such as...
pelvic muscular nonrelaxation, psychological reasons or obstruction. Only in 17% (four of 24 cases) the acontractile bladder was confirmed by ambulatory urodynamic measurements. (van Koeveringe et al., 2009). In order to display abdominal straining, the anal catheter must be fixed firmly so as to avoid its exodus from the anus. The female patient had better to change the position from supine to sitting. Sitting position was more normal than supine. (Xu et al., 2009, a).

Fig. 11. Detrusor underactivity in women: a female patient aged 50 years old, complained of urine loss with cough/laugh for 3 months. She finished urination fluently even without detrusor contraction and her sphincter relaxation was complete as indicated by solid arrows (a), another female aged 73 years old, complained of poor-weak flow and pain with voiding for 4 years (b). She was found passing urine by using abdominal straining without simultaneous sphincter relaxation to empty their bladder (b).

In order to study the efficacy of low-frequency electrotherapy (LFE) for female patients with an early stage DUA due to neuromuscular deficiency, we have conducted a conservative treatment program to patients with DUA. Between April 2008 and April 2010, a total of 35 female patients with urodynamically confirmed DUA were subjected to LFE using an electrical stimulator (ES-420, Ito CO, Ltd. Tokyo, Japan). Patients received two treatment sessions (each lasting for 70 min) daily for two weeks. Patients were divided into DUA-NC (n=20, DUA with normal compliance) or DUA-LC (n=15, DUA with low compliance). Comprehensive urodynamic evaluations were performed in each patient prior to the LFE as the baseline and at 4 weeks following the procedure. At last, of the 20 patients with DUA-NC, 18 (90%) regained detrusor contractility after LFE (Fig. 12). The pattern of their detrusor contraction changed from low sustained contraction (in 5 cases), or wave-like contraction (13 cases) to normal parabola contraction. The percent of patients with DUA-NC who relied on
catheterization for bladder emptying decreased by 40% (from 50% to 10%, P<0.001). Those with DUA-LC decreased by only 6.7% (from 86.7% to 80%, P>0.05) and none regained detrusor contractility following LFE. LFE was more effective for patients with DUA-NC than for those with DUA-LC. Patients with DUA-NC due to an early stage neuromuscular deficiency benefited from LFE but those with DUA-LC did not benefit from this procedure.

![Fig. 12. Comprehensive urodynamic tracings of female patients with DUA-NC and the efficacy of LFE were displayed. The dashed arrows indicate normal compliance and solid arrows indicate the state of the detrusor contractility and the sphincter relaxing ability during voiding phase. (a) A women aged 32 years old, complained of chronic retention for 2 weeks due to iatrogenic reason, and her detrusor was still underactive when more than 500 ml saline was infused into the bladder. (b) After LFE, her detrusor function recovered, sphincter overactivity improved and got rid of catheterization thereafter. (c) A female patient, 52 years old, complained of urgency and frequency for two months, urodynamic study showed DUA and sphincter overactivity before LFE. (d) After the procedure, detrusor became contractile and sphincter overactivity still remained. EMG, electromyogram; I. Vol, infused volume; Pabd, abdominal pressure; Pdet, detrusor pressure; Pves, vesical pressure; Q, flow rate; Qvol, uro volume.]

2.2.7 Significance of symptoms and existing discrepancy between symptomatic and laboratory findings, and between symptomatic location and investigative location

There exists discrepancy between symptomatic and urodynamic findings in patients with LUTD. It is generally known that most epidemiological studies have relied on questionnaires to obtain widespread information about prevalence, symptoms, treatment usage and compliance; however, the results of such surveys must be interpreted with caution. So we should not let this prevent us from furthering our disease awareness via the use of physiological tests including urodynamics combined with sphincter EMG (Agur et
al., 2009). In October 2006 The National Institute for Health and Clinical Excellence (NICE) published guidance on the management of urinary incontinence in women. (NICE, 2006). The guideline states that “the use of multichannel cystometry is not routinely recommended before surgery in women with a clearly defined clinical diagnosis of pure SUI”. However, in a retrospective study of 6276 women with urinary incontinence, Agur W et al found that only 324 (5.2%) women had pure SUI; moreover, a quarter of those with pure SUI symptoms ultimately had urodynamic diagnoses other than urodynamic SUI, that could affect the outcome of continence surgery. These findings indicated that only a small group of women fulfilled the NICE criteria of pure SUI. These strict criteria did not ensure that all women with potentially important urodynamic findings were evaluated accordingly. The symptomatic assessment had a sensitivity of only 11.4%, and specificity of 98%. There were 1866 false negative cases with a negative predictive value of 69%, including voiding dysfunction (506 cases, 24.0%), overactive bladder wet (OAB wet) (1209 cases, 57.4%), OAB dry (151 cases, 7.2%) and 83 false positive cases with a positive predictive value of 74%, including normal (35 cases, 10.8%), voiding dysfunction (4 cases, 1.2%), and detrusor overactivity (44 cases, 13.6%). (Agur et al., 2009). Furthermore, even the symptom location of the patients is usually very factitious and sometimes vague and so clinical investigation is necessary. What characteristics of the pain caused patients with interstitial cystitis/painful bladder syndrome to perceive that it involved the bladder was wondered by many surgeons. Recently, preliminary work for a RAND (Santa Monica, CA) prevalence study of interstitial cystitis/painful bladder syndrome revealed difficulty in finding a single symptom-based definition of interstitial cystitis/painful bladder syndrome with both sufficient sensitivity and specificity to separate patients with interstitial cystitis/painful bladder syndrome from those with physician-diagnosed OAB, vulvodynia, or endometriosis. (Berry, et al., 2010, as cited in Warren et al., 2011). The symptoms of other syndromes, for example, irritable bowel syndrome, and systemic syndromes, such as fibromyalgia, do overlap those of interstitial cystitis/painful bladder syndrome. (Warren et al., 2009, as cited in Warren et al., 2011). Symptoms are the heralds of a disease that prompt a patient to seek medical care. It then is the task of the patient and clinician to reveal a sufficient description of the symptoms to prompt an appropriate diagnosis of the disease. If symptoms overlap to a large degree, it would imply that the distinction among these various syndromes is rather arbitrary. An extrapolation of this logic to the pelvic and systemic syndromes we have noted suggests that a similarity of symptoms might have only nosologic implications; if boundaries cannot be identified between syndromes, are they different diseases? Based on above-mentioned data, in order to locate the origin of symptoms and to validate the nature of the disease, necessary examination, including urodynamics and EMG, had to be carried out.

3. Conclusion

Anal sphincter EMG is an indispensable parameter for diagnosis and treatment option for patients with LUTD or pelvic floor dysfunction. It could present functional state of the urethral sphincter, and further replace urethral sphincter EMG thereafter. According to our experience, nearly 70%-80% so-called “artifacts” or bad recordings of EMG came actually from an open circuit, not from a closed circuit, of the electrocircuit system. We must make
every effort to record an excellent EMG trace for the purpose of developing evidence-based medicine as well as performance-based medicine, not only based on experience-based medicine.

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