Committee 7 A

Clinical Neurophysiological Tests

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ABBREVIATIONS

BCR	bulbocavernosus reflex
CMAP	compound muscle action potential
CMCT	central motor conduction time
CNEMG	concentric needle electromyography
EAS	external anal sphincter
ED	erectile dysfunction
EMG	electromyography
GSI	genuine stress incontinence
IP	interference pattern
MEP	motor evoked potential
MSA	multiple system atrophy
MU	motor unit
MU	motor unit potential
PD	Parkinson's disease
PNTML	pudendal nerve terminal motor latency
QST	quantitative sensory testing
SEP	somatosensory evoked potential
SFEMG	single fibre electromyography
SSR	sympathetic skin responses
SUI	stress urinary incontinence
T/A	turns/amplitude

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I. INTRODUCTION

Neurophysiological investigations of muscles and nerves in the perineum and pelvis originated over 60 years ago, and have evolved with the developments in general clinical neurophysiology. The data from these investigations can assist clinicians in diagnosing neurological disease or injury, and are applicable in research. Compared to neurophysiological testing of the limbs and trunk, pelvic neurophysiological testing is relatively limited because of the restrictions imposed by pelvic neuroanatomy.

This text details the investigations, their applications and limitations, enabling investigators and clinicians to make a well informed decision about using these tests. The present text is based on the previous chapter on clinical neurophysiology prepared for the International Consultations on Incontinence [1], which has been updated by a literature search in Medline using key words incontinence, clinical neurophysiology, electro-myography, reflex, evoked potentials.

1. CLASSIFICATION OF CLINICAL NEUROPHYSIOLOGICAL TESTS

Although different types of tests may be included under the term “neurophysiological”, it is particularly the electrophysiological tests that shall be discussed in the present text.

Electrophysiological tests are an extension of the clinical examination, and a functional anatomic approach to classification makes most sense. For the purpose of this categorisation, the nervous system is divided into the somatic and the autonomic nervous systems. The somatic nervous system provides motor innervation to the skeletal muscles, and sensory innervation from skin and muscle spindles. The autonomic nervous system provides motor innervation to the viscera and other end-organs not under voluntary control (e.g., sweat glands). Its sensory fibres are referred to as visceral afferents. Both systems have central pathways (neurons participating in spinal cord and supraspinal control) and peripheral nerves (those going to and from end-organs).

Thus, electrophysiological tests can be divided into: a) somatic motor system tests (EMG, terminal motor latency measurements/ motor nerve conduction studies, and motor evoked potentials (MEP)); b) somatosensory system tests (sensory neurography, somatosensory evoked potentials (SEP)); c) reflexes; and d) the autonomic nervous system tests (for sympathetic or parasympathetic fibres).

Electrophysiological tests may also be categorized “technically” into those which “just” record some bioelectrical activity (for instance: electromyography), and those which record some biological response to stimulation (these may be subsumed under the term “conduction tests”).

2. BIOLOGICAL CORRELATES OF ELECTROPHYSIOLOGICAL TESTS

a) Conduction Tests: Nerve Conduction, Evoked Potential and Reflex Studies

The electrophysiological responses obtained on stimulation are compound action potentials and relate to populations of biological units (neurons, axons, motor units, muscle fibres, etc.). Latency and amplitude are commonly measured parameters of responses during neurophysiological testing. If the onset of the potential is measured, the latency of a compound potential represents the fastest conduction through a particular neural channel. As a general rule, latency measurements are not markedly affected by technical factors, but provide little information about the loss of biological units (e.g., motor neurons or axons).

The amplitude of the compound potential correlates with the number of activated biological units. In theory, the amplitudes are the more relevant physiological parameter, as they reflect the functional or structural loss of biological units. Unfortunately, amplitudes are also strongly influenced by many poorly controllable technical factors. Measurements of latencies and amplitudes of evoked potentials and reflex responses, including sympathetic skin responses, relate not only to conduction in peripheral and central neural pathways, but also to trans-synaptic transmission.

b) Electromyography (EMG)

Knowledge of the structure and function of the motor unit (**Figure 1**) is fundamental to understanding the application of EMG. Motor neurons, which innervate striated muscle, lie in the anterior horn of the spinal cord and are called "lower motor neurons". (Neurons that innervate the sphincters lie in Onuf's nucleus in the sacral spinal cord; they are somewhat smaller than those innervating skeletal limb and trunk muscles).

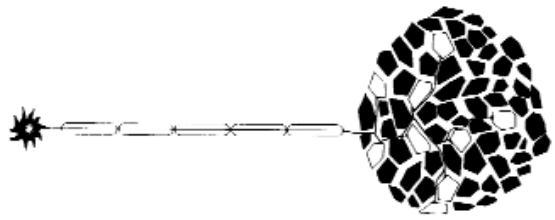


Figure 1 : Schematic representation of a motor unit. The alpha motor neuron with its cell body, its myelinated axon and the peripheral nerve endings is shown. The muscle fibres innervated by this alpha motor neuron are shown in white. (Note that the muscle fibres from one motor unit are intermingled with motor fibres from other motor units).

Within the muscle, the motor axon branches to innervate a certain number of muscle fibres, which are scattered throughout the muscle. All muscle fibres innervated by one lower motor neuron are activated simultaneously; all these constituents together are called "motor unit". The innervation of muscle fibres is such that it is unlikely that muscle fibres that are part of the same motor unit will be adjacent to one another.

It is difficult to estimate the number of muscle fibres innervated by a single axon (i.e., the "innervation ratio") or the number of motor units supplying a muscle, by clinically available neurophysiological techniques.

3. GENERAL METHODOLOGICAL CONSIDERATIONS

To date, there are no universally accepted standards for conducting individual uro-genital-anal neurophysiological tests, but the variations of testing in different laboratories are minor.

There are technical standards on equipment safety ; standardisation of concentric needle EMG and penilo/clitoro-cavernosal testing has been proposed.

a) Equipment

Clinical neurophysiological tests are conducted with complex electronic instruments and various devices that come into contact with the patient. Though this equipment is mostly standard, some specially constructed electrodes or stimulating devices have been devised to conform to uro-genito-anal anatomy. As long as the standards of electrical safety are adhered to, the risk to patients is negligible.

The common form of neurophysiological testing is electrophysiological. Surface electrodes, which are applied to skin or mucosal surfaces, or needle electrodes are used for electrical stimulation and to record bioelectrical activity. The important neurophysiological difference between surface and needle electrodes is their selectivity, and the practical difference is their invasiveness. The choice and application of electrodes is guided by the need for selective recording or stimulation. Less commonly, special devices are used for magnetic and mechanical stimulation.

Stimulation Parameters. The electrical stimulus should be specified and characterised both in technical (e.g., rectangular pulse, 0.2 ms, 15 mA) and physiological terms (e.g., 3-times sensory threshold). A stimulus with defined technical parameters may have variable biological effects because of the variable influences of electrode condition, contact, tissue conductivity etc. Supramaximal stimulation is preferred to elicit a compound muscle action potential (CMAP) or sensory nerve action potential. Supramaximal stimuli yield responses with the largest amplitude and shortest latency, and are the least variable and most reproducible. The sites at which stimulation electrodes are applied should be described using anatomical terms.

b) Recording

1. Apparatus settings

For recording, the apparatus settings (gain, sweep speed) have to be adapted to the known range of amplitudes, latencies, and duration of the response and it has to be appropriately displayed for analysis. Particularly important is the frequency setting of filters: for surface electrode recordings it is typically 2 Hz – 1 kHz; for concentric needle EMG recordings, it is 5 Hz – 10 kHz.

Placement of electrodes on the scalp for evoked potential recordings is defined according to the 10-20 International EEG System.

2. Reproducibility and Reliability

Any potential elicited by stimulation should be reproducible; therefore, as a rule, at least two consecutive recording procedures need to be performed. To improve the signal-to-noise ratio some small amplitude responses need to be averaged. Therefore, many repetitions of stimulation/recording need to be done (typically 100-200). Even such an averaged recording needs to be repeated at least twice. CMAPs (i.e., M-waves), MEP, sacral reflexes and sympathetic skin responses (SSR) are recognisable after single stimuli. However, as a rule, several responses are recorded to demonstrate reproducibility. In contrast, other responses (e.g., SSR) show marked fatigability with stimulus repetition.

3. Waveform Analysis

For a particular stimulation procedure, the shape, latency, and amplitude of the recorded potentials are analysed. Morphologically, a particular response (or part of it) needs to be recognised as present or absent. The shape of potentials is important to accurately determine the latency and amplitude of the response. The onset of the response (for M-waves, MEP and sacral reflex testing) or the individual peaks of the potentials (for SEP) are used to determine the latency. The amplitudes are analysed relative to the baseline or "peak to peak".

II. CLINICAL NEUROPHYSIOLOGICAL TESTS

1. SOMATIC MOTOR SYSTEM TESTS

a) Electromyography (EMG)

The term "EMG" is often used for several different procedures, the common denominator of which is the recording of bioelectrical activity from muscle. The term applies particularly to recordings from striated muscles.

EMG is used a) “just” to record muscle activity (as for instance in combined sphincter EMG and pressure-flow study) and b) to differentiate between normal, denervated, reinnervated, and myopathic muscle. For a) see below - “Kinesiological EMG”.

Although EMG abnormalities are detected as a result of a host of different lesions and diseases, there are in principle only two standard manifestations which can occur: a) disease of the muscle fibres themselves ("myogenic" changes), and b) changes in their innervation ("neuropathic" changes). Myogenic changes may result from muscle disease, probably also from direct trauma (e.g., the anal sphincter tear during vaginal delivery). Neurogenic changes may be attributable to injury at any level along the lower motor neuron supplying the external anal sphincter, extending from the motor neuron body, sacral nerve roots to the small branches within the external sphincter. In the pelvic floor muscles, only neurogenic changes are well recognised and routinely evaluated.

The EMG signal may be further used to indicate that muscle has been activated through its motor nerve, either by stimulation applied to motor pathways (M-wave, MEP) or to sensory pathways (reflex response).

1. General Technique for Needle EMG in Pelvic Floor Striated Muscles

All tests requiring needle electrodes are invasive and some pain is inevitable, even with use of local anaesthetics. Local anaesthesia is infrequently used for needle EMG examination. Intramuscular electrodes need to be appropriately placed in the target muscle. The pelvic floor and perineal muscles can be examined, including the levator ani, the bulbocavernosus muscle and the striated anal and urethral sphincter muscle. Facility with needle examination requires some practice. As a rule, several sites from one or more skin penetrations are sampled, which is difficult in small muscles.

The audio output from the loudspeaker of the EMG apparatus helps in assessment of the quality of recording as well as in recognition of the electrophysiologic phenomena.

2. Concentric needle EMG (CNEMG)

The examination is conducted with a single use, disposable electrode. The commonly used amplifier filter settings for CNEMG are 5 Hz – 10 kHz, and need to be defined if MUP parameters are to be measured, as are filter settings employed during data acquisition.

The concentric needle electrode consists of a central insulated platinum wire inserted through a steel cannula and the tip ground to give an elliptical area which can record spike or near activity from about 20 muscle fibres [2]. The number of motor units recorded therefore depends both upon the local arrangement of motor units within the muscle fascicle and the level of contraction of the muscle.

CNEMG can provide information on a) insertion activity, b) abnormal spontaneous activity (**Figure 2**), c) MUPs, and d) interference pattern (IP).

In normal muscle, needle movement elicits a short burst of "insertion activity," which is due to mechanical stimulation of excitable muscle cell membranes. This is recorded at a gain setting of 50 µV per division (sweep speed 5 – 10 ms/division), which is also used to record spontaneous activity. Absence of insertion activity with appropriately placed needle electrode usually means a complete denervation atrophy of the examined muscle.

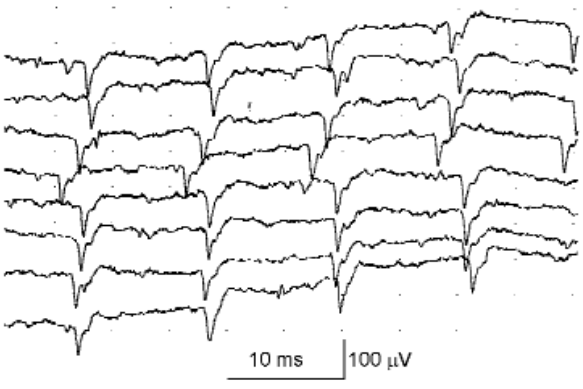


Figure 2 : Concentric needle EMG recording from right bulbocavernosus muscle of a 49-years old male with urinary incontinence diagnosed as possible Multiple system atrophy. Pathological spontaneous activity (a burst of positive sharp waves) is shown.

The amount of recruitable motor units during voluntary and reflex activation can also be estimated. Normally, MUPs should intermingle to produce an "interference" pattern on the oscilloscope during muscle contraction, and during a strong cough. In addition, the number of continuously active MUPs during relaxation [3], MUP variability as well as MUP recruitment on reflex and voluntary activation can be observed [4].

MUPs (and occasionally encountered end-plate activity) are recordable in normal resting sphincter muscles in a relaxed subject. This is in contrast to limb muscles where relaxation is associated with “electrical silence” by EMG. In addition to continuously firing motor units, new MUPs are recruited voluntarily and reflexly in the sphincters. It has been shown that the two MUP populations differ in their characteristics: reflexly or voluntarily activated “high-threshold MUPs” being larger than continuously active "low-threshold MUPs". As a consequence, standardised level of activity at which a template based multi-MUP analysis obtains 3-5 MUPs on a single muscle site was suggested [5]. In partially denervated sphincter muscle there is – by definition – a loss of motor units (MUs). This can be estimated during relaxation by counting the number of continuously firing low-threshold MUPs. In patients with cauda equina or conus medullaris lesions, fewer MUPs fire continuously during relaxation [6], probably due to partial axonal loss. The main obstacle to qualified assessment of reduced number of activated MUs and activation of MUs at increased firing rates (as occurs in limb muscles) is a lack of concomitant measurement of level of contraction of the examined muscle (this can be readily assessed when studying limb muscles).

There are two approaches to analysing the bioelectrical activity of motor units: either analysis of individual motor unit potentials (MUPs), or analysis of the overall activity of intermingled MUPs. (This is the so called "interference pattern" – IP. Exploring different sites of the activated muscle with a needle electrode provides “samples” of intermingled motor unit potentials (IP epochs), which can be analysed).

Generally three different techniques of MUP analysis (manual-MUP, single-MUP and multi-MUP) and 1 technique of IP analysis (turn/amplitude – T/A) are available on advanced EMG systems [6].

It is easy to grasp the “motor unit potential analysis”, as it is simply a measurment (by different methods) of the “parameters” of single individual MUPs (ie. its amplitude, duration, number of phases…). The changes in MUP parameters furthermore are “direct” results of understandable physiological changes, and are thus “meaningful” to the interpreter.

The changes in IP parameters are, however, less readily grasped. These are: numbers of turns per second (any peak or trough of the signal where the activity changes by more than 100 μV); amplitude/turn (change in volts between two turns); number of short segments (parts of signal that has “sharp” activity) percent activity (percent of epoch with sharp activity); envelope (peak to trough amplitude exceeded by 1% of peaks/troughs). These parameters relate both to MUP parameters and to the activation level of the muscle. Recorded data are log transformed and linear regression lines are created. Amplitude/turn, and number of turns/second data from normal subjects can be used to create upper and lower boundaries (95 % confidence intervals) for assembly of future data from individual patients. Individual data create a scatter plot (“cloud”) which compares to the normative boundaries (**Figure 3**). It has been asserted that this approach does not require a standardized muscle contraction, but the shape of the “cloud” is dependent on the strength of muscle contraction. Therefore it has been suggested to standardize the method by measuring pressure exerted by the contracting sphincter [7].

Both the template based multi-MUP analysis of MUP and T/A analysis of IP are fast (5-10 and 2-3 minutes per muscle, respectively), easy to apply, and, technically, represent clinically useful techniques.

i) CNEMG Findings due to denervation and reinnervation

After complete denervation, all motor unit activity ceases. In a denervated muscle, complete "electrical silence" is noted in the first days after such an event. The diagnosis of complete denervation is confirmed by the absence of muscle response during electrical stimulation. Because motor axons take days to degenerate after injury, this proof is not available for up to 5-7 days after a denervation injury. However, it is rarely necessary to demonstrate complete denervation in the acute stage because the clinical condition is usually obvious. Denervated muscle fibres become hyperexcitable and start to fire spontaneously giving rise to abnormal spontaneous activity, but these may take up to three weeks to appear. The "insertion activity" becomes prolonged and short biphasic spikes (fibrillation potentials) and biphasic potentials with prominent positive deflections (positive sharp waves) appear (Figure 2). Thus, concentric needle EMG (CNEMG) correlates of denervation are pathologically prolonged insertion activity and pathological spontaneous activity. Completely denervated muscle may be reinnervated by axonal regrowth from the proximal nerve stump with few muscle fibres constituting "nascent" motor units. These are short, bi- and triphasic, soon becoming polyphasic, serrated and with prolonged duration. In partially denervated muscle, collateral reinnervation takes place. Surviving motor axons will sprout and grow out to reinnervate those muscle fibres that have lost their nerve supply. This results in a change in the arrangement of muscle fibres within the unit. Whereas in healthy muscle, it is unusual for two adjacent muscle fibres to be part

of the same motor unit, following reinnervation, several muscle fibres belonging to the same motor unit come to be adjacent to one another. CNEMG correlates are changes in MUPs (duration, amplitude, number of phases, turns, etc). Early in the process of reinnervation, the newly outgrown motor sprouts are thin. Therefore, they conduct slowly such that the time taken for excitatory impulses to spread through the axonal tree is abnormally prolonged. Moreover, the neuromuscular transmission is unstable due to immaturity of the motor end-plates. The CNEMG correlate is instability of long-duration complex potentials.

In partially denervated muscle, some MUPs remain and mingle eventually with abnormal spontaneous activity. Changes due to collateral reinnervation are reflected by: prolongation of the wave form of the MUP (**Figure 3**) which may have small, late components ("satellite potentials"). MUPs show "instability" due to insecure transmission in newly formed axon sprouts and end-plates. This "instability of potentials" (meaning both "jitter" and "blocking" of individual components in a complex potential) is not routinely assessed during sphincter EMG. Nonetheless, it can be a helpful parameter, and may be evaluated not only by SFEMG, as originally described [8], but also by CNEMG, if a low frequency cut-off filter of 0.5 (up to 2) kHz is used along with a trigger – delay unit. In skeletal muscle, the diameter of reinnervating axonal sprouts and conduction velocity increase with time, thereby improving synchrony of activation in the reinnervated motor unit. Thus MUP amplitude increases while MUP duration reverts towards normal. However, in degenerative neurological diseases (such as multiple system atrophy), long duration motor units are a prominent feature of anal sphincter reinnervation [9]. It is important to note that in patients with more severe neurogenic lesions, reinnervation may be inefficient resulting in MUP with parameters below confidence limits describing size (area, duration) [10].

The changes in MUP parameters (along with changed number of MUPs and changes in activation frequency of MUPs) will be reflected also in IP parameters.

Abnormalities of parameters evaluated by needle EMG are in principle non-specific, i.e. most abnormalities can occur both in neuropathic or myopathic conditions. It is the overall clinical picture that dictates interpretation of results.

ii) CNEMG of the External Anal Sphincter

The external anal sphincter (EAS) is the most practical indicator muscle for sacral myotomes because it is easy to access, has enough muscle bulk for exact EMG analysis, and its examination is not too uncomfortable.

The needle electrode is inserted into the subcutaneous EAS muscle about 1 cm from the anal orifice, to a depth of a 3-6 mm under the non-keratinised epithelium. For the deeper part of the EAS muscle 1-3 cm deep insertions are made at the anal orifice, at an angle of about 30° to the anal canal axis [5]. In most patients only examination of the subcutaneous EAS muscle is necessary. Separate examinations of the left and right EAS muscles are recommended. The needle is inserted into the middle of the anterior and posterior halves of each side ("quadrants") of the EAS muscle. After insertion in two positions on each side the electrode is turned backwards and forwards in a systematic manner. At least 4 sites in each of the subcutaneous and/or the deeper EAS muscle are thus analysed [5, 11].

Use of quantitative MUP and IP analyses of the EAS is further facilitated by the availability of normative values [11] that can be introduced into the EMG systems' software. It has been shown that normative data are not significantly affected by age, gender [11], number of uncomplicated vaginal deliveries [12], mild chronic constipation [12], and the part of EAS muscle (i.e. subcutaneous or deeper) examined [12].

Intramuscular electrode insertion into other perineal muscles and pelvic floor muscles is not standardized and is described in textbooks and primary literature.

3. Single fibre EMG (SFEMG)

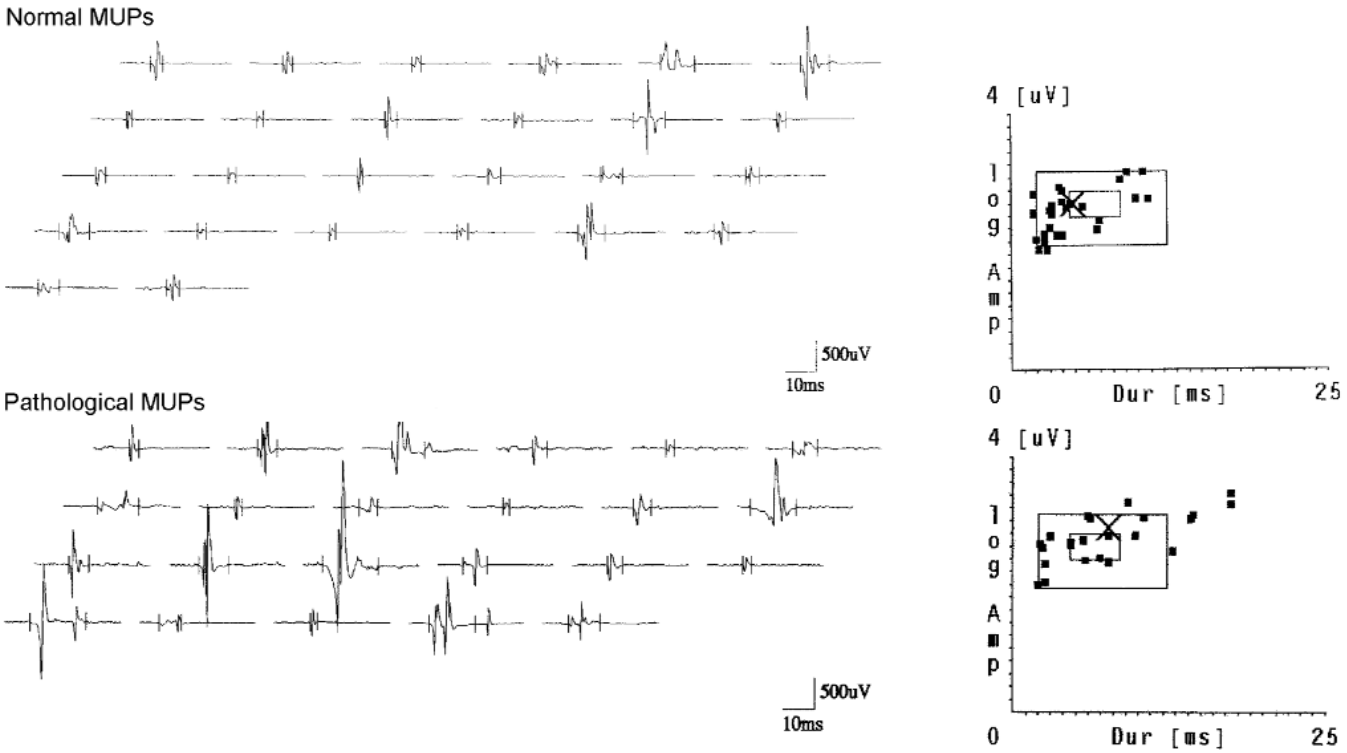
The SFEMG electrode has similar external proportions to a concentric needle electrode, but with a smaller recording surface. It will pick up activity from within a hemispherical muscle volume 300 µm in diameter, much smaller than the volume of 2-3 mm diameter from which a concentric needle electrode records [2]. Because of the arrangement of muscle fibres in a normal motor unit, a SFEMG needle will record only 1-3 single muscle fibres from the same motor unit.

The SFEMG parameter that reflects motor unit morphology is fibre density, which is defined as the mean number of muscle fibres belonging to an individual motor unit per detection site. To assemble this data, recordings from 20 different intramuscular detection sites are necessary [8].

SFEMG recording needles are very expensive, and disposable needles are not available.

4. Kinesiological EMG

Kinesiological EMG is used to assess patterns of individual muscle activity/inactivity during defined manoeuvres (**Figure 4**), typically during urodynamics.



Anal sphincter muscle	#	Amp uV	Amp rel.SD	Amp <min	Amp >max	Dur ms	Dur rel.SD	Dur <min	Dur >max	Area uVms	Area rel.SD	Area <min	Area >max	Poly %
Normal	24	236	-1.6	1	0	5.0	-0.5	1	0	215	-1.0	2	0	8
Pathological	26	393	0.1	2	0	4.8	-1.8	2	0	317	-0.4	2	0	15

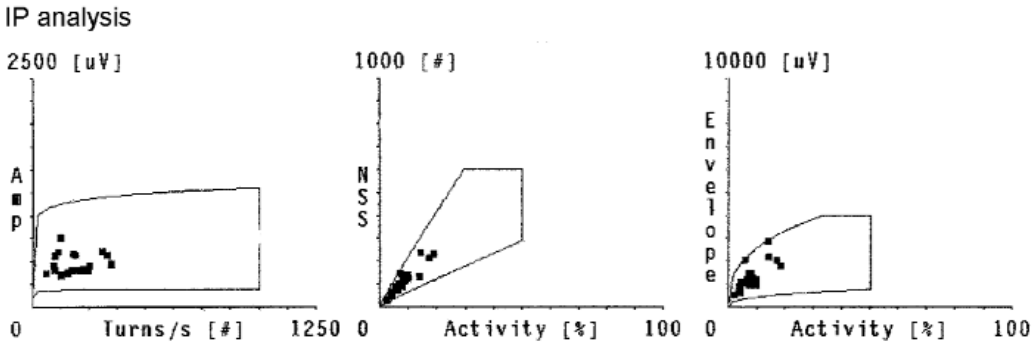


Figure 3 : Consecutive firings of motor unit potential (MUP) and its average (left and right, respectively) as obtained from the external anal sphincter (EAS) muscle of 59-year-old woman by multi-MUP analysis. Note that multi-MUP analysis does not preclude inclusion of late components into MUP duration measurement; this is possible by manual correction of duration cursor (see arrow).

As such, the specific interpretation of electrical activity within a muscle is based on its presence or absence, rather than the type of activity. Technical issues will be dealt here; the relevance for diagnostics will be discussed in the Chapter on dynamic testing.

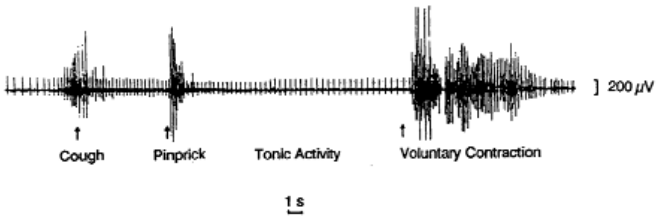


Figure 4 : Kinesiological EMG recording from the urethral sphincter muscle of a healthy 53 years old continent female. Recruitment of motor units on reflex manoeuvres and on a command to contract is shown; regular continuous activity of motor units represents "tonic activity". (Recorded with concentric needle electrode).

When using surface electrodes there are problems related to validity of signal (e.g., artefacts, contamination from other muscles). With intramuscular electrodes, the procedure is more invasive, and there are questions as to whether the whole muscle in large pelvic floor muscles is properly represented by the sampled muscle portions. Intramuscular electrodes should ideally be fine wire electrodes, as they do not dislodge, and no pain is induced with muscle contraction.

The kinesiological sphincter EMG recordings in health show continuous activity of MUPs at rest. It can be recorded in many but not all detection sites of the levator ani muscle. The urethral and anal sphincter as well as the other pelvic floor musculature (e.g. pubococcigei) can be voluntarily activated typically for less than 1 minute [13]. Timely activation of the levator ani muscle has been demonstrated to be an important aspect of stable bladder neck support; its activation precedes activity of other muscles in the cough reflex [14].

Sphincter activity during voiding is characterised by the cessation of all EMG activity prior to detrusor contraction. Pathologic incoordination of the detrusor and sphincter is called detrusor sphincter dyssynergia.

5. Clinical application of EMG

i) Neurogenic Conditions

Trauma, surgery, and neurological disease have all been implicated in denervation of pelvic floor and perineal muscles and pelvic organs.

Following a cauda equina or a conus medullaris lesion, the MUP of pelvic floor and perineal muscles are prolonged and polyphasic, of increased amplitude, area, number of turns [6]. Surgical dissections can also affect the innervation of the sphincter and lead to loss of motor units and reinnervation of those surviving [15]. After pelvic trauma, gross changes of denervation and reinnervation may be detected in pelvic floor muscles. The bulbocavernosus muscle is particularly useful in suspected recent minor partial denervation as it lacks on-going activity of low-threshold MU during relaxation. (In women the muscle is thin).

Neuropathic changes can also be recorded in sphincter muscles of patients with multiple system atrophy (MSA) [16]. MSA is a progressive neurodegenerative disease, which can be mistaken for Parkinson's disease (PD). Urinary incontinence and erectile dysfunction occur, often some years before the onset of obvious neurological features [17]. Sphincter EMG has been used to distinguish MSA from Parkinson's disease. EMG is probably not helpful to distinguish MSA from the later stages of Parkinson's disease and from progressive supranuclear palsy. Extensive discussion on the subject can be found elsewhere [18].

In patients with acute idiopathic autonomic neuropathy and lower urinary tract (LUT) dysfunction the EMG of external sphincter muscles was reported as normal [19].

ii) "Changes in Primary Muscle Disease

In skeletal muscle, the "typical" features of a myopathy are small, low amplitude polyphasic units recruited at mild effort. There are few reports of pelvic floor muscle EMG in generalised myopathy. In a nulliparous woman with limb-girdle muscular dystrophy, histology revealed involvement of pelvic floor muscles, but concentric needle EMG of the urethral sphincter was normal [20]. Myopathic EMG changes were observed in the puborectalis and the EAS in patients with myotonic dystrophy (21), but not in another group of patients with myopathy [22].

iii) Stress Incontinence

Pelvic floor muscle denervation has been implicated in the pathophysiology of USI [23]. EMG techniques have been used to identify sphincter injury after childbirth and to evaluate women with USI. Stress incontinence and genitourinary prolapse were associated with partial denervation of the pelvic floor [24]. The changes were most marked in women who were incontinent after delivery, who had a prolonged second stage of labour, and had given birth to heavier babies.

Myogenic histological changes in pelvic floor muscles after vaginal delivery were also reported [25], with some EMG support by another group [26]. Myopathic EMG changes (i.e. short, small MUPs) may, however, be a consequence of deficient reinnervation [27]. There were claims urethral sphincter EMG can assist in selecting the type of surgery for patients with intrinsic sphincter deficiency [25].

Although CNEMG of the urethral sphincter seems the logical choice in patients with urinary incontinence of possibly neurogenic origin, only a small amount of pathological muscle tissue remains in many incontinent parous women which makes EMG of the muscle impractical [15]. CNEMG findings generally will not affect therapeutic considerations [28].

iv) "Idiopathic" Faecal Incontinence

"Idiopathic" faecal incontinence refers to patients in whom this symptom is not attributable to an underlying disorder, but it has been often implied that it is a neurogenic condition. Vaginal delivery is proven to cause structural sphincter defects; it may cause outright sphincter denervation in rare cases, but its more widespread implication in causing "idiopathic" incontinence is controversial.

CNEMG may be helpful in selected patients with faecal incontinence if a specific neurogenic condition (e.g., trauma or disease affecting the conus, sacral roots, sacral plexus or pudendal nerves) is suspected on clinical grounds.

v) Idiopathic Urinary Retention in Women

In young women with urinary retention (or obstructed voiding) complex repetitive discharges in profuse amounts in the external urethral sphincter against a background of firing motor units have been described, suggesting that these findings are of pathogenic and diagnostic significance. The external urethral sphincter was reported to be hypertrophic. A percentage of these women were hirsute and had polycystic ovaries [29, 30].

Repetitive discharges are, however, prone to develop in chronically partially denervated sphincters, and are present even in a proportion of asymptomatic women [31]. The distinguishing feature of the spontaneous EMG activity defining the particular pathology in women with retention seems to be its abundance, but the issue remains in dispute.

vi) EMG in Urodynamic and Functional Anorectal Studies

In health, voiding is characterised by cessation of motor unit firing in the urethral sphincter prior to detrusor contraction, as can be demonstrated by recording of “kinesiological EMG”. Bladder-sphincter coordination is impaired with lesions between the lower sacral segments and the upper pons. Consequently, sphincter activity is not inhibited, and often increases before detrusor contraction (i.e., 'detrusor-sphincter dyssynergia'). On the basis of the temporal relationship between urethral sphincter and detrusor contractions, three types of dyssynergia have been described [32].

There are other clinical situations that mimic detrusor sphincter dyssynergia. Sphincter contraction or at least failure of relaxation during involuntary detrusor contractions can be seen in patients with Parkinson's disease. The pelvic floor muscle contractions of the so-called non-neurogenic voiding dyssynergia may be a learned abnormal behaviour [33], and are a feature of dysfunctional voiding [30].

The pubococcygeus in the healthy female reveals similar activity patterns to the urethral and anal sphincters at most detection sites: continuous activity at rest, often some increase of activity during bladder filling, and reflex increases in activity during any activation manoeuvre performed by the subject such as talking, deep breathing, coughing. The pubococcygeus relaxes during voiding; the muscles on either side act in unison [13]. In stress-incontinent patients, the patterns of activation and the co-ordination between the two sides can be lost [34]. A delay in muscle activation on coughing has also been demonstrated, as compared to continent women [14].

Little is known about the complex activity patterns of different pelvic floor muscles (the urethral sphincter, urethrovaginal sphincter, anal sphincter muscle, different parts of the levator ani) during different manoeuvres. It is generally assumed that they all act in a co-ordinated fashion functionally as one muscle. However there are demonstrable differences between the intra- and peri-urethral sphincter in healthy females [35] and in activation of the levator ani and the urethral sphincter [36]. Co-ordinated behaviour is frequently lost in abnormal conditions.

Kinesiological needle EMG analysis of the urethra with the patient at rest and coughing may predict the outcome after certain types of incontinence surgery [37].

Current concepts suggest that defecation requires increased rectal pressure co-ordinated with relaxation of the anal sphincters and pelvic floor muscles. Pelvic floor relaxation allows opening of the anorectal angle and perineal descent, facilitating faecal expulsion. During defecation puborectalis activity is as a rule inhibited, but was unchanged in 9 % and increased in 25% of healthy subjects [38]. Thus, while "paradoxical" puborectalis contraction during defecation is used to diagnose pelvic floor dyssynergia in patients with typical symptoms, this finding may be a variation of the normal.

d) Pudendal nerve conduction tests

Measurement of motor conduction velocity is routinely used to evaluate limb motor nerves, distinguishing between a demyelinating and axonal neuropathy. To make the measurement requires access to the nerve at two well-separated points and measurement of the distance between them, a requirement that cannot be met in the pelvis. Another way to evaluate peripheral motor nerve function is the measurement of the) motor latency of a muscle response, requiring only a single

stimulation site. The muscle response is the compound muscle action potential (CMAP) or M-wave. Because in limb nerves the site of stimulation to obtain only the motor latency (without measuring the actual conduction velocity) is as a rule placed distally on the nerve, it is also called the distal (or terminal) latency. For the pudendal nerve the site of stimulation may be more or less “distally”, but the term distal or terminal has – in accordance to general clinical neurophysiology – become generally used. Distal motor latency can be measured by recording with a concentric needle electrode from the bulbocavernosus, the EAS and the urethral sphincter muscles in response to bipolar surface stimulation placed in the perianal/perineal region, or with selective needle stimulation of the pudendal nerve (branches) in the perineum. The most widely employed technique to obtain pudendal nerve terminal motor latency (PNTML) relies on stimulation with a special surface electrode assembly fixed on a gloved index finger, known as the St Mark's stimulator [39]. It consists of a bipolar stimulating electrode on the tip of the gloved finger with the recording electrode pair placed proximally on the base of the finger. The finger is inserted into the rectum or vagina and stimulation is applied close to the ischial spine. If a catheter-mounted electrode is used for recording, EMG responses from the striated muscle of the urethral sphincter can be obtained. Experts differ in their estimation of validity of this test. A prospective evaluation of anorectal physiologic tests in 90 patients with faecal incontinence did not find that PNTML results changed treatment decisions [40]. Indeed, the American Gastroenterological Association statement indicated that "PNTML cannot be recommended for evaluation of patients with faecal incontinence" [41].

c) Anterior sacral root (cauda equina) stimulation

Anterior root stimulation has been used to study conduction of the sacral nerve roots. Electrical stimulation with needle electrodes at vertebral laminae Th12-LI elicit M-waves in the bulbocavernosus and EAS muscle [42].

Transcutaneous stimulation of deeply situated nervous tissue became possible with development of special electrical and magnetic stimulators. When applied over the spine, these stimulators activate the roots as they exit the vertebral canal. Needle EMG rather than non-selective surface electrodes should be used to record pelvic floor and particularly sphincter responses to electrical or magnetic stimulation of the cauda equina. These stimuli non-selectively depolarise underlying neural structures, thereby activating several muscles innervated by lumbosacral segments [43].

Invasive percutaneous stimulation of individual roots in sacral foramina is used to identify patients with lower urinary and anorectal dysfunction who are likely to benefit from long-term stimulation, e.g. with the Interstim (Medtronic, Inc., Minneapolis, USA). Electrical stimulation of nerve roots at the level of the appropriate sacral foramina results in observable muscle contraction in the foot and perineum. These responses can be identified as MEP or reflex responses on the basis of their latency.

In conclusion, demonstrating the presence of a perineal MEP on stimulation over lumbosacral spine may occasionally be helpful in patients without voluntarily activated muscles. It also identifies the particular nerve root before introducing therapeutic electrical stimulation. However, the clinical value of the test has yet to be established and there are no sensitivity and specificity data on test results in individual patients.

d) Motor evoked potentials

Using magnetic or electric stimulation, it is possible to depolarise the motor cortex and record a response from the pelvic floor. Magnetic cortical stimulation is better tolerated than electrical stimulation, which has been abandoned in awake subjects, but may be useful for intraoperative monitoring.

By performing the stimulation at two different sites (brain and spinal roots), it is possible to record three different conduction times: a total conduction time, a peripheral conduction time, and a central conduction time (**Figure 5**). The total conduction time corresponds to the transit time from brain to target muscle. The peripheral conduction time is the transit time from sacral roots to the muscle. The central conduction time is obtained by subtracting the peripheral conduction time from the total conduction time. The total conduction time can be measured both at rest and during a facilitation procedure. MEPs from the EAS, the urethral sphincter, the bulbocavernosus muscle, and the levator ani muscle have been reported, but normative values have only been obtained (for transcranial magnetic stimulation) for the urethral sphincter and the puborectal muscle in adult women [44]. The necessity to use concentric needle EMG for recording has been reconfirmed [45].

Substantially longer central conduction times have been found in patients with multiple sclerosis and spinal cord lesions as compared to healthy controls [46]. However all patients in this study had clinically recognisable cord disease.

Conceptually, MEP may help to differentiate between involvement of motor and sensory pathways. However, the clinical utility of these measurements is not established. MEP have opened an avenue of research on excitability of motor cortex. It has been demonstrated that in comparison to the motor area for hand muscles the anal sphincter motor cortex has less intracortical inhibition [47].

2. SENSORY SYSTEM TESTS

There are several methods of sensory testing for the perineum, the genitourinary and anorectal tract. Clinical

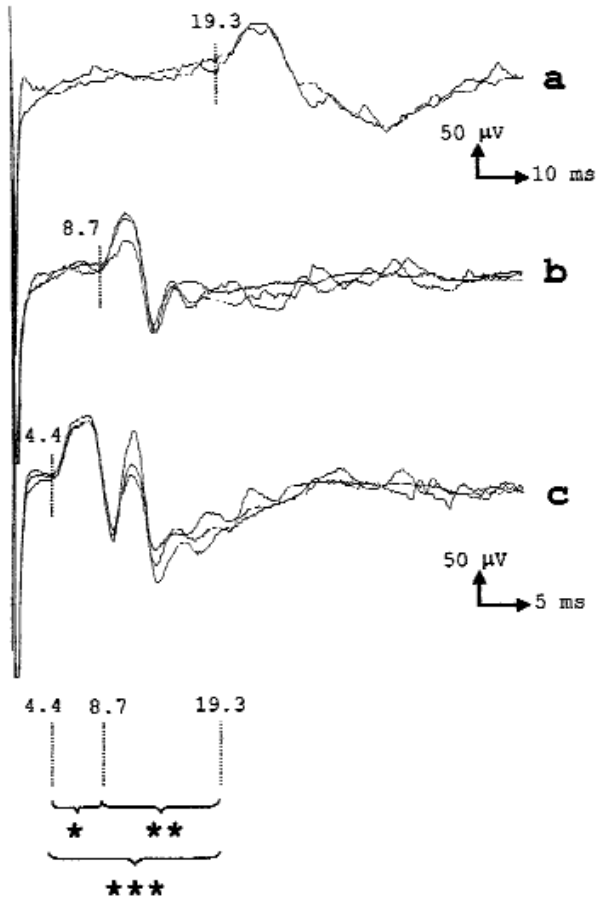


Figure 5 : MEPs recorded by concentric needle in the external urethral sphincter of a 51-year-old-woman. Cortical (a), thoracic (b), and sacral (c) stimulation. Central motor conduction time (CMCT) is calculated as cortical - lumbar latency (** = 10.6 ms). Cauda equina motor conduction time is calculated as lumbar - sacral latency (* = 4.3 ms). (From (44), with permission).

testing includes perineal and external genital skin sensation for light touch and pinprick, and sensation of bladder filling during cystometry. Anorectal sensory testing can be clinically assessed through rating of applied stimuli. More objective sensory testing can be performed with quantitative sensory testing (QST), which assesses sensory perception. For evaluation of the integrity of sensory pathways sensory neurography, and somatosensory evoked potentials (SEP) can be used.

a) Sensory Measurements During Cystometry

During routine cystometry bladder sensation is assessed by recording first sensation of bladder filling, first desire to void and strong desire to void.

Bladder and urethral sensory thresholds have also been measured using electrical stimulation [48], and mechanical traction on the bladder trigone [49]. There is no established clinical use for any of these tests other than simple reporting of sensation during cystometry.

b) Assessment of Anorectal Sensation

Rectal sensation is assessed by progressively distending a balloon manually or by a barostat while measuring thresholds for first perception, desire to defecate, and severe discomfort. The intensity of perception during rectal distension can be recorded by a visual analogue scale during phasic distensions of graded intensity [50]. The rate and pattern of distension affect rectal perception and internal sphincter relaxation [51].

Anal sensation can be assessed by determining the perception threshold to an electrical stimulus or temperature change in the anal canal. Electrical testing does not activate mucosal receptors. Anal sensitivity to temperature change has been reported reduced in faecal incontinence [52].

c) Quantitative Sensory Testing

Quantitative sensory testing (QST) of the urogenitoanal system should provide more objective and reproducible data than routine clinical testing. QST sensory modalities applied to the evaluation of urogenital function include vibration [53], temperature [54], and electrical current [55]. There is no commonly accepted, detailed, standardised test, and the specificity and sensitivity of the tests are not known. The relationship of cutaneous quantitative sensory tests to bladder and urethral sensation and function is unknown. The physiological, psychophysiological and methodological issues and controversies will not be addressed in this chapter.

d) Sensory Neurography

Nerve conduction velocities of the dorsal nerve of the penis can be calculated by placing a pair of stimulating electrodes across the glans and a pair of recording electrodes across the base of penis. A nerve action potential can be recorded with amplitude of about 10 µV. It can also be recorded by stimulating trans-rectally or transperineally. There is no known association between penile sensory neuropathy and bladder/sphincter dysfunction.

A few studies have recorded activity in sacral roots during electrical stimulation. Intraoperatively, when the sacral roots are exposed, compound sensory action potentials on stimulation of dorsal penile and clitoral nerve may be recorded directly [56]. This helps to preserve roots mediating perineal sensation in spastic children undergoing dorsal rhizotomy, and reduce the incidence of postoperative voiding dysfunction. These tests are limited to their very specific intraoperative indications.

e) Somatosensory Evoked Potentials (SEP)

Somatosensory evoked potentials are electric waveforms of biologic origin elicited by stimulation of a sensory nerve (or a sensory innervated skin area

– dermatome). The most commonly performed tests in the urogenitoanal region are pudendal somatosensory evoked potentials (SEP), which assesses conduction in large fibre pathways between the site of nerve stimulation and the parietal sensory cortex. Potentials can also be measured at the spinal level (spinal SEP). Visceral (thin) fibre pathways are assessed by recording SEPs while stimulating the proximal urethra and bladder, although this is technically not depolarization of nerves, but a mesh of afferents.

1. Pudendal Somatosensory Evoked Potentials

i) Cerebral Pudendal SEP

On electrical stimulation of the dorsal penile/clitoral or perineal nerve, a cerebral SEP can be recorded. (**Figure 6**) This SEP is as a rule of highest amplitude at the central recording site (Cz - 2 cm : Fz of the International 10-20 EEG System) and is highly reproducible. The first positive peak at about 40 ms (called P40) is usually clearly defined in healthy subjects using a stimulus 2-4 times stronger than the sensory threshold [57]. The presence and amplitude of subsequent negative and positive waves are quite variable between subjects. Classically described pudendal SEP techniques stimulate both dorsal penile/clitoral nerves, thus reducing the sensitivity of the test. However, techniques of pudendal SEP that isolate each dorsal penile/clitoral nerve may be more sensitive for identifying pathology [58].

Pudendal SEPs have been advocated in patients with neurogenic bladder dysfunction, e.g. in multiple sclerosis [59]. However, even in patients with multiple sclerosis and bladder symptoms, the tibial cerebral SEP was more often abnormal than the pudendal SEP. The combination of an abnormal pudendal SEP with a normal tibial SEP suggests isolated conus involvement [60]. The pudendal SEP was less useful than neurological examination for identifying neurological disease in patients with uro-genital symptoms [61]. Following spinal cord injury, tibial and pudendal SEPs may be of some value for predicting recovery in bladder control [62]. Cerebral SEP during penile/clitoral stimulation may be useful for intraoperative monitoring. Pudendal SEP were used to study the mechanism of sacral neuromodulation [63].

3. SACRAL REFLEXES

a) Sacral Reflex on Electrical Stimulation

Electrical stimulation of the dorsal penile or clitoral nerve elicits (somato-somatic) sacral reflexes in perineal muscles with a typical latency approx. 33 ms, traditionally called the bulbocavernosus reflex (Figure 6). Stimulation of the perianal skin, bladder neck or proximal urethra elicits sacral reflexes with latencies above 50 ms. This latency is longer compared to responses conveyed by the pudendal nerve, suggesting that the afferent limb for these responses involves visceral afferent fibres accompanying the pelvic nerves, which are thinly myelinated and have a slower conduction velocity than the thicker pudendal afferents. With visceral denervation (e.g. following radical hysterectomy) the viscerosomatic reflexes (from both bladder and urethral stimulation) may be lost while the bulbocavernosus (penilo-/clitorocavernosus) reflex is preserved. Loss of bladder-urethral reflex with preservation of bladder-anal reflex has been described with urethral afferent injury after recurrent urethral surgeries [64].

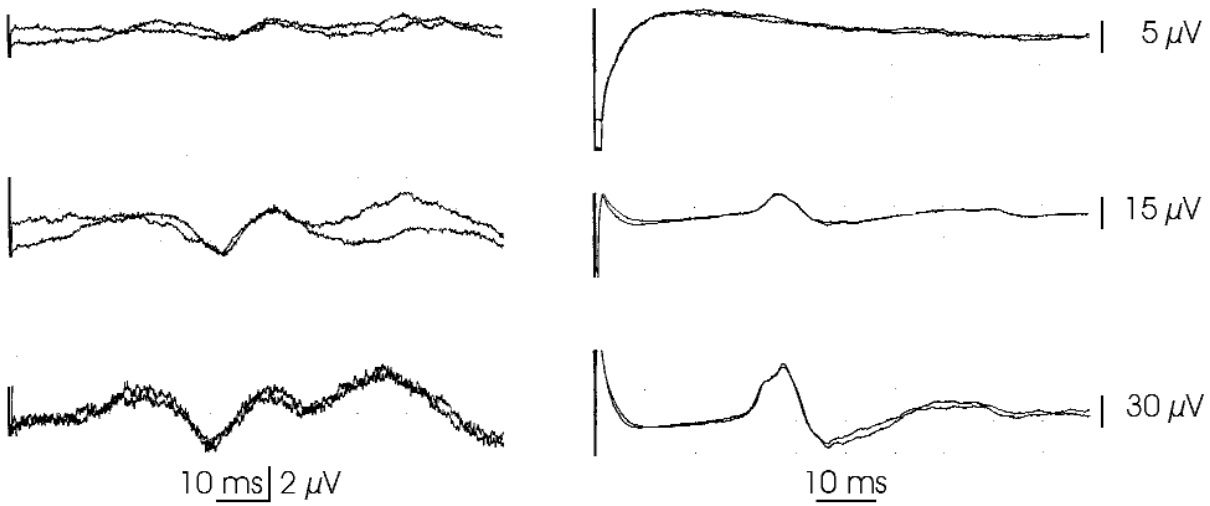


Figure 6 : SEPs (traces on the left) and sacral reflexes (traces on the right) in a healthy woman. Cerebral SEPs are recorded from Cz - 2 cm; sacral reflexes from the anal sphincter. The dorsal clitoral nerve is being stimulated with rectangular electrical pulses at 2 Hz. Stimulation and recording is performed with surface electrodes. The cerebral SEP and sacral reflex are recorded simultaneously. In the upper row the stimulation is just above sensory threshold, in the middle row the stimulation is 1.5, and in the lower row at 2-times sensory threshold (pulse duration 0.2 ms; two consecutive averages of 128 responses are superimposed).

The longer latency anal reflex (the contraction of the EAS on stimulation of the perianal region) is quite variable thus limiting its usefulness as a diagnostic tool.

On perianal stimulation, a short latency response can also be recorded, as a result of depolarisation of motor branches to the EAS, possibly involving antidromic travelling of the depolarisation, with "returning" of the depolarisation orthodromically to the sphincter at a branching point of the motor axon.

EMG recording of the sacral reflex has been shown to be more reliable than the clinically assessed response (e.g. observing and palpating the contraction) in males and particularly in females [65]. In men with cauda equina lesions penilo-cavernosus reflex could not be elicited in 64%, 47% and 47% of patients on single electrical, double electrical, and mechanical stimulation, respectively. Measurement of the reflex latency increased the sensitivity to record abnormalities for 17%, 36%, and 34%, respectively. Furthermore, it has been shown that sacral reflex measurement increase sensitivity of quantitative EMG of the EAS muscles from 73% to 81-83% using the different stimulation techniques mentioned [66].

Sacral reflex testing has been studied extensively and is used in many laboratories in everyday practice to demonstrate objectively the integrity of the S2-S4 reflex arc. The sacral reflex evoked on dorsal penile or clitoral nerve stimulation (the bulbocavernosus or penilo-/clitoro-cavernosus reflex) was shown to be a complex response, often forming two components. The first component with a typical latency of about 33 ms, is the response that has been most often called the bulbocavernosus reflex. It is stable, does not habituate, and has other attributes of an oligosynaptic reflex response [67]. The second component has latency similar to the sacral reflexes evoked by stimulation perianally or from the proximal urethra, and is not always demonstrable as a discreet response. In those subjects in whom the first reflex component is difficult to elicit, stimulation strength should be increased, but preferably double electrical stimuli should be used. A complete reflex arc lesion should not be inferred by absence of a response if only single pulse is used for stimulation.

During voiding sacral reflexes are un-elicitable but in presence of spinal cord lesions such as myelodysplasia this normal suppression is lost.

Sacral reflex responses recorded with needle or wire electrodes can be analysed separately for each side from the EAS or bulbocavernosus muscle. Using unilateral dorsal penile nerve blocks, the existence of two unilateral BCR arcs has been demonstrated. Thus by detection from the left and right bulbocavernosus (and also the EAS) muscles separate testing of right and left reflex arcs can be performed. Some authors reported that the sensitivity of the test can be increased by use of the inter-side latency difference (normative limits: < 3 ms), but this finding could not be confirmed by others (normative limits: < 7.2 ms) [68]. In cases of unilateral (sacral plexopathy, pudendal neuropathy) or asymmetrical lesions (cauda equina), a healthy reflex arc may obscure a pathological one on clinical elicitation, but not on neurophysiologic measurements of the sacral reflexes.

As described above, penilo-cavernosus reflexes were absent in 47-64%, and delayed in additional 17-19% of patients with conus/cauda lesions. Of these patients 47% were incontinent for urine and 47% for faeces. However, a reflex with a normal latency does not exclude the possibility of an axonal lesion in its reflex arc, as demonstrated by pathologic quantitative EMG of the EAS in 79-86% of patients with conus/cauda lesions [66]. Furthermore, much delayed sacral reflex responses are compatible with normal bladder and sexual function as found in patients with hereditary motor and sensory demyelinating neuropathy.

Sacral reflex recording is suggested as a complementary test to CNEMG examination of pelvic floor muscles in patients with suspected peripheral nervous lesions [4]. In addition to latency, a number of other parameters can also be measured using electrical, but not mechanical stimulation.

These are the sensory threshold (i.e., the stimulus strength (mA) at which subjects feels stimulation), reflex threshold (i.e., the stimulus strength (mA) at which the reproducible penilo/clitoro-cavernosus reflex appears on the screen), and stimulation strength (i.e., the stimulation intensity (mA) at which the response latency does not shorten and amplitude does not increase in spite of increasing the stimulus strength). Although for men normative data for these parameters is available (68), their utility in clinical situation remains unclear.

Continuous intraoperative recording of sacral reflex responses on penis/clitoris stimulation is feasible if double pulses or a train of stimuli are used [69].

b) Sacral Reflex on Mechanical Stimulation

Mechanical stimulation has been used to elicit BCR in both sexes and found to be a robust technique. Either a standard reflex hammer or a customised electromechanical hammer can be used. Such stimulation is painless and can be used in children. The latency of the BCR elicited mechanically is comparable to the electrically elicited reflex in the same patients, but depends on the electromechanical device used.

4. AUTONOMIC FUNCTION TESTS

Most uro-neurophysiological methods discussed so far assess myelinated fibres, but not the autonomic nervous system, especially the parasympathetic component, which is most relevant for pelvic organ functions. Methods for evaluating the autonomic nerves

innervating the pelvic viscera are not available. Cystometry indirectly evaluates the parasympathetic innervation to the bladder. However, from a clinical neurophysiological point of view direct electrophysiological testing would be desirable.

a) Tests in Generalised Autonomic Neuropathy

Cardiovascular autonomic function tests are useful for identifying generalised autonomic dysfunction in patients with bladder or gastrointestinal motility disturbances.

In cases when a general involvement of thin fibres is expected, an indirect way to examine autonomic fibres is to assess thin sensory fibre function. Thin visceral sensory fibres are tested by stimulating the proximal urethra or bladder, and by recording sacral reflex responses or cerebral SEP.

b) Smooth Muscle Electromyography

Technical problems have so far limited smooth muscle electromyography of the detrusor muscle, and of genital smooth muscle. There is no evidence to prove the clinical utility of these tests in the evaluation of urinary tract function.

c) Sympathetic Skin Response (SSR)

The sympathetic nervous system mediates sweat gland activity in the skin. Changes in sweat gland activity lead to changes in skin resistance. On noxious stimulation (such as a sudden noise, electrical pulse, etc.) a potential shift can be recorded with surface electrodes from the skin of the palms and the soles, and has been reported to be a useful parameter in assessment of neuropathy involving non-myelinated nerve fibres. The response, known as the SSR, can also be recorded from perineal skin and the penis.

The SSR is a reflex, which consists of myelinated sensory fibres, a complex central integrative mechanism and a sympathetic efferent limb with postganglionic nonmyelinated C fibres. SSR is the only electrophy-siological method directly testing sympathetic fibres. Limited literature exists regarding the relationship between SSR results and bladder dysfunction. A correlation has been shown between the absence of the SSR response in the foot and bladder neck dyssynergia following spinal cord injury [62]. Recording from the perineal assesses sympathetic nerve function within the thoracolumbar cord [70].

Only complete absence of response can be regarded as abnormal. Its utility in evaluating bladder and urethral dysfunction is not established.

III. EVIDENCE BASED USE OF CLINICAL NEUROPHYSIOLOGICAL TESTS

Evidence-based medicine is founded on the assessment of evidence for and against the efficacy of particular types of therapeutic intervention. Clinical neurophysiology testing should thus demonstrate evidence that testing improves outcome (through treatment choice and patient selection), which would provide a strong basis for its use. However, testing and therapeutic intervention are different concepts, and neurophysiological testing has another important objective, which is not applicable to interventions and lies outside the scope of evidence-based medicine. It is to generate knowledge about the situation to be treated in a given patient, so that the practitioner can formulate rational treatment options based on knowledge rather than do so blindfold; that is, he or she can practice "knowledge-based medicine".

To judge the importance of this second objective different criteria are needed. Particularly in the referral setting, the physician is confronted with complicated cases in whom the underlying pathophysiology is quite uncertain, and what is required is to identify all the factors that may be contributing. Neurophysiology is helpful in assessment of neurogenic dysfunction because it contributes to "knowledge-based medicine", whether or not there is narrowly-defined "evidence" that it improves outcomes.

Of course, it remains true that we should seek evidence of the conventional kind for and against testing. Any test should be subjected to three questions:

1. Does the test have good technical performance?
2. Does the test have good diagnostic performance, ideally against a "gold standard" measure?
3. Does the test have good therapeutic performance, that is, does the use of the test alter clinical management, does the use of the test improve outcome?

Clinical diagnosis requires that measures obtained in individual patients be compared to population norms with the intent of determining whether they are "normal" or "abnormal". Data can be classified as "abnormal" only with the understanding that they are compared to a sample from the normal population. Predictive statements are made possible by the use of tolerance limits. For most clinical neurophysiological tests, one-tailed tolerance limits are recommended. For any given limit of normality, there is a certain probability

of falsely interpreting values (obtaining false-positives or false-negatives). Further confounding these issues is the practice of applying multiple criteria of abnormality. But ultimately, the adequacy of any given normal limit in discriminating between normal and abnormal must be supported by appropriate clinical or clinico-pathological correlations; for uroneurophysiological techniques, such data are scarce.

1. USEFULNESS OF CLINICAL NEUROPHYSIOLOGICAL TESTS IN EVALUATION OF INDIVIDUAL PATIENTS

Whenever pathophysiology is uncertain or unpredictable, and especially if irreversible treatment is necessary or contemplated, it seems logical to gather quantitative knowledge of the dysfunction in order to make a rational treatment choice. In most patient groups with neurogenic incontinence, the pathophysiology is unpredictable and comprehensive urodynamic evaluation is essential in order to practice knowledge-based medicine. In selected patients from these groups, clinical neurophysiological testing will clarify issues related to the neural control of lower urinary tract, relevant for understanding pathophysiology. Most patients, however, will not require a precise definition of the neurological lesion.

As is generally true for electrophysiological tests, uroneurophysiological examinations are particularly useful for substantiating the clinical diagnosis of a peripheral nerve lesion. The potential usefulness of testing in an individual patient needs to be analysed in the overall clinical setting. The indications for testing are guided primarily by expert opinion, not on definitely established criteria derived from controlled studies.

In the incontinent patient without other signs or symptoms of a neurological condition, neurophysiological testing is generally unnecessary. In patients with stress/urge, or mixed urinary incontinence electrophysiological testing is as a rule non-contributory [28].

2. USEFULNESS OF CLINICAL NEUROPHYSIOLOGICAL TESTS IN RESEARCH

Uro-neurophysiological techniques have been most often applied in research, for instance to elucidate the innervation of pelvic floor muscles; to study the physiology of contraction of sphincter muscle, to describe activation patterns of pelvic floor muscles. Suggestions of increased efficacy of sacral neurostimulation with the use of neurophysiologic tests have been made [71]. As understanding pathophysiology and neural control is essential in the application of more sophisticated therapeutic methods, such as electrical stimulation techniques, there seems to be a continuing place for clinical neurophysiology in research on neurogenic urinary and anorectal dysfunction and their therapy.

IV. RECOMMENDATIONS

1. RECOMMENDATION FOR CLINICAL NEUROPHYSIOLOGICAL TESTING

The information gained by clinical examination and urodynamic testing may be enhanced by uroneurophysiological tests in selected patient groups with suspected neurogenic urinary incontinence with lesions within the nervous reflex arcs of sacral segments 2 - 5. Concentric needle EMG to diagnose denervation and reinervation of pelvic floor and perineal muscles, and sacral reflex testing to assess the continuity of the sacral reflex arc, are the recommended tests.

Level of evidence: 2b

Level of recommendation: B

Clinical neurophysiological testing should be performed in accredited laboratories, by trained and certified staff, with formal control of the quality of the results. Ideally, the uroneurophysiologist should be in liaison with general clinical neurophysiologists.

It seems optimal to create interdisciplinary programs between urology, urogynecology, proctology, and neurology departments. Organisation of such teams in tertiary medical centres should be encouraged.

2. RECOMMENDATION FOR TECHNICAL STANDARDS

Even in the more widely used "general" clinical neurophysiology there is no universal standardisation of tests. This is mainly due to different historical backgrounds of testing developed in different countries. The need to standardise methods is, however, recognised.

Proposals for standardisation for external anal sphincter CNEMG [4] and the bulbocavernosus (penilo/clitoro-cavernosus) reflex [68] have been made, and seem to be widely adopted.

Level of evidence: 2b

Level of recommendation: B

3. RESEARCH RECOMMENDATIONS

Clinical neurophysiological methods should be further used to better define the neural control in lower urinary tract function, demonstrating both the nervous system's "hardware" (integrity of anatomy) as well as "software" (level of activity, excitation thresholds) for co-ordinated urinary storage and voiding, in physiological and in pathological conditions. In particular, there is the opportunity to better define neurophysiological changes induced by therapeutic electrostimulation.

There is the challenge to develop tests to assess directly the sacral parasympathetic system.

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