Chapter 14
Bladder Filling and Storage: "(Involuntary) Contractions"

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Urodynamics (UDS) is the dynamic study of the storage and evacuation of urine. Its role is to provide the clinician with information about a patient's lower urinary tract function. Ideally, the patient's symptoms are reproduced during the study to provide objective measurements which correlate with the patient's symptoms. UDS should be considered as an adjunct to a detailed history and physical exam to better characterize the clinical picture and facilitate optimal treatment planning. An important portion of the UDS is the bladder filling and storage phase. Involuntary detrusor contractions (IDCs) may occur during this portion of the study and may provide insight into the pathophysiology of the patient's complaints. This chapter will focus on the different UDS manifestations of IDCs by investigating the UDS tracings of neurogenic and idiopathic detrusor overactivity, stress-induced overactivity, phasic overactivity, detrusor overactivity incontinence, detrusor after-contractions (DAC), and terminal detrusor overactivity.

The International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction was published in 2010 [1]. It contains terminology for common urodynamic terms and observations and it is recommended that clinicians performing UDS consider using the current IUGA/ICS terminology in order to maintain consistency. It defines *detrusor overactivity* (*DO*) as the "*occurrence of involuntary detrusor contractions during filling cystometry. These contractions, which may be spontaneous or provoked, produce a wave form on the cystometrogram of variable duration and amplitude*." (see Fig. 14.1a) *Urgency* (urinary) is defined as the "*complaint of a sudden, compelling desire to pass urine which is difficult to defer.*" It is important to note that urgency is a symptom and may or may not be represented by

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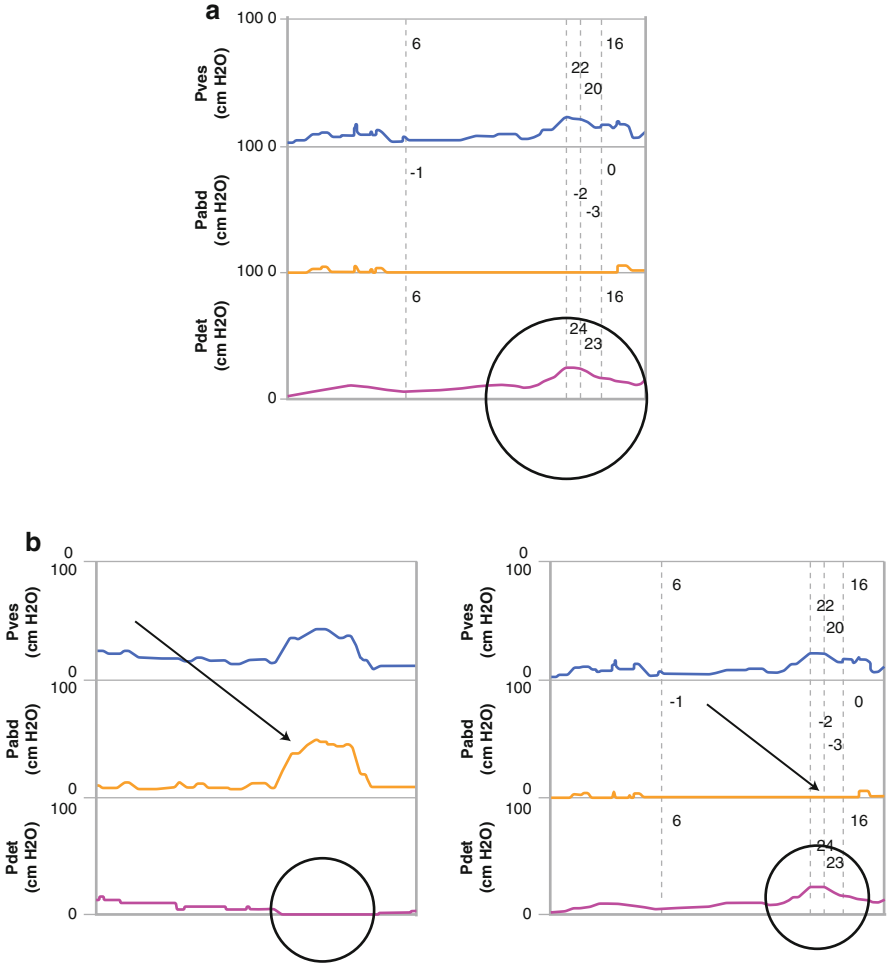

Fig. 14.1 (**a**) This is an example of detrusor overactivity (highlighted by the *circle*) during the filling cystometrogram. The P_{det} tracing is calculated using P_{ves} and P_{abd} values as explained in (**b**). (**b**) Using the equation $\mathbf{P_{det} = P_{ves} - P_{abd}}$, the calculated P_{det} (*circled*) in the figure on the left is zero since the rise in P_{ves} is associated with a rise in P_{abd} (highlighted by the *arrow*). This can be seen, as in this case, due to a Valsalva maneuver which causes a slow controlled rise in the P_{abd} and P_{ves} tracings for the duration of the maneuver. Conversely, the calculated P_{det} (*circled*) in the figure on the right represents a true increase in detrusor pressure since the rise in P_{ves} is not associated with a rise in P_{abd} (highlighted by the *arrow*). In other words, the increase in P_{det} is independent of any intraabdominal pressure activity.

findings of DO on UDS. In fact, it is well established that up to 50 % of patients with urgency incontinence do not demonstrate DO on UDS tracings [2].

Vesical pressure (P_{ves}) is the *measured* pressure inside the bladder. It is measured with a pressure transducer catheter placed into the bladder. Abdominal pressure (P_{abd}) is the *measured* pressure inside the abdomen. It is usually measured with a pressure

transducer catheter placed into the rectum. Detrusor pressure (P_{det}) is a *calculated* value of the difference between P_{ves} and P_{abd} such that $\mathbf{P_{det} = P_{ves} - P_{abd}}$ (Fig. 14.1b). P_{det}, though a completely derived number, represents the true value of the pressure generated by the bladder and is distinguished from changes in the pressure tracings due to increased abdominal pressure such as that seen with coughing or straining. From a clinical perspective, evaluating the value of P_{det} is important, as documented by McGuire, due to the potentially deleterious effects on the upper tracts when the detrusor pressure (P_{det}) during filling or voiding is sustained above 40 cm H_2O [3].

The normal micturition cycle involves passive low-pressure filling of the bladder and a coordinated detrusor contraction coupled with urinary sphincter relaxation for the evacuation of urine. A normally functioning bladder stores urine at low pressures as a result of the bladder's viscoelastic properties and compliance. Filling cystometry provides information regarding bladder sensation, bladder capacity, detrusor activity, and bladder compliance. In addition, it is during this phase of the urodynamic study that the bladder outlet can be assessed for weakness (stress urinary incontinence). The two phases of UDS include the filling/storage phase and voiding phase (Fig. 14.2). Detrusor activity is normal during the voiding phase as the bladder contracts to expel urine. Detrusor activity should not be present during the filling and/or the storage phases and when seen is considered involuntary and is referred to as DO. DO can occur spontaneously or as a result of provocation. Provocative maneuvers such as coughing (Fig. 14.3) and Valsalva maneuvers are performed during the storage phase to assess for SUI. These provocative maneuvers can also provoke DO in a phenomenon described as stress-induced DO which will be covered later in the chapter.

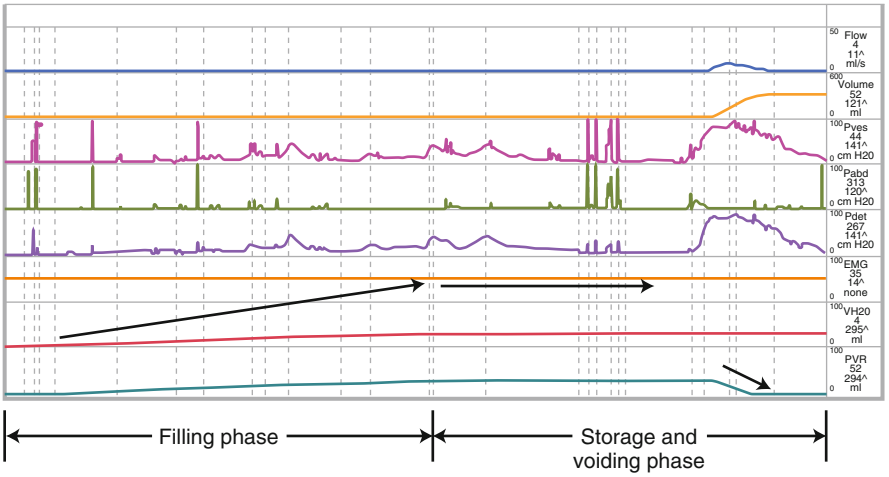

Fig. 14.2 The *filling phase* is indicated by the steady increase in volume in the bladder as it is being filled. The storage phase can be identified by a stable volume in the bladder. The voiding phase begins when the patient is given permission to void. Note that in this instance, the patient is able to void successfully. There is an increase in flow with a concomitant decrease in the post void residual (PVR) volume

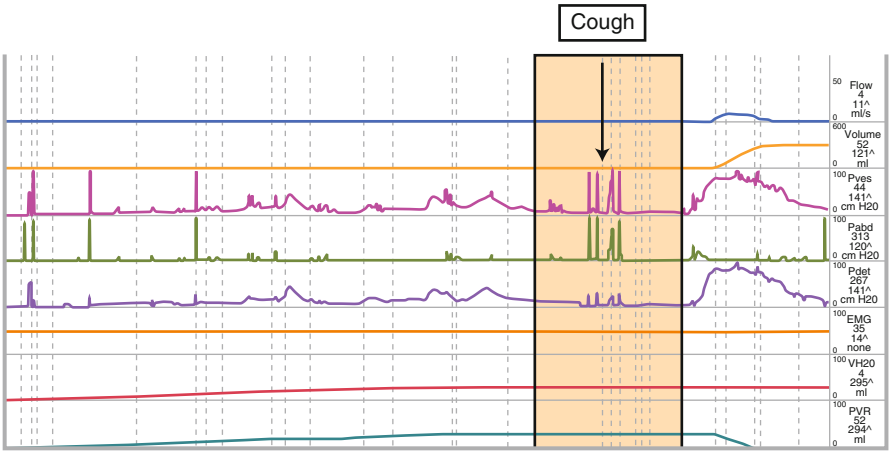

Fig. 14.3 Provocative maneuvers such as coughing (indicated by the arrow) result in increases in P_{ves} and P_{abd}. The P_{det} value is the calculated value determined by the difference in the measured P_{ves} and P_{abd} such that $\mathbf{P_{det} = P_{ves} - P_{abd}}$. In the case of a cough, P_{det} is negligible as there is no pressure coming from the detrusor itself. Acute increases in the P_{det} lead during provocative maneuvers such as coughing represent artifact and should not be misinterpreted as detrusor overactivity

The smooth muscle fibers of the bladder can exhibit spontaneous action potentials, a phenomenon thought to be the cause of phasic detrusor activity [4]. According to the 2002 ICS standardization of terminology [5], *phasic detrusor overactivity* (*PDO*) is defined as *a characteristic waveform, and may or may not lead to urinary incontinence*. Although the contour of the characteristic waveform is not specified, as the name suggests one would expect cyclical increases and decreases in the P_{det} tracing (Fig. 14.4).

Neurogenic detrusor overactivity (*NDO*) replaces the old terminology "detrusor hyperreflexia" and is defined as DO in a patient with a neurologic condition (Fig. 14.5) [1].

Idiopathic detrusor overactivity (*IDO*) (Fig. 14.6) replaces the old term "detrusor instability" and is the term used when there is no identifiable neurological cause for the DO. It is important to note that NDO and IDO may look identical on UDS. These terms are strictly defined by the patient's neurologic status and not by the presence of IDCs on the UDS tracings.

A causative relationship between SUI and DO has been suspected but never confirmed. Hindmarsh et al. proposed that proximal urethral underactivity with falls in the urethral closure pressure below 30 cm H_2O was often associated with DO. They suggested that DO may originate from stimuli to the bladder outlet [6]. Jung et al. measured the urethral perfusion pressure and isovolumetric bladder pressure in the anesthetized rat and concluded that the activation of urethral afferents by urethral perfusion can modulate the micturition reflex. They further speculated

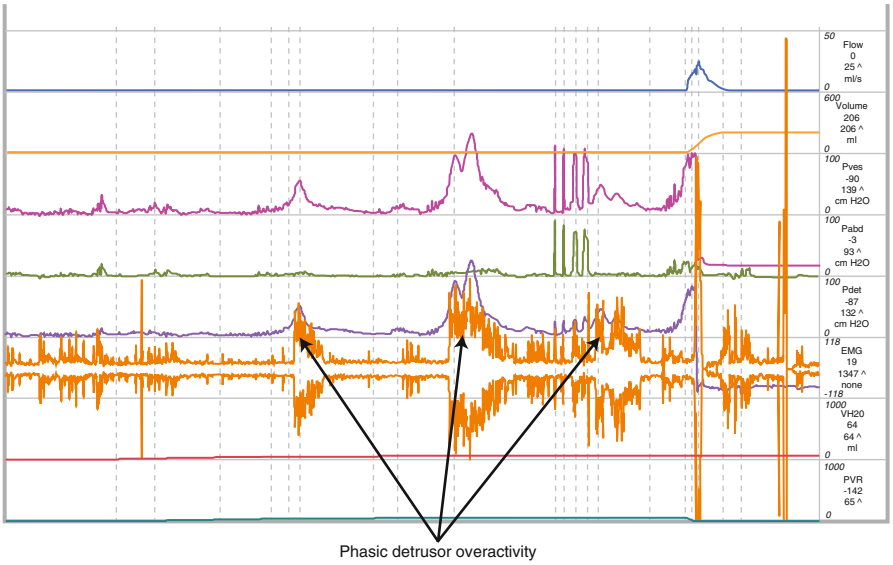

Fig. 14.4 This UDS demonstrates the "waveforms" in the P_{det} tracing with phasic DO. There is no standardization regarding the characteristics of the "waveform," but is generally recognized as cyclical increases and decreases in P_{det}. Note that the P_{abd} is silent suggesting that the increases in P_{det} are due to contractions arising in the bladder

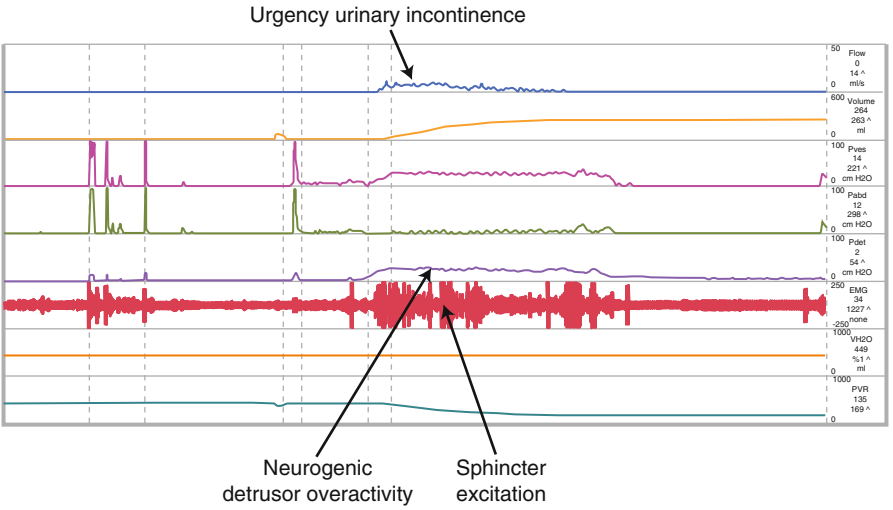

Fig. 14.5 This patient, who is recovering from a cerebral vascular accident, has NDO during the *storage phase* with associated UUI. It is important to highlight that the NDO occurred during the *storage phase* and not during the voiding phase. The compensatory EMG recruitment could be erroneously labeled as detrusor sphincter dyssynergia (DSD) which may occur during the *voiding phase* in patients with neurologic pathology

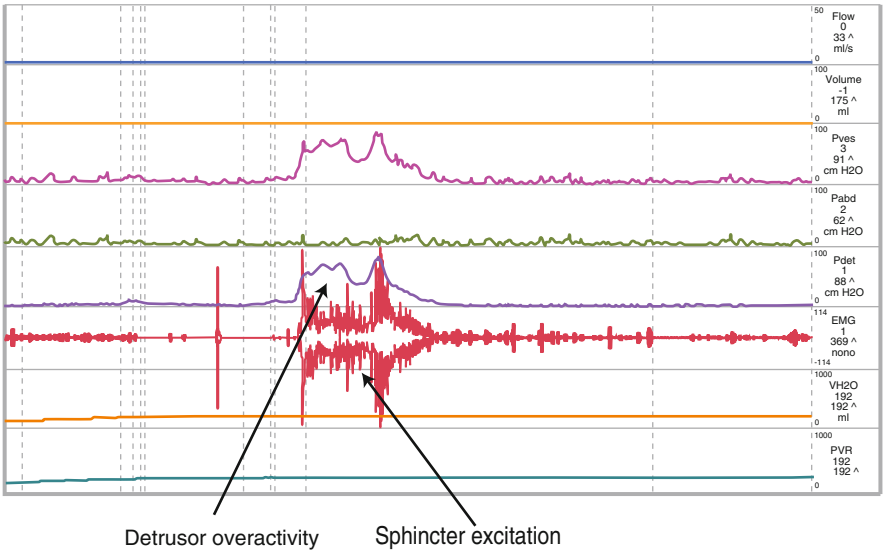

Fig. 14.6 This patient has no known neurologic disease. The detrusor overactivity is therefore referred to as idiopathic detrusor overactivity (IDO). It is important to reiterate that NDO and IDO P_{det} tracings can appear identical. The only difference between NDO and IDO is the patient's history of an associated neurologic disease. Conversely, if a patient is described as having NDO, it is implicit that he/she has an underlying neurologic disease. Again, note the compensatory EMG recruitment to prevent UUI

that the theoretical urethral-detrusor facilitative reflex or "stress-induced DO" could be a result of an incompetent bladder neck which allowed urine to reach the proximal urethra [7–9]. Interestingly, some women with mixed urinary incontinence (MUI) have resolution of urgency following surgical correction of SUI while other women may conversely develop de novo urgency after anti-incontinence surgery [10]. Unfortunately, current diagnostic methodology does not provide the means to accurately determine which patients with MUI would be best served with initial management of their SUI versus treatment of their urgency symptoms. SUI and DO can also potentially coexist as independent mechanisms which may explain the contradictory results reported in the literature evaluating the effects of pharmacotherapy and/or surgery in women with MUI. Although defining the predominant subjective component is important in the assessment of women with MUI, symptom analysis may not always be the most accurate diagnostic tool. The findings of stress-induced DO (Fig. 14.7) may help the clinician to better understand the patient's picture and ultimately guide the patient and the clinician in their discussions regarding treatment.

There are two measureable leak point pressures according to the IUGA/ICS joint report on urodynamic terminology are the *detrusor leak point pressure* (*DLPP*) and *abdominal leak point pressure* (*ALPP*). DLPP is defined as *the lowest detrusor*

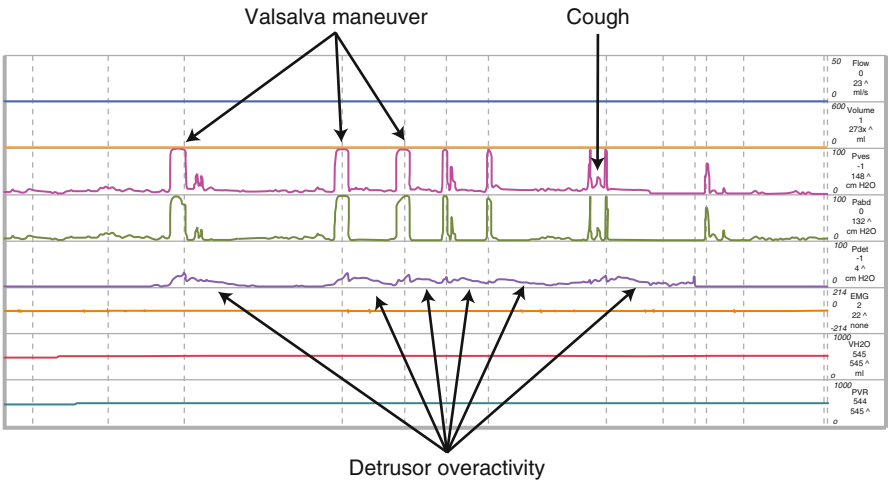

Fig. 14.7 Stress-induced DO occurs following episodes of provocative maneuvers (Valsalva and cough)

pressure at which leakage is observed in the absence of increased abdominal pressure or a detrusor contraction. ALPP is defined as the *lowest value of intentionally increased intravesical pressure (P_{ves}) that provokes urinary leakage in the absence of a detrusor contraction.* ALPP can be elicited during coughing (cough LPP) or Valsalva maneuver (Valsalva LPP). A group recently investigated incontinence that occurred with DO (Fig. 14.8), the pressure at which leakage occurred, and its possible association with subjective severity of urgency and UUI. They noted that subjective symptom severity and bother from urgency and UUI measured by validated questionnaires were greater in patients with urodynamic evidence of DO incontinence than those with urgency incontinence without DO [11]. They also found that patients with subjective MUI (UUI + SUI) leaked at a lower mean detrusor pressure than those who had pure UUI (19.6 cm H_2O vs 31.2 cm H_2O, respectively, $p = 0.004$). The clinical relevance of the detrusor pressure at which patients leak with UUI remains undefined.

Terminal DO (TDO) is defined as *a single involuntary detrusor contraction occurring at cystometric capacity that cannot be suppressed and results in incontinence usually resulting in bladder emptying (voiding)*. Investigators from France evaluated the demographics, urodynamic findings, and sphincter behavior in 166 women with a diagnosis of DO. They found that the incidence of PDO (Fig. 14.9) and TDO (Fig. 14.10) was similar at 46.9 and 53.1 % respectively. The PDO group were younger (52 ± 19) than the TDO group (63 ± 16) ($p = 0.0003$), and TDO occurred more frequently than PDO with increase in age ($p = 0.006$). The frequency of neurologic disease in the TDO group was high (60.5 %) in the pre and peri-menopausal age groups (45–74 years) and remained elevated in the other age groups (45.8 %) [12]. Other studies support the finding that TDO is related to aging

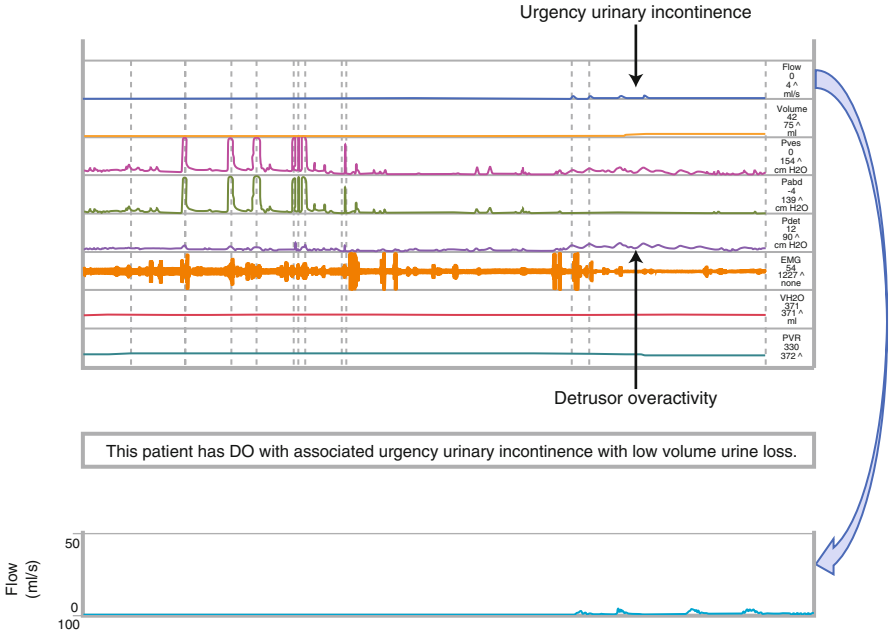

Fig. 14.8 This patient has DO with associated urgency urinary incontinence with low volume urine loss

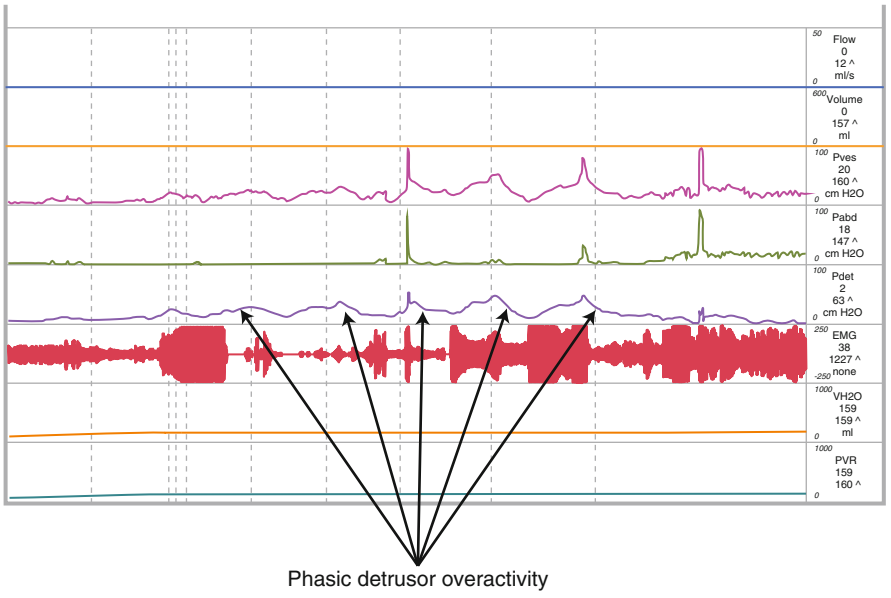

Fig. 14.9 UDS tracing shows phasic DO

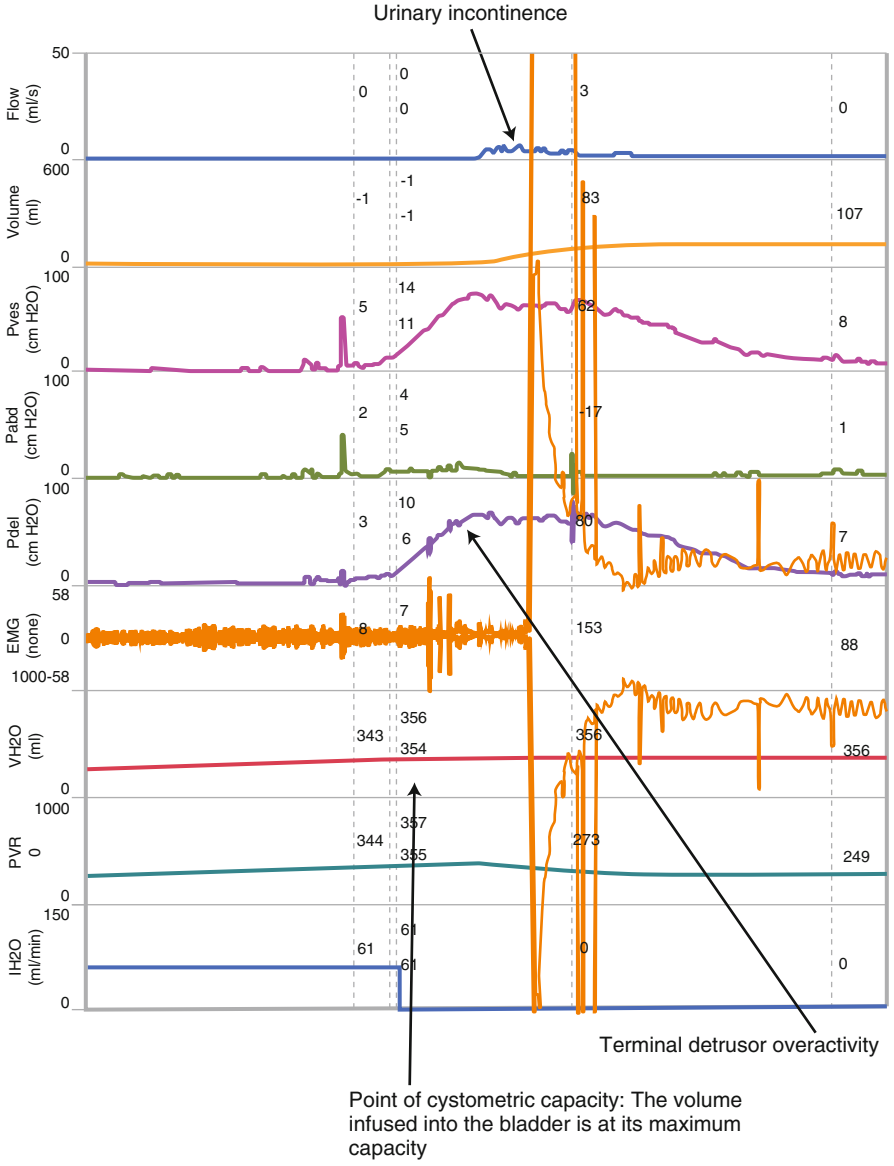

Fig. 14.10 This patient has terminal DO defined as a *single involuntary detrusor contraction occurring at cystometric capacity, which cannot be suppressed, and results in incontinence* [1]

and occurs particularly frequently in elderly patients with neurological conditions such as cerebrovascular accidents (CVA) [13–15].

A sudden increase in detrusor pressure after cessation of voiding and in the absence of flow has been referred to as a detrusor after contraction (DAC). There are various opinions on the causes of DAC. Some believed DAC may be an artifact in

the P_{det} measurement when the sensor directly contacts the bladder wall [16]. Others performed cystometric evaluations using three pressure transducers (two microtip transducers and one external fluid-filled pressure transducer) and observed that in 50 % of the recordings of DAC, the increase in P_{det} was present in all three transducers [17]. Consequently, they concluded DAC was a true detrusor contraction. A different interpretation of DAC was proposed by Vereecken and colleagues. They noted that in 59 of 65 patients with DAC, the DAC was preceded by a contraction of the anal or urethral sphincter seen as a burst of activity in the EMG. They theorized that DAC developed from the sudden cessation of the outflow of urine either by voluntary or involuntary interruption of the urethral sphincter causing an acute rise in the P_{det} [18]. Although this urodynamic observation is not new and was first reported back in the early 1930s [19], only a few studies have been published which focus on DAC over the past 60 years. To date, no conclusive explanation of its significance or clinical importance has been established.

The goal of UDS is to reproduce the patient's symptoms and to determine the potential cause of these symptoms by urodynamic measurements or observations. In this chapter of "bladder filling and storage; (involuntary) contractions," the goal was to identify IDCs during the filling and storage phases of UDS. Learning to identify the presence of DO during UDS is important. However, its interpretation in the context of the patient's symptoms and condition, for example identifying high storage pressure ($P_{det} > 40$ cm H_2O) and recognizing its deleterious effects on the upper tracts, is far more important in order to better characterize the clinical picture and facilitate optimal treatment planning.

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